



## **Feeding young children aged 1 to 5 years**

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# Executive summary

## Background

- S.1 Between 1974 and 1994, the Committee on Medical Aspects of Food and Nutrition Policy (COMA) published a series of reports on infant feeding practices in the UK and made recommendations for infant and young child feeding. The last of these reports, [‘Weaning and the weaning diet’](#), was published in 1994 and has been the basis for much of the advice on feeding young children in the UK (DH, 1994b).
- S.2 Subsequent recommendations made by the Scientific Advisory Committee on Nutrition (SACN) and by international expert committees have carried implications for current infant feeding policy. These include the adoption of [World Health Organization \(WHO\) Growth Standards](#) (SACN/RCPCH, 2007; WHO MGRS, 2006a; WHO MGRS, 2006b) and [revisions to energy requirements](#) (FAO, 2004; SACN, 2011a).
- S.3 Accordingly, SACN requested its Subgroup on Maternal and Child Nutrition (SMCN) to review recent developments in this area. To complement this work, the Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT) was asked by the Department of Health to conduct a review of the risks of toxicity from chemicals in the diets of infants and young children. COT was also asked to examine the evidence relating to the influence of the infant diet on development of allergic and autoimmune disease.
- S.4 This report covers the period from 1 to 5 years of age (12 to 60 months) and accompanies the [‘Feeding in the first year of life’ report](#), which was published in 2018 (SACN, 2018).

## Terms of reference

- S.5 The terms of reference as they apply to this report are:
- to review the scientific basis of current recommendations for feeding children aged 1 to 5 years (12 to 60 months)
  - to consider evidence on developmental stages and other factors that influence eating behaviour and diversification of the diet in the early years
  - to make recommendations for policy, practice and research.
- S.6 The key dietary factors considered in this report are:
- energy requirements

- macronutrients
- micronutrients (focussing on vitamins A, C and D, iron and zinc)
- foods, food components and dietary patterns (including consideration of vegetarian and vegan diets, and consumption of different food groups)
- drinks
- eating and feeding behaviours
- chemical contaminants (or the risk of chemical toxicity).

S.7 The key child and adolescent health outcomes considered in this report are:

- growth and body composition
  - linear growth
  - body composition (body mass index, adiposity)
  - excess weight (overweight and obesity)
- neurodevelopment and cognitive development
- bone or skeletal health outcomes
- oral health
- morbidities, including respiratory diseases.

S.8 The key adult health outcomes considered in this report are:

- overweight or obesity
- cardiovascular outcomes (coronary heart disease, diabetes)
- cancer.

S.9 SACN considers evidence for the general population and does not make recommendations related to clinical assessment or management of children with clinical conditions requiring specialist care.

## Methods

- S.10 [SACN's Framework for the Evaluation of Evidence](#) (SACN, 2012) was used as the basis for considering appropriate evidence for inclusion in the review. It should be noted that the Framework has since been updated. The [latest version of SACN's Framework](#) was published in 2023.
- S.11 Consideration of the evidence was primarily focused on systematic reviews (SRs) and meta-analyses (MAs) of randomised controlled trials (RCTs), prospective cohort studies (PCS) and non-randomised studies of interventions (NRSIs).

- S.12 SACN also considered evidence on young child feeding from large national surveys. The report includes data on food and drink consumption, and nutrient intakes and status, in young children living in the UK from the 2011 Diet and Nutrition Survey of Infants and Young Children (DNSIYC) for children aged 12 to 18 months (Lennox et al, 2013) and the National Diet and Nutrition Survey rolling programme (mainly from years 2016 to 2019) (NDNS) for children aged 18 to 60 months (Bates et al, 2020). The report also includes data on the prevalence of overweight and obesity in children entering primary school (aged 4 to 5 years) from the National Child Measurement Programme (for England), the Child Health Surveillance Programme School system (for Scotland) and the Child Measurement Programme for Wales (there are currently no comparable data in children aged under 5 years for Northern Ireland).
- S.13 In parallel with SACN, COT considered the risks of toxicity from chemicals in the diet of young children aged 1 to 5 years and whether current government advice should be revised.

## Assessment of the systematic review evidence

- S.14 The methodological quality of individual SRs was assessed using [SACN's Framework for the Evaluation of Evidence](#) (SACN, 2012) and the quality assessment tool, AMSTAR 2 (AMSTAR, 2021).
- S.15 The certainty of evidence from SRs was assessed using modified methods based on those outlined in the SACN reports '[Carbohydrates and Health](#)' (SACN, 2015) and '[Saturated Fats and Health](#)' (SACN, 2019).
- S.16 The certainty of the evidence was graded 'adequate', 'moderate', 'limited', 'inconsistent' or 'insufficient'. Evidence that was graded 'adequate' or 'moderate' was used to inform conclusions and recommendations of this report (alongside findings from national dietary surveys). These are summarised in Table S.1.

**Table S.1 Systematic review evidence in children aged 1 to 5 years graded 'moderate' or 'adequate'**

Topic area	Systematic review finding	Certainty of evidence
Energy	Larger portion sizes of snacks and meals provided in preschool settings are associated with higher food and energy intakes in the short term (less than 6 months)	Moderate
Macronutrients	Higher total protein intake in children aged 1 to 5 years is associated with higher body mass index (BMI) in childhood	Moderate
Macronutrients	Higher free sugars intake is associated with increased dental caries (increment, incidence or prevalence) in childhood and adolescence	Adequate
Drinks	Higher sugar-sweetened beverage (SSB) consumption in children aged 1 to 5 years is associated with greater odds of overweight or obesity in childhood	Adequate
Drinks	Higher SSB consumption in children aged 1 to 5 years is associated with a greater increase in BMI in childhood and adolescence	Moderate
Eating and feeding behaviours	Feeding practices (including repeated taste exposure, pairing with positive stimuli such as liked foods, modelling of vegetable consumption and offering the child non-food rewards) increase vegetable consumption in children aged 1 to 5 years (in the short term, up to 8 months)	Moderate
Eating and feeding behaviours	Repeated taste exposure to vegetables increases vegetable consumption in children aged 1 to 5 years (in the short term, up to 8 months)	Moderate
Excess weight and obesity	Higher child BMI or weight status at age 1 to 5 years is associated with higher adult BMI or risk of overweight or obesity	Adequate
Excess weight and obesity	Child BMI at age 6 years and under is not associated with incidence of coronary heart disease in adulthood	Moderate
Excess weight and obesity	Child BMI at age 6 years and under is not associated with incidence of stroke in adulthood	Moderate

Topic area	Systematic review finding	Certainty of evidence
Oral health	Breastfeeding beyond 12 months is associated with lower odds of malocclusion (teeth that are not aligned correctly)	Moderate

## Limitations of the evidence base

S.17 A range of limitations was identified in the evidence base provided by SRs and dietary surveys. These are summarised below.

### General limitations of the systematic review evidence

- S.18 There was either no or insufficient SR evidence for a number of dietary exposures (including saturated fat and dietary fibre) and health outcomes (including paediatric cancers, allergy and autoimmune diseases, and bone and skeletal health) which were included in the scope and literature search for this risk assessment.
- S.19 Many of the SRs identified for this report had a broad search strategy that included population groups outside the age range of interest for this report (children aged 1 to 5 years) and it was difficult to determine whether their search strategy for the target population was comprehensive.
- S.20 Most of the SR evidence that was specific to children aged 1 to 5 years was observational (from PCS) or from NRSIs and may have been subject to confounding and selection bias.
- S.21 The evidence base on many topic areas was highly heterogeneous in terms of exposures, dietary assessment methods, outcome measures, populations, settings, and study designs, which prevented the pooling of results by MA or other methods of quantitative synthesis.
- S.22 Due to the lack of quantitative syntheses in the included SRs, risk of publication bias was seldom formally assessed.
- S.23 The SR evidence identified on micronutrients was drawn almost exclusively from supplementation and food fortification trials designed for populations in low income, lower-middle or upper-middle income countries (defined according to the World Bank classification system) and therefore may not be generalisable to children living in the UK.

- S.24 Primary studies, particularly those conducted in high income countries, seldom considered whether the impact of dietary exposures on nutritional status (for example, vitamin D) or health outcomes differed among different ethnic groups.
- S.25 The majority of primary studies had short follow-up periods, limiting the ability to draw conclusions about the longer-term health effects of nutrient or dietary intake in children aged 1 to 5 years.

## **General limitations of the evidence from dietary surveys**

- S.26 DNSIYC was conducted in 2011. Dietary patterns may have changed significantly in the period since the data were collected.
- S.27 The number of children that provided blood samples for status measures in NDNS was small and may not be representative of the wider population. Children who gave a blood sample were more likely to come from higher socioeconomic status households.
- S.28 Misreporting of food consumption, specifically underreporting, and therefore underestimation of total dietary energy intake (TDEI) in self-reported dietary methods is a well-documented source of bias and is an important consideration when interpreting survey data.

## **Conclusions**

- S.29 The current diet of young children in the UK, as captured in both DNSIYC and NDNS, does not meet current dietary recommendations for several nutrients.
- S.30 The following conclusions are informed by the main findings from DNSIYC and NDNS together with SR evidence that was graded 'adequate' and 'moderate' (Table S1).

## **Energy and macronutrients**

- S.31 Evidence from DNSIYC and NDNS indicated that:
- mean intakes of total dietary energy (TDEI) for children aged 1 to 3 years were above the Estimated Average Requirement (EAR)
  - mean intakes of free sugars for children aged 1.5 to 5 years were above the current recommendation of no more than 5% TDEI
  - mean intakes of dietary fibre for children aged 1.5 to 5 years were below the recommended intake of 15 grams per day

- mean intakes of saturated fats were above the current recommendation of no more than 10% TDEI (which applies in full from age 5 years)
- mean intakes of protein were above the Reference Nutrient Intake (RNI).

S.32 Evidence identified from SRs indicated that:

- larger portion sizes provided in preschool settings are associated with higher food and energy intakes in the short term (less than 6 months)
- higher free sugars intake in children aged 1 to 5 years is associated with increased dental caries (increment, incidence or prevalence) in childhood and adolescence
- higher total protein intake in children aged 1 to 5 years is associated with higher BMI in childhood
- higher child BMI or weight status is associated with higher risk of adult overweight or obesity.

S.33 These findings are of concern in relation to wider evidence on:

- the high prevalence of overweight and obesity in childhood in the UK particularly in lower socioeconomic groups and in some ethnic groups
- the high prevalence of dental caries in children in the UK.

## Micronutrients

S.34 Evidence from DNSIYC and NDNS indicated that mean salt intake was above the target average salt intake in children aged 1.5 to 4 years, where 76% of children in this age group had intakes above the target salt intake.

S.35 Evidence from DNSIYC and NDNS indicated that certain groups of children, including children from lower socioeconomic status households (measured by the Index of Multiple Deprivation) and some ethnic groups, may be at risk of inadequate intakes of iron, zinc, vitamin A and vitamin D, and low vitamin D status. Conversely, intakes of vitamin C exceeded the RNI across all age groups.

S.36 Evidence from NDNS indicated that use of vitamin D supplements in the general population of children aged 1 to 5 years was low (no comparable data were available for supplements containing vitamin A or C); while the latest available data indicated variable uptake of Healthy Start vitamins (containing vitamins A, C and D).

## Foods

S.37 Currently there are no UK government recommendations on portion sizes for vegetables and fruit for young children. Evidence from NDNS indicated that

children ate more fruit than vegetables. Consumption of total vegetables and fruit decreased with increasing deprivation. Encouraging consumption of vegetables as children grow and develop more independence around food is important to support children to meet population dietary recommendations.

- S.38 Evidence identified from SRs indicated that repeated taste exposure to a vegetable (around 8 to 10 times) can increase consumption of that vegetable in the short term (less than 8 months).
- S.39 Evidence from DNSIYC indicated that the food group (sugar-sweetened) 'yoghurts, fromage frais and dairy desserts' was among the top contributors to free sugars intake in children aged 1 to 1.5 years, providing 18% of free sugars intake at a population level.
- S.40 Evidence from NDNS indicated that foods that are energy dense and high in saturated fat, salt or free sugars contributed approximately 16% TDEI, 24% TDEI and 30% TDEI in children aged 1 to 1.5 years, 1.5 to 4 years and 4 to 5 years, respectively. Of these, biscuits, buns, cakes and pastries were the largest contributor to TDEI.
- S.41 Evidence from DNSIYC indicated that among children aged 12 to 18 months who consumed commercially manufactured foods and drinks marketed specifically for infants and young children (65% of this age group), these products provided approximately 20% of free sugars intakes.
- S.42 A PHE evidence review (2019) found that the nutrient composition of many of these products was inconsistent with UK dietary recommendations for this age group, particularly for sugar and salt. The PHE review highlighted that commercially manufactured finger foods have been the main driver in the growth of the infant food market in recent years.

## Drinks

- S.43 Evidence from DNSIYC and NDNS indicated that:
- formula milks (mainly follow-on formula and milks marketed for children over the age of 1 year, also known as 'toddler milks' and 'growing-up milks') were consumed by 36% of children aged 1 to 1.5 years and contributed 50% of free sugars intake in consumers (18% of free sugars intake at a population level)
  - fruit juice (100% fruit juice and smoothies) contributed nearly 11% to free sugars intake in children aged 1.5 to 4 years and less than 10% in the other age groups at a population level.
- S.44 Substitution analysis using data from DNSIYC indicated that replacing whole cows' milk with semi-skimmed cows' milk for children aged 1 to 1.5 years would be unlikely to have a detrimental effect on nutrient intakes at the population level. By

contrast, replacing whole milk with skimmed or 1% milk may result in a greater risk of inadequate intakes of vitamin A in young children.

- S.45 Evidence identified from SRs indicated that higher sugar-sweetened beverage (SSB) consumption in children aged 1 to 5 years is associated with a greater odds of overweight or obesity in childhood.
- S.46 Evidence identified from SRs indicated that continued breastfeeding beyond the age of 1 year is protective against malocclusion (teeth that are not correctly aligned).

## Risks of chemical toxicity

- S.47 COT assessed toxicity issues from the infant and young child diet for a number of nutrients, substances and contaminants in breast milk, infant formula and solid foods. They concluded there were unlikely to be concerns over toxicity in the diet of young children for substances considered at current levels of exposure. Issues where COT has identified there may be potential concerns are described in chapter 10.
- S.48 Nutritional and toxicological aspects associated with the consumption of plant-based drinks by children aged 1 to 5 years in the UK are being considered in a benefit:risk assessment conducted jointly by SACN and COT. Findings are expected to be published in 2024 and will include recommendations on plant-based drink consumption. More information on the work of the [joint SACN-COT working group](#) is available on the SACN page of GOV.UK.
- S.49 SACN's Feeding in the first year of life report (2018) considered findings from a benefit:risk assessment on timing of introduction of peanut and hen's egg into the infant diet and the risk of developing allergy to these foods. The available evidence indicated that the deliberate exclusion or delayed introduction of peanut or hen's egg beyond 6 to 12 months of age may increase the risk of allergy to the same foods. These findings will have a bearing on children in the older age group (1 to 5 years).

## Recommendations

- S.50 The following recommendations are suitable for children aged 1 to 5 years who are able to consume a varied diet and are growing appropriately for their age.
- S.51 Between 1 to 2 years of age, children's diets should continue to be gradually diversified in relation to foods, dietary flavours and textures. A flexible approach is recommended to the timing and extent of dietary diversification, taking into account the variability between young children in developmental attainment and

the need to satisfy their individual nutritional requirements. [SACN 2023, SACN 2018]

- S.52 Current UK dietary recommendations as depicted in the Eatwell Guide should apply from around age 2 years [SACN 2023], with the following exceptions:
- UK dietary recommendations on average intake of free sugars (that free sugars intake should not exceed 5% of total dietary energy intake) should apply from age 1 year [SACN 2023]
  - milk or water, in addition to breast milk, should constitute the majority of drinks given to children aged 1 to 5 years [SACN 2023]
  - pasteurised whole and semi-skimmed cows' milk can be given as a main drink from age 1 year [SACN 2023], as can goats' and sheep's milks [SACN 2023, COMA 1994].
  - pasteurised skimmed and 1% cows' milk should not be given as a main drink until 5 years of age. These lower fat milks can be used in cooking. [SACN 2023, COMA 1994]
  - children aged 1 to 5 years should not be given rice drinks as they may contain too much arsenic [SACN 2023 endorses COT 2016, 2021]
  - children aged 1 to 5 years should not be given sugar-sweetened beverages [SACN 2023]
  - dairy products (such as yoghurts and fromage frais) given to children aged 1 to 5 years should ideally be unsweetened. [SACN 2023, COMA 1994]
- S.53 Formula milks (including infant formula, follow-on formula, 'growing-up' or other 'toddler' milks) are not required by children aged 1 to 5 years. [SACN 2023 endorses WHO 2013]. Specialised formula, including low-allergy formula, are also usually not required after the first year of life. [SACN 2023]
- S.54 Foods (including snacks) that are energy dense and high in saturated fat, salt or free sugars should be limited in children aged 1 to 5 years in line with current UK dietary recommendations. [SACN 2023]
- S.55 Commercially manufactured foods and drinks marketed specifically for infants and young children are not needed to meet nutritional requirements. [SACN 2023]
- S.56 Salt should not be added to foods given to children aged 1 to 5 years. Children aged 1 to 3 years should, on average, aim to have no more than 2g of salt per day; the figure for children aged 4 to 6 years is 3g per day. [SACN 2023, SACN 2003]
- S.57 Children aged 1 to 5 years should be presented with unfamiliar vegetables on multiple occasions (as many as 8 to 10 times or more for each vegetable) to help develop and support their regular consumption. [SACN 2023]

- S.58 Deliberate exclusion of peanut or hen's egg (and foods containing these) beyond 12 months of age may increase the risk of allergy to the same foods. Importantly, once introduced, these foods should continue to be consumed as part of the child's usual diet in order to minimise the risk of allergy to peanut or hen's egg developing after initial exposure. [SACN 2023, SACN-COT 2018]
- S.59 Children aged 1 to 5 years should continue to be offered a wide range of foods that are good sources of iron. They do not require iron supplements unless advised by a health professional. [SACN 2023, SACN 2018]
- S.60 Children aged 1 to 5 years should be given a daily supplement of 10µg (400 IU) vitamin D and 233µg vitamin A unless, contrary to recommendations, they are consuming more than 500ml of formula milk per day (see S.53). [SACN 2023, SACN 2016, COMA 1994]
- S.61 Vitamin C supplements are not necessary for the general population. However, there is no evidence that taking vitamin C supplements at the current recommended level of supplementation has any adverse effects. [SACN 2023]
- S.62 It is recommended that government considers a range of strategies and actions to improve the diets of children aged 1 to 5 years, and continues to monitor dietary intakes, and the nutritional, weight and oral health status of young children as outlined below.
- S.63 Consider strategies to support and promote:
- continuation of breastfeeding into the second year of life [SACN 2023]
  - current UK dietary recommendations to children aged 1 to 5 years [SACN 2023]
  - feeding of an appropriate and diverse diet to children aged 1 to 5 years that meets nutritional requirements but does not exceed energy requirements [SACN 2023]
  - awareness and uptake of current advice on vitamins D and A supplements at the current recommended levels in children aged 1 to 5 years, particularly in at-risk groups such as children from some ethnic groups and lower socioeconomic status households [SACN 2023]
  - good oral health in children aged 1 to 5 years [SACN 2023]
- S.64 Consider strategies to reduce consumption of:
- free sugars and excess protein in children aged 1 to 5 years [SACN 2023]
  - foods (including snacks) that are energy dense and high in saturated fat, salt or free sugars in children aged 1 to 5 years, while encouraging uptake of healthier snacks [SACN 2023]
  - sugar-sweetened beverages in children aged 1 to 5 years [SACN 2023]

S.65 Actions for consideration:

- develop and communicate age-appropriate portion sizes for food and drinks, including for vegetables, fruit, fruit juice and milk, for children aged 1 to 5 years [SACN 2023]
- review advice on the need for vitamin C supplements for children aged 1 to 5 years [SACN 2023]
- support parents or caregivers of children aged 1 to 5 years following vegetarian, vegan and plant-based diets to ensure the nutritional requirements (including for iron, iodine, calcium and vitamin B12) of their children are met [SACN 2023]

S.66 Monitoring of children aged 1 to 5 years for consideration:

- collect detailed, nationally representative data on nutrient intakes and status [SACN 2023]
- collect detailed data on nutrient intake and status of population subgroups, including ethnically diverse populations and socially disadvantaged groups, [SACN 2023]
- monitor the nutritional impact of a population shift towards adopting vegetarian, vegan and plant-based diets [SACN 2023]
- continue to monitor the prevalence of both overweight and obesity and the extent of excess energy intakes [SACN 2023]
- continue to monitor oral health [SACN 2023]
- monitor intakes of low or no calorie sweeteners [SACN 2023]

## Research recommendations

S.67 Throughout the development of this report, SACN identified a number of significant gaps in the evidence relating to infant and complementary feeding as well as limitations in the study design for some of the available research. The Committee has therefore made a number of recommendations for research which are described in the report (see chapter 13).

# 1 Background

- 1.1 Between 1974 and 1994, the Committee on Medical Aspects of Food and Nutrition Policy (COMA) published a series of reports on infant feeding practices in the UK and made recommendations for infant and young child feeding. The last of these reports, [‘Weaning and the weaning diet’](#), was published in 1994 and has been the basis for much of the advice on feeding young children in the UK (DH, 1994b).
- 1.2 Subsequent recommendations made by the Scientific Advisory Committee on Nutrition (SACN) and by international expert committees have carried implications for current infant feeding policy. These include the adoption of [World Health Organization \(WHO\) Growth Standards](#) (SACN/RCPCH, 2007; WHO MGRS, 2006a; WHO MGRS, 2006b) and [revisions to energy requirements](#) (FAO, 2004; SACN, 2011a).
- 1.3 Accordingly, SACN requested its Subgroup on Maternal and Child Nutrition (SMCN) to review recent developments in this area. To complement this work, the Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT) was asked by the Department of Health (DH) to conduct a review of the risks of toxicity from chemicals in the diets of infants and young children. COT was also asked to examine the evidence relating to the influence of the infant diet on development of allergic and autoimmune disease.
- 1.4 This report covers the period from 12 to 60 months of age (1 to 5 years). It forms part of a wider piece of work considering the scientific basis of current recommendations for feeding children up to 5 years of age, of which the first part, [‘Feeding in the first year of life’](#), was published in 2018 (SACN, 2018). The decision to split the review into 2 age groups covering infants aged 0 to 12 months and young children aged 1 to 5 years was largely pragmatic. SACN recognises that this boundary does not reflect the underlying biology, which is a continuum; feeding in the first year of life impacts on nutritional status and health outcomes in the second year of life and beyond.
- 1.5 SACN provides independent scientific advice on, and risk assessment of, nutrition and related health issues. It advises the 4 UK governments. In line with the [SACN Framework for the Evaluation of Evidence](#) (SACN, 2012; SACN, 2020; SACN, 2023), SACN’s role is to assess scientific information (risk assessment) to assist policy making and translation into advice (risk management), which is the responsibility of government health departments. The committee does not advise on how recommendations are taken forward for policy nor evaluate their wider implications (for example, agricultural, political, economic).
- 1.6 The role of government, the health service, and non-governmental organisations in protecting, promoting and supporting breastfeeding fall under risk management and are not in the scope of this report.

1.7 This report was developed using SACN process and was signed off by SACN.

## Terms of reference

1.8 The terms of reference for [‘Feeding in the first year of life’](#) and for the current report are:

- to review the scientific basis of current recommendations for complementary and young child feeding up to 5 years (60 months) of age<sup>1</sup>. The current report covers young children aged 1 to 5 years of age (12 to 60 months)<sup>2</sup>
- to consider evidence on developmental stages and other factors that influence eating behaviour and diversification of the diet in the early years
- to review the nutritional basis for current dietary recommendations applying to breastfeeding mothers (where relevant to the health of the infant). As this report covers the 1 to 5 age group, it was not considered relevant to address this term of reference.
- to make recommendations for policy, practice and research.

1.9 The key dietary factors considered in this report are:

- energy requirements
- macronutrients
- micronutrients (focus on vitamins A, C and D<sup>3</sup>, iron and zinc)
- foods, food components and dietary patterns (including consideration of vegetarian and vegan diets, and consumption of different food groups)
- drinks
- eating and feeding behaviours
- chemical contaminants (or the risk of chemical toxicity).

## Health outcomes considered

1.10 The health outcomes considered in this report are divided into child and adolescent health outcomes and adult health outcomes.

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<sup>1</sup> To note that this should be understood as 5 completed years of age.

<sup>2</sup> The original terms of reference specified the age group in months (12 to 60 months) but SACN considered that designating the age group in years would make this report more accessible.

<sup>3</sup> For vitamin D it was agreed that only data published since the SACN report ‘Vitamin D and health’ (2016) cut-off date for inclusion of evidence would be included.

1.11 Child and adolescent<sup>4</sup> health outcomes are:

- growth and body composition
  - linear growth
  - body composition (body mass index, adiposity)
  - excess weight (overweight and obesity)
- neurodevelopment and cognitive development
- bone or skeletal health outcomes
- oral health
- morbidities, including respiratory diseases.

1.12 Adult health outcomes are:

- overweight or obesity
- cardiovascular outcomes (coronary heart disease, diabetes)
- cancer.

1.13 SACN considers evidence for the general population and does not make recommendations related to clinical assessment or management of children with clinical conditions requiring specialist care.

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<sup>4</sup> [Defined by the WHO](#) as children aged 10 to 19 years.

# History of policy development

## UK recommendations on feeding children aged 1 to 5 years

- 1.15 In 1991, the COMA convened a working group (WG) to review the scientific evidence in relation to nutritional adequacy of the weaning diet. While previous 'Present Day Practice' reports addressed the diet of infants in the first months after birth, ['Weaning and the weaning diet'](#) (DH, 1994b) included recommendations on when and what types of first foods to introduce and the progression of complementary feeding.
- 1.16 The terms of reference of the WG were "To review the nutrition of young children during weaning and to make recommendations". The WG considered the nutrition of infants and young children between the ages of about 6 weeks to about 2 years and defined weaning as "the process of expanding the diet to include foods and drinks other than breast milk or infant formula". The report focused on the first 2 years of life as being the likely limits of the weaning period but acknowledged the continuing importance of diet and nutrition for older children.
- 1.17 The recommendations from ['Weaning and the weaning diet'](#) (DH, 1994b) underpin many current UK government dietary recommendations including:
- the timing of introduction of whole, semi-skimmed and skimmed milk
  - the use of other drinks
  - the use of drinking vessels
  - recommended quantities of milk and dairy products
  - advice on dietary fat intake
  - vitamin supplementation (specifically vitamins A and D alongside longstanding advice on vitamin C supplementation)
  - the amount and types of foods (number of meals or snacks per day).
- 1.18 Earlier recommendations on vitamin supplementation were revised in the COMA report (DH, 1994b) to state that from the age of 6 months, infants receiving breast milk as their main drink, or less than 500ml per day of infant formula, should be given supplements of vitamins A, C and D.
- 1.19 The UK recommendations on feeding young children and the evidence informing these are listed in Annex 1, Table A1.1.

## **Statutory schemes to improve the dietary intakes of young children in the UK**

- 1.20 In addition to UK government dietary advice, there are several statutory schemes (see Annex 1, Table A1.2) that aim to improve the dietary intakes of young children in the UK, including the Healthy Start scheme and the Best Start Foods scheme.
- 1.21 In 1999, COMA undertook a review of the Welfare Food Scheme (DH, 2002). Based on recommendations made by COMA, the scheme (which had been in place since 1940) was changed in a number of respects and re-designated 'Healthy Start'. Healthy Start replaced the means-tested elements of the Welfare Food Scheme throughout the UK in 2006. Important aspects were the rebranding of the vitamin preparations as 'Healthy Start' vitamin supplements for young children (providing vitamins A, C and D) and mothers (providing folic acid and vitamins C and D). The range of foods offered was also widened through the introduction of exchangeable vouchers which could be used at participating retailers towards the cost of plain cows' milk, infant formula suitable from birth, and fresh vegetables and fruit. Since 2021, the NHS Business Services Authority, which delivers the Healthy Start scheme as directed by the Department of Health and Social Care (DHSC), has led the work to digitise the Scheme. This includes the introduction of an online application form and a pre-paid card to replace the paper form and vouchers in use since the scheme's introduction in 2006.
- 1.22 In Scotland, the Healthy Start scheme was replaced by the Best Start Foods (BSF) scheme from August 2019. Recipients receive a payment card which can be used to buy any of the following foods: fresh eggs; milk (plain cows' milk and first infant formula); fresh, frozen or tinned vegetables or fruit; and dried, fresh, frozen or tinned pulses. Entitlement to vitamins was not included in the BSF scheme. Instead, this was replaced by universal access to vitamin D provision for children under age 3 years and breastfeeding mothers.

## **World Health Organization recommendations**

- 1.23 The World Health Organization (WHO) has published several reports which provide recommendations for infant and young child feeding, focusing largely on breastfeeding and complementary feeding. The WHO defines complementary feeding as "the provision of foods or fluids to infants in addition to breast milk" (WHO Europe, 2003). Further information on complementary feeding and its principles can be found elsewhere (SACN, 2018; WHO Europe, 2003).
- 1.24 Since 2001, the WHO has recommended that mothers worldwide exclusively breastfeed their infants for the first 6 months to achieve optimal growth, development and health (WHO, 2001a). Thereafter they should be given nutritious

solid foods as breastfeeding continues up to the age of 2 years or beyond. This recommendation was reiterated in WHO/UNICEF (2003). These recommendations also cover:

- the salt and sugar content of solid foods
- the energy density of solid foods
- the texture of solid foods.

1.25 The WHO/UNICEF (2003) recommendations are further summarised in Annex 1 Table A1.3. Since 2003, WHO has published 3 further reports on complementary feeding which are of direct relevance to the UK context, details of which can be found in the SACN report '[Feeding in the first year of life](#)' (SACN, 2018).

## Other national or international recommendations

1.26 Several other international bodies have considered young child feeding and established recommendations. General healthy eating guidelines for young children across the different international bodies are broadly consistent. These, together with more specific advice on recommended intakes of salt, sugars, dietary fat, dietary fibre, breast milk or milk and other beverages for young children, are summarised in Annex 1 Table A1.3.

## Current context in the UK

1.27 Food consumption, nutrient intakes and nutritional status in children in the UK are captured in 2 large national surveys, the [Diet and Nutrition Survey for Infants and Young Children](#) (Lennox et al, 2013) and the [National Diet and Nutrition Survey](#) (NDNS) (Bates et al, 2020). The DNSIYC was a stand-alone survey in infants and young children aged 4 to 18 months and was carried out over 8 months in 2011. The NDNS is a continuous cross-sectional survey in children aged 18 months upwards (as well as adults and adolescents). For a summary of the methods used in DNSIYC and NDNS see Annex 2.

1.28 The DNSIYC and the latest available NDNS data (years 2016 to 2019, or years 9 to 11 of the Rolling Programme) indicated that the diets of young children in the UK are not in line with current government recommendations. Children aged 1.5 to 4 years exceeded recommendations for dietary energy, protein, saturated fats and free sugars and did not meet recommendations for dietary fibre. The NDNS also suggested that there are proportions of children (>5 to 10%) in some age groups under 5 years who may have inadequate intakes of iron, zinc, vitamin A and vitamin D.

- 1.29 A consequence of inadequate diets is that the prevalence of overweight and obesity in young children remains too high and oral health remains poor.
- 1.30 The latest available data from child measurement programmes in England and Scotland for the collection year 2021 to 2022 indicated that the prevalence of overweight and obesity combined in children aged 4 to 5 years was 22.3% and 24.1%, respectively. The prevalence of obesity in England and Scotland (at 10.1% and 11.7%, respectively) decreased from that in the collection year 2020 to 2021 when measurements were taken during the beginning of the COVID-19 pandemic but remained higher than before the pandemic. In Wales, limited data from the collection year 2020 to 2021 also indicated that the prevalence of obesity (approximately 18%) had increased compared with the pre-pandemic collection year 2018 to 2019 (no comparable data are available for Northern Ireland). Data from these measurement programmes also indicated that deprivation is a major risk factor for obesity in childhood, while increased BMI in early childhood is a strong predictor of obesity in later childhood.
- 1.31 Dental caries in children remains a major public health problem. The latest available survey data indicated that nearly 11% of children aged 3 years (PHE, 2021c) and 23% of children aged 5 years (PHE, 2020b) in England experienced obvious tooth decay. In Scotland, 27% of children aged 5 years had obvious tooth decay (Public Health Scotland, 2020), while in Wales and Northern Ireland, the figures were 34% (Cardiff University, 2017) and 40% (HSCIC, 2015), respectively. Almost 9 out of 10 hospital tooth extractions among children aged 0 to 5 years are due to preventable tooth decay and tooth extraction is still the most common hospital procedure in children aged 6 to 10 years (PHE, 2020b; PHE, 2021b).

## **Determinants of dietary behaviours and lifelong health and disease**

- 1.32 Normal growth and development are characterised by a regulated increase in the size, mass and complexity of function of tissues and organs. Differential growth and development during fetal life and early childhood could lead to differences in body composition, metabolic, physiological function, and influence chronic disease risk in adulthood (SACN, 2011b). For example, epidemiological evidence has suggested modest inverse associations between birthweight and risk of coronary heart disease; while lower birthweight, lower weight at age 1 year and increased BMI in childhood have been associated with an increased risk of cardiovascular disease (SACN, 2011b).
- 1.33 There are many biological, environmental and social factors that can shape food preferences in young children and ultimately their dietary behaviours in later life (see chapter 7 for details). While the food preferences and eating habits of young children are strongly shaped by their caregivers' attitudes and beliefs about

feeding, and culture and behaviours around food (Mennella et al, 2006; Schwartz et al, 2011), the food and drink choices that caregivers make for their children are also shaped by their socioeconomic circumstances (food security or insecurity) and the wider food environment, including what foods and drinks are available and how these are marketed and advertised to parents and caregivers (PHE, 2019a; Silventoinen et al, 2010). For example, the nutritional composition, messaging and marketing of commercially manufactured foods and drinks that are marketed specifically for young children are not always in line with young child feeding dietary guidelines (PHE, 2019a). In addition, while home-prepared foods are generally recommended to help introduce infants and young children to a range of appropriate flavours and textures, one fifth of children in the UK eat food purchased from 'out of home' food outlets (such as takeaways and restaurants) at least once a week (PHE, 2017b). Meals and snacks from such outlets are typically higher in energy, salt and saturated fat than home-cooked meals (Huang et al, 2021; Robinson et al, 2018).

- 1.34 Evidence also suggests that food marketing aimed at children and adolescents across a multitude of platforms (for example, television, digital and social media) has a sizeable influence in shaping attitudes, beliefs and behaviours around food, and is a cause for concern for many parents (Boyland et al, 2022; WHO, 2022).
- 1.35 In the UK, providers of childcare also play a vital role in supporting the healthy development of children (PHE, 2017a; Warren et al, 2022). The majority of children aged under 5 years spend time in some form of childcare provided by early years settings (such as nurseries) or childminders, with many receiving the majority of their meals during their time in childcare (DfE, 2022; Scottish Government, 2022). Childcare providers therefore have a duty to ensure that the meals they provide follow young child feeding dietary guidelines (NHS Health Scotland, 2018; PHE, 2017a).

## 2 Methods

- 2.1 As noted in chapter 1, this report forms the second part of SACN's review of the scientific basis of recommendations for feeding young children under 5 years, the first part of which considered feeding in the first year of life (SACN, 2018). For both reports, [SACN's Framework for the Evaluation of Evidence](#) (SACN, 2012) was used as the basis for assessing the evidence. The [latest version of the SACN Framework](#) was published in 2023.
- 2.2 The SACN Framework is based on an evidence hierarchy which ranks the certainty of the evidence according to study design. More weight is given to evidence from randomised controlled trials (RCTs) since well-conducted RCTs minimise the potential for selection bias and confounding. Less weight is given to observational studies because these study designs are potentially subject to confounding and reverse causality. However, in the absence of RCTs, observational evidence from non-randomised studies of interventions (NRSI) and prospective cohort studies (PCS) is still considered stronger than observational evidence from other study designs (case-control, cross-sectional and case reports).
- 2.3 While SACN (2018) considered evidence from primary studies (mainly RCTs and PCS, but also cross-sectional studies and case reports), this report is based on evidence provided by systematic reviews (SRs) or meta-analyses (MAs) of PCS, NRSI and RCTs. Well-conducted, comprehensive, high quality SRs and MAs reduce the potential for biased study selection or overlooking relevant studies since they are systematic and provide a comprehensive and quantitative analysis of the research in a particular field. SACN's preferred approach is to use evidence provided by published SRs and MAs to inform its evaluations rather than conducting its own systematic reviews of primary evidence. This is because undertaking a SR is time and resource intensive. SACN's approach makes use of existing published evidence and draws upon broader scientific expertise. However, there are also limitations since the value of SRs in informing recommendations is dependent on their quality, the quality of the included studies and the analyses conducted. In addition, the relevance and generalisability of the results of SRs are dependent on how closely the SR question matches SACN's research question, the specific inclusion or exclusion criteria and comparators.
- 2.4 This report also reflects how SACN's approach to evaluating the evidence has evolved to reflect changes in methodologies in the broader scientific community. This includes the incorporation of formal quality assessment into SACN's process for evaluating evidence (for details see ['Evaluation of the quality and certainty of systematic review evidence'](#)).

## Inclusion criteria

- 2.5 The research question underpinning the literature search for this report was ‘What is the impact of/relationship between diet/nutrition/food and drink consumption in children aged 12 to 60 months old and health?’.
- 2.6 The following types of studies met the inclusion criteria: SRs and MAs of RCTs, NRSI and PCS.
- 2.7 Additional eligibility criteria included:
- English language publications, conducted in populations in health and directly relevant to the UK, and published in peer-reviewed scientific or medical journals from January 1990.
  - Evidence from studies conducted in high income countries (HICs). Evidence from studies conducted in low income, lower-middle income and upper-middle income countries (LICs, LMICs and UMICs, respectively, defined according to the [World Bank classification system](#)) that was potentially relevant to the UK context was also considered.

## Exclusion criteria

- 2.8 The following types of studies were excluded: primary studies, reviews that included only case-control studies, and narrative (non-systematic) reviews.
- 2.9 Additional exclusion criteria were:
- reviews published in grey literature, such as dissertations, conference proceedings, magazine articles, books or book chapters, opinion pieces, information from websites, and other non-peer reviewed articles
  - studies in hospitalised or malnourished patients and those in children with a disease, including infectious disease
  - interventions to reduce obesity prevalence that did not have a dietary or feeding style component of interest; childcare setting intervention, unless they had a dietary or feeding style component of interest; weight management interventions.

## Literature search

- 2.10 The Knowledge and Library Services team at Public Health England (PHE) conducted online database searches to identify SRs, MAs and pooled analysis examining the relationship between the diet of young children in health aged 1 to 5 years and health outcomes (see chapter 1, paragraphs 1.11 and 1.12).

- 2.11 EMBASE, Ovid MEDLINE, Food Science Technology Abstracts, Scopus and the Cochrane Library were searched, using the search terms outlined in Annex 3 (Tables A3.1 and A3.2), for relevant publications meeting the inclusion criteria (see paragraphs 2.5 and 2.7).
- 2.12 Interested parties were invited to highlight any additional evidence (which met the inclusion criteria for the review) to that identified by the PHE literature search in a call for evidence published on the SACN website (from 11 March to 5 April 2019).
- 2.13 Reference lists of all included publications (identified through the online database search or highlighted by interested parties, up to May 2019) were hand searched. Reference lists of relevant reviews by international organisations were also considered.
- 2.14 A supplementary online database search was performed for oral health in October 2019.
- 2.15 The agreed initial cut-off date for consideration of eligible evidence for the draft report was 22 May 2019.
- 2.16 The draft report was made available for public consultation from 20 July 2022 to 20 September 2022 and interested parties were invited to alert SACN to any evidence it may have missed.
- 2.17 The Committee considered additional relevant SR evidence that was identified through the consultation process or published before the cut-off date of 11 November 2023. It was agreed that the report would be amended if any evidence so identified was judged to have an important bearing on the conclusions.

## **Selection of studies**

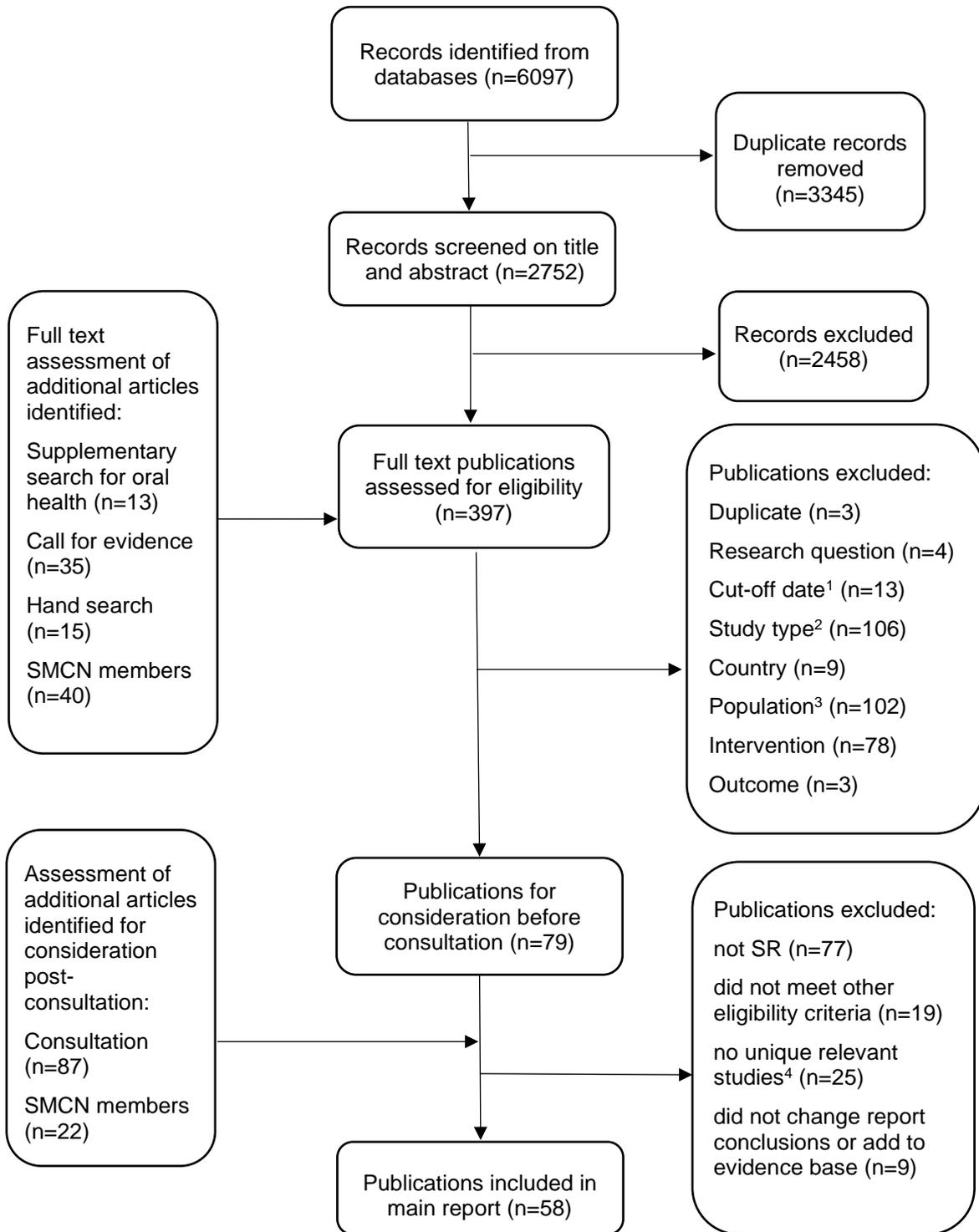
- 2.18 After removing duplicates, titles and abstracts of the identified publications were screened for eligibility.
- 2.19 The steps for screening on title and abstract and screening on full text were performed using [Eppi-Reviewer 4](#). At both stages of screening, 10% of the publications were independently screened by 2 reviewers to ensure reliability and reproducibility of the screening tool. Differences were resolved by consensus. Where uncertainty remained, advice from SMCN was sought.
- 2.20 A total of 6097 records were identified from 5 online databases (see paragraph 2.11). After removal of duplicates (n=3345), 2752 records identified through the online database search were screened for eligibility on title and abstract. A further 2458 records were excluded. The full texts of 294 records were retrieved and screened. Ninety additional full-text publications identified from other sources were also screened:

- 35 highlighted by interested parties through the call for evidence
  - 40 by members of SMCN
  - 15 through hand searching of reference lists.
- 2.21 The supplementary online database search on oral health returned 2701 records, and full texts of 13 were retrieved and screened.
- 2.22 Of the 397 full-text articles that were screened, 79 SRs met the inclusion criteria, while 318 publications were excluded for the following reasons:
- 3 were duplicates
  - 4 were not sufficiently relevant to the research question for this risk assessment
  - 13 were published before the cut-off dates for consideration of evidence for previous SACN reports (SACN, 2010; SACN, 2016)
  - 106 were either not an SR or did not include eligible studies (RCT or NRSI or PCS) in the 1 to 5-year age group
  - 9 were conducted in countries which did not provide findings relevant to the UK context
  - 102 did not include studies or findings in the 1 to 5-year age group or in children in health
  - 78 examined interventions that did not meet the inclusion criteria for this risk assessment
  - 3 examined outcomes that were not covered by this risk assessment.
- 2.23 Details of the excluded references and reasons for their exclusion are presented in Annex 4, Tables A4.1 to A4.3.
- 2.24 Of the 79 eligible SRs identified before the public consultation (see paragraph 2.23), evidence from 25 SRs was included in more recent or comprehensive reviews (see Annex 4, Table A4.3 for a list of these SRs). Therefore, data from these SRs were not extracted into evidence tables (see [Data extraction](#)). Details of the remaining 54 SRs are presented in Annex 5 (Tables A5.1 to A5.7).
- 2.25 A total of 109 publications were identified for consideration after the consultation. Of these, 87 publications were highlighted through the consultation process and 22 publications (all published after the May 2019 cut-off date) were suggested by the committee.
- 2.26 Of the 109 publications, 13 SRs met the inclusion criteria and the quality of the evidence they provided was preliminarily assessed. Of these, 7 SRs were assessed in detail (see [Grading of the evidence from systematic reviews](#)) because they could potentially change existing conclusions or add to the evidence base

(see Annex 6, Tables A6.1 to A6.3 for details of these SRs). Of the 7 SRs, 4 SRs were included in the final report.

2.27 A total of 58 publications were included in the final report. The process for study selection and inclusion is presented in Figure 2.1.

**Figure 2.1 Flow diagram showing the literature selection process (described in paragraphs 2.20 to 2.27)**



<sup>1</sup> Published before the cut-off dates for consideration of evidence for previous SACN reports (SACN, 2010; SACN, 2016).

<sup>2</sup> Excluded for not being a SR or for not including studies (RCT or NRSI or PCS) in the 1 to 5 age group.

<sup>3</sup> Excluded for not including studies or findings in the 1 to 5 age group or in children in health.

<sup>4</sup> All relevant studies included in more recent or comprehensive SRs considered in the report.

## Data extraction

2.28 The following data were extracted into evidence tables (Annex 5 and 6): first author, year of publication, study design, funding, declaration of interest, research question, total number of participants, demographics, outcome measures, confounders, study findings, the method for assessing study quality and limitations identified by the SR authors.

2.29 To enable a more detailed assessment and interpretation of the evidence from SRs without MAs, further data extraction of the characteristics and findings of primary studies included in the SRs was carried out (Annex 9, Tables A9.1 to A9.50). Data extracted included: sample size, country, age, intervention duration or duration of follow-up, exposure, outcome, confounding factors, study power, funding sources. Data were extracted from primary studies if they:

- included participants aged 1 to 5 years (12 to 60 months) at baseline or if the mean age of participants at baseline was younger than age 5 years; and for the lower boundary, primary studies with participants aged 1 to 5 years at the end of the intervention
- were RCTs, NRSI or PCS; if the SR reported results from cross-sectional analyses from a RCT or PCS, these results were not extracted.

2.30 For SRs with MAs, summary estimates from MAs were extracted (rather than individual findings of primary studies). Summary estimates were not extracted if:

- the summary estimate pooled estimates from cross-sectional or case control studies
- studies in children aged 1 to 5 contributed less than 50% weighting to the summary estimate.

If a subgroup analysis in children aged 1 to 5 years was performed, only the estimate from that subgroup was extracted.

## **Prioritisation of systematic review evidence and reporting of results**

- 2.32 In this report, 'body composition' was used to collectively denote anthropometric measures such as body mass index (BMI), body fat (% or in grams) and weight-for-height z-scores; while 'weight status' was used to collectively denote excess weight (overweight, obesity or severe obesity). 'Overweight', 'obesity' and 'severe obesity' are defined in chapter 8.
- 2.33 Where appropriate, SR evidence that relates dietary or nutritional intakes to measures of body composition or weight status were grouped together and presented ahead of evidence that relates dietary or nutritional intakes to other health outcomes.
- 2.34 If the evidence informing a topic area was derived from  $\geq 2$  SRs without MAs that overlapped (that is, included the same primary studies), findings from the largest, most comprehensive SR (number of primary studies) were reported in full. For smaller SRs, only findings from primary studies that were uniquely identified by and included in those SRs were reported.
- 2.35 If the evidence informing a topic area came from  $\geq 2$  SRs with MAs that overlapped, all summary estimates from the MAs were reported but the overlap between the MAs was considered when assessing the certainty of the evidence (see Grading of the evidence).
- 2.36 The study overlap between eligible SRs is presented in Annex 7, Tables A7.1 to A7.9.
- 2.37 Findings were reported as they were reported in the SRs. If statistical findings (effect estimates, confidence intervals, p-values, and statistical heterogeneity) were not reported in SRs, this was indicated by 'NR' (not reported). The age of study participants was reported in months, unless SRs reported this in years.
- 2.38 The word 'effect' was used to describe evidence from RCTs and the word 'association' was used to describe evidence from PCS and other NRSI. An effect or association was deemed to be statistically significant using the  $p < 0.05$  criterion.
- 2.39 When describing the direction of an association between a nutrient, food or dietary component (exposure) and a health outcome, the term 'direct' was used to indicate when an increase in the exposure was associated with an increase in the outcome variable; the term 'inverse' was used to describe the opposite association.

## Health outcomes for which systematic review evidence was identified

- 2.41 For this report, SACN sought to identify SR evidence on a number of child (including adolescent) and adult health outcomes. Table 2.1 and Table 2.2 lists the health outcomes for which SR evidence was identified and where in the report the evidence is described. SR evidence was also sought but not identified on paediatric cancer (such as childhood leukaemia), child allergic and autoimmune disease, adult neurological health, and adult bone or skeletal health.

**Table 2.1. Child and adolescent health outcomes for which systematic review evidence was identified. Adolescence is defined as children aged 10 to 19 years (WHO, 2023).**

Outcome – child and adolescent health	Location in the report
<p>Growth, body composition or weight status</p> <p>Note: ‘Body composition’ denotes outcome measures including body mass index, body fat (% or grams) and weight-for-height z-scores. ‘Weight status’ denotes excess weight (overweight, obesity and severe obesity), which are defined in chapter 8.</p>	<p>Chapter 3 – energy and macronutrients</p> <p>Chapter 4 – micronutrients</p> <p>Chapter 5 – foods, dietary patterns, and dietary components</p> <p>Chapter 6 – drinks</p> <p>Chapter 7 – eating and feeding behaviours</p> <p>Chapter 8 – excess weight and obesity</p>
Neurodevelopment or cognitive development	<p>Chapter 3 – energy and macronutrients</p> <p>Chapter 4 – micronutrients</p> <p>Chapter 5 – foods, dietary patterns and dietary components</p> <p>Chapter 6 – drinks</p>
Bone or skeletal health outcomes	<p>Chapter 3 – energy and macronutrients</p> <p>Chapter 4 – foods, dietary components and dietary patterns</p>
Oral health	Chapter 9 – oral health
Morbidities, including respiratory diseases	Chapter 4 – micronutrients

**Table 2.2. Adult health outcomes for which systematic review evidence was identified**

Outcome – adult health	Location in the report
Overweight or obesity	Chapter 8 – excess weight and obesity
Cardiovascular outcomes (coronary heart disease, diabetes)	Chapter 8 – excess weight and obesity
Adult cancers	Chapter 8 – excess weight and obesity

## Evaluation of the quality and certainty of systematic review evidence

- 2.42 For this report, [SACN's Framework for the Evaluation of Evidence](#) (SACN, 2012) was used as the basis for assessing SR evidence. It should be noted that since work commenced on this report, the SACN Framework has been updated. The latest version of [SACN's Framework for the Evaluation of Evidence](#) was published in 2023.
- 2.43 The methodological quality of SRs was also assessed using [A Measurement Tool to Assess Systematic Reviews 2](#) (AMSTAR 2) tool (AMSTAR, 2021).

### SACN Framework (2012)

- 2.44 The following criteria were considered during the evidence evaluation for this report:
- SRs, MAs and pooled analyses
    - scope and aims
    - search dates (publication dates of studies included in the reviews or MAs)
    - inclusion and exclusion criteria
    - number of primary studies and total number of participants
    - conduct of review and reporting of pre-specified outcomes consistent with registered protocol.
  - Primary studies considered within SRs or MAs
    - whether the primary studies were RCTs, NRSI or PCS

- populations considered and relevant characteristics, for example, the number of studies which included children in the age range under consideration (1 to 5 years)
- sample size or power
- exposure or intervention duration and follow-up
- quality of the dietary assessment methods and outcome assessment methods
- Interpretation of results and their analysis
  - appropriateness of statistical methods used
  - whether and which confounding factors were taken into account in the study design and subsequent analysis
  - consistency of the effect or association (taking account of overlap in the primary studies considered)
  - heterogeneity – an  $I^2$  statistic of 0 to 25% was considered to represent low heterogeneity, 26 to 75% was considered to represent medium heterogeneity and >75% was considered to represent high heterogeneity. While a high  $I^2$  statistic reflects uncertainty regarding the value of the pooled estimate, it does not necessarily reflect uncertainty regarding the direction of the effect/association (which may be consistent across studies)
  - direction and size of effect and statistical significance
  - results of subgroup and sensitivity analyses.

## AMSTAR 2 assessment

- 2.45 For each eligible publication, the methodological quality was assessed using AMSTAR 2. The methodological quality of each eligible publication was assessed by 2 members of the secretariat and any differences were resolved by discussion between assessors. Advice was sought from SMCN if consensus could not be reached between assessors.
- 2.46 More information on the [AMSTAR 2 checklist and guidance on how to use the tool](#) is available online.
- 2.47 Briefly, AMSTAR 2 comprises 16 items for evaluation (AMSTAR, 2021) which are listed in Box 1 below.

### **Box 1. AMSTAR 2 criteria for evaluation**

1. Did the research questions and inclusion criteria for the review include the components of PICO (population, intervention, control group, outcome)?
2. Did the report of the review contain an explicit statement that review methods were established prior to the conduct of the review and did the report justify any significant deviations from the protocol?
3. Did the review authors explain their selection of the study designs for inclusion in the review?
4. Did the review authors use a comprehensive literature search strategy?
5. Did the review authors perform study selection in duplicate?
6. Did the review authors perform data extraction in duplicate?
7. Did the review authors provide a list of excluded studies and justify the exclusions?
8. Did the review authors describe the included studies in adequate detail?
9. Did the review authors use a satisfactory technique for assessing the risk of bias in individual studies that were included in the review?
10. Did the review authors report on the sources of funding for the studies included in the review?
11. If MA was performed, did the review authors use appropriate methods for statistical combination of results?
12. If MA was performed, did the review authors assess the potential impact of risk of bias in individual studies on the results of the MA or other evidence synthesis?
13. Did the review authors account for risk of bias in primary studies when interpreting or discussing the results of the review?
14. Did the review authors provide a satisfactory explanation for, and discussion of, any heterogeneity observed in the results of the review?
15. If they performed quantitative synthesis, did the review authors carry out an adequate investigation of publication bias (small study bias) and discuss its likely impact on the results of the review?
16. Did the review authors report any potential sources of conflict of interest, including any funding they received for conducting the review?

2.48 The authors of AMSTAR 2 proposed a scheme for interpreting weaknesses detected in critical and non-critical questions to rate overall confidence in the results of the review as shown in Table 2.3.

**Table 2.3. Rating overall confidence in the results of the review**

Overall rating	Description
High	No or one non-critical weakness: the systematic review provides an accurate and comprehensive summary of the results of the available studies that address the question of interest.
Moderate	More than one non-critical weakness: the systematic review has more than one weakness but no critical flaws. It may provide an accurate summary of the results of the available studies that were included in the review. (Note: multiple non-critical weaknesses may diminish confidence in the review and it may be appropriate to move the overall appraisal down from moderate to low confidence.)
Low	One critical flaw with or without non-critical weaknesses: the review has a critical flaw and may not provide an accurate and comprehensive summary of the available studies that address the question of interest.
Critically low	More than one critical flaw with or without non-critical weaknesses: the review has more than one critical flaw and should not be relied on to provide an accurate and comprehensive summary of the available studies.

- 2.49 The items identified as critical by AMSTAR 2 are items 2, 4, 7, 9, 11, 13 and 15. In the context of this risk assessment, SMCN agreed that question 2 (relating to protocol registration) and question 7 (relating to the list of excluded studies) were not considered as critical domains as few of the included SRs met these best practices. Therefore, the critical domains for this risk assessment were items 4, 9, 11, 13 and 15.
- 2.50 As many of the SRs identified for this risk assessment included all study designs, item 3 was not considered applicable.
- 2.51 Ten percent of the publications were independently reviewed by 2 reviewers. Differences were resolved by consensus. A summary of the AMSTAR 2 assessment is provided in Annex 8 (Table A8.1 to A8.10).

## Approach to considering statistical methods

- 2.52 The results of 2 statistical models of MA, fixed effects and random effects, are increasingly being reported in SRs with MAs. There are differences in the underlying assumptions and statistical considerations of the models. Random-effects models generally give proportionally more weight to small than to large primary studies, while fixed-effects models give weight in direct proportion to the

size of the primary studies. However, the choice of models and their interpretation remains an area of debate among statisticians (SACN, 2019). More detailed information on the 2 models is available in the [Cochrane Handbook for Systematic Reviews of Interventions](#) (Higgins et al, 2022).

- 2.53 The following approach, used in the SACN report '[Saturated Fats and Health](#)' (SACN, 2019), was used when considering the MAs:
- Where results of only 1 model (that is, fixed-effects model or random-effects model) were stated, these were reported and used to draw conclusions.
  - Where results of both models were stated, both were reported. The following factors were considered: appropriateness of the model assumptions, direction and magnitude of the effect, statistical significance and level of agreement between the models. Where the results of the 2 models differed, the totality of the evidence and expert judgement were used to draw conclusions and considered in the final grading of the evidence (see next section below).

## Grading of the evidence from systematic reviews

- 2.54 The certainty of evidence from SRs and MAs was assessed using modified methods based on those outlined in the SACN reports '[Carbohydrates and Health](#)' (SACN, 2015) and '[Saturated Fats and Health](#)' (SACN, 2019).
- 2.55 The certainty of evidence for each exposure-outcome relationship covered by included SRs was graded 'adequate', 'moderate', 'limited', 'inconsistent' or 'insufficient'.
- 2.56 The evidence was first assigned an interim grade based on the number of identified SRs or MAs (and their primary studies) for that exposure-outcome relationship. Expert judgement, based on the criteria detailed in Table 2.4, was then used to upgrade or downgrade the certainty of the evidence. If MAs were identified for a given exposure-outcome relationship, the evidence grade was based on the findings of the best quality or largest MA (by number of studies or participants).
- 2.57 Summary tables of the evidence grading process for each exposure-outcome relationship are presented in Annex 10 (Tables A10.1 to A10.35).
- 2.58 Exposure-outcome relationships for which there were fewer than 3 RCTs, NRSI or PCS were automatically graded 'insufficient'. The exposure-outcome relationships for which evidence was graded 'insufficient' are listed in Annex 10 (Table A10.36).
- 2.59 Evidence for exposure-outcome relationships that was graded 'adequate' or 'moderate' was used to inform conclusions and recommendations of this report,

together with findings from national diet and nutrition surveys (see [Other evidence considered](#)).

**Table 2.4 Criteria for grading evidence (SACN, 2019)**

Certainty of evidence	Explanatory notes
Adequate	<p>There is ‘adequate’ evidence to make a decision about the effect or association of a factor(s) or intervention(s) in relation to a specific outcome.</p> <p>Taking into account overlap of primary studies included in the identified publications, evidence from meta-analyses goes in the same direction.</p> <p>The results of MAs are statistically significant or, in systematic reviews without MA, there is convincing evidence of a consistent significant effect or association in the primary studies considered. Effects or associations are also consistent when major population subgroups or other relevant factors are considered in additional analyses.</p> <p>The identified publications are considered to be of good quality based on the key factors listed above.</p> <p>The inclusion and exclusion criteria of the identified publications are well defined and appropriate.</p> <p>A judgement of ‘adequate’ evidence is also made based on the number, size, quality and durations or follow-ups of RCTs or PCS included in the identified SRs, MAs and pooled analyses.</p> <p>Where only 1 SR, MA or pooled analysis is identified on a specific outcome, evidence is considered ‘adequate’ if the publication reports primary data from <math>\geq 3</math> RCTs or <math>\geq 5</math> PCS, of ‘adequate’ size, considered to be of good quality and which were included in a MA or pooled analysis. Alternatively, for a single SR without a MA or pooled analysis, evidence may be considered ‘adequate’ if a total of <math>\geq 4</math> RCTs or <math>\geq 5</math> PCS studies, of ‘adequate’ size and considered to be of good quality, consistently went in the same direction.</p>

Certainty of evidence	Explanatory notes
Moderate	<p>There is 'moderate' evidence (therefore less conclusive) to make a decision about the effect or association of a factor(s) or intervention(s) in relation to a specific outcome.</p> <p>Taking into account overlap of primary studies included in the identified publications, the majority of the evidence from MAs goes in the same direction.</p> <p>The results of MAs are statistically significant or, in SRs without MA, there is moderate evidence of a consistent significant effect or association in the primary studies considered.</p> <p>Effects or associations may be less consistent when major population subgroups or other relevant factors are considered in additional analyses.</p> <p>The identified publications are considered to be of moderate to good quality based on the key factors listed above.</p> <p>The inclusion and exclusion criteria of the identified publications are reasonably well defined and generally appropriate.</p> <p>Compared with evidence considered adequate, there may be fewer and smaller RCTs or PCS, of moderate quality with sufficient durations or follow-ups, included in the identified SRs, MA and pooled analyses.</p> <p>Where only 1 SR, MA or pooled analysis is identified on a specific outcome, evidence is considered moderate if the publication reports primary data from <math>\geq 3</math> RCTs or 3-4 PCS of moderate size, considered to be of moderate quality and which were included in a MA or pooled analysis. Alternatively, for a single SR without a MA or pooled analysis, evidence may be considered moderate if a total of <math>\geq 3</math> RCTs or 5 PCS, of moderate size and considered to be of moderate quality, consistently went in the same direction.</p>

Certainty of evidence	Explanatory notes
Limited	<p>There is 'limited' evidence (therefore, even less conclusive) to make a decision about the effect or association of a factor(s) or intervention(s) in relation to a specific outcome.</p> <p>Taking into account overlap of primary studies included in the identified publications, the majority of the evidence from meta-analyses goes in the same direction.</p> <p>The results of meta-analyses are statistically significant or, in the case of systematic reviews without meta-analysis, there is 'limited' evidence of a consistent significant effect or association in the primary studies considered.</p> <p>Effects or associations may be inconsistent when major population subgroups or other relevant factors are considered in additional analyses.</p> <p>The identified publications are considered to be of poor to moderate quality based on the key factors listed above.</p> <p>The inclusion and exclusion criteria of the identified publications are not well defined and may not be appropriate.</p> <p>Compared with evidence considered 'adequate' or 'moderate', there may be fewer and smaller RCTs or PCS, of low quality with inadequate durations or follow-ups, included in the identified SRs, MA and pooled analyses.</p> <p>Where only 1 SR, which did not include a meta-analysis, is identified on a specific outcome, evidence was considered 'limited' if primary data from 3 to 4 RCTs or PCS of 'limited' size and considered to be of low quality were identified but there was some evidence that the results were in the same direction.</p>
Inconsistent	<p>There is 'inconsistent' evidence after taking into account the above quality criteria and overlap of primary studies included in the identified SR, MA and pooled analyses, the results in relation to a specific outcome are conflicting and it is not possible to draw a conclusion.</p>
Insufficient	<p>There is 'insufficient' evidence as a result of no SRs, MA or pooled analyses of appropriate quality identified in relation to a specific outcome or, in a single review or analysis, &lt;3 to 4 eligible RCTs or PCS were identified. Therefore, it is not possible to draw conclusions.</p>

## Other evidence considered

- 2.60 Two large national surveys informed the sections describing nutrient intakes, food and drink consumption and nutritional status of young children living in the UK (see chapters 3 to 6). These were the [Diet and Nutrition Survey of Infants and Young Children](#) (DNSIYC) for children aged 4 to 18 months (Lennox et al, 2013) and the [National Diet and Nutrition Survey rolling programme](#) (NDNS) for children aged 18 to 60 months (Venables et al, 2022). DNSIYC was a standalone survey of food consumption, nutrient intake and nutritional status in infants and children aged 4 to 18 months, carried out over eight months in 2011. The NDNS is a continuous cross-sectional survey of food consumption, nutrient intake and nutritional status in adults and children aged 18 months upwards. Data collection started in 2008. Most of the NDNS data and secondary analyses presented in this report is based on the 3 most recent collection years available (years 2016 to 2019). Some secondary analyses are based on all 11 years of data available (years 2008 to 2019) where larger cell sizes were required. For a summary of the methods used in the DNSIYC and the NDNS see Annex 2. Full details of the methods and findings from the 2 surveys can also be found elsewhere (Bates et al, 2020; Lennox et al, 2013). In the current report, age groups are reported in months rather than years in line with how these were reported in the published reports (Bates et al, 2020; Lennox et al, 2013).
- 2.61 Data from the [National Child Measurement Programme](#) in England, the Child Health Surveillance Programme School system in Scotland, and the Child Measurement Programme for Wales informed sections on the prevalence of overweight and obesity in young children (in chapter 8). There are currently no comparable data in children aged under 5 years for Northern Ireland.
- 2.62 The Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT) was asked to examine the risks of toxicity from chemicals in the diet of young children aged 1 to 5 years and to consider whether current government advice should be revised. Details of the approach taken and weblinks to the COT statements describing COT's assessments, findings and associated advice are provided in chapter 10.
- 2.63 In addition to the literature searches outlined (see [Literature search](#)), previously published SACN reports<sup>5</sup> of relevance to this report were considered and searches were undertaken to update evidence that might have accrued since their publication.

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<sup>5</sup> SACN reports on: [Salt and health](#) (2003), [Review of dietary advice on vitamin A](#) (2005), [Iron and health](#) (2010), [Dietary Reference Values for energy](#) (2011), [The influence of maternal, fetal and child nutrition on the development of chronic disease in later life](#) (2011), [Vitamin D and Health](#) (2016), [Joint SACN/RCPC report on: Application of WHO growth standards in the UK](#) (2007).

- 2.64 Key national and international SRs and reports from the US Department of Agriculture or Nutrition Evidence Library, World Health Organization, National Institute for Health and Care Excellence and Scottish Intercollegiate Guidelines Network were also considered where appropriate.

## Process for assessment of the evidence

- 2.65 SACN considered SRs, MAs and pooled analyses that met the inclusion criteria. Chapters were initially drafted by members of the SMCN secretariat with support from the committee. These chapters provided the basis for SMCN discussions with the final text, conclusions and recommendations discussed and agreed by the SACN main committee.
- 2.66 This report was made available for public consultation and the comments received from interested parties were taken into consideration before the report was finalised.

## General limitations of the evidence

- 2.67 This section describes a number of general limitations that were identified in the evidence base provided by SRs, MAs and dietary surveys. Limitations specific to each topic area are summarised in their respective chapters. Additional details on the limitations of the evidence from NDNS are provided in chapter 4 (see [Limitations of the evidence on micronutrients](#)) and Annex 2.

## General limitations of the evidence from systematic reviews

- 2.68 There was either no or insufficient SR evidence for a large number of exposure-outcome relationships of interest for this risk assessment (see [Health outcomes](#), and Annex 10, Table 10.36).
- 2.69 SRs were included in this report if they searched for evidence in children aged 1 to 5 years. However, many of the SRs had a broader search strategy that included population groups outside the age range of interest (that is, children aged under 12 months or above the age of 5 years). It was therefore difficult to determine whether the search strategy for the target age group was comprehensive.
- 2.70 Risk of publication bias was seldom formally (statistically) assessed because the majority of SRs either did not include MAs or any other method of quantitative synthesis. Of those SRs that did include MAs, many had insufficient numbers of primary studies to enable a quantitative assessment of publication bias (for example, the use of funnel plots).

- 2.71 SRs without MAs reported findings from primary studies in varying degrees of detail. Effect sizes or associations and measures of uncertainty (confidence intervals or exact p-values) were not always reported, making interpretation of findings difficult. The clinical or biological relevance of studies that demonstrated a small effect size or association was not always clear.
- 2.72 Most of the evidence from SRs was from observational studies (PCS) or NRSI, which are study designs that are at high risk of confounding bias. A confounding factor is an unmeasured variable that influences both the exposure of interest (for example, nutrient intake) and the outcome (for example, body weight). These include gender, physical activity, social and economic influences, and ethnicity. Even among studies that accounted for potential confounding, it can be difficult to obtain accurate and precise measures for confounding factors so that their effects can be accurately quantified or adjusted for in analyses, leading to residual confounding (SACN, 2011a). Moreover, there was often a lack of consistency in the confounding factors accounted for in primary studies, which made it difficult to compare study findings.
- 2.73 SRs did not always account for risk of selection bias. If there were systematic differences between participants lost to follow up and participants who completed a study, this could lead to attrition bias, a form of selection bias.
- 2.74 Although many SR authors declared potential conflicts of interests, SRs did not always report the funding sources of the primary studies included. Commercially funded studies may be more likely to report favourable rather than unfavourable findings (Helfer et al, 2021).
- 2.75 SRs that included RCTs seldom included information on the type of analysis (intention-to-treat [ITT] or per protocol [PP]) reported. ITT analysis includes all participants originally allocated at randomisation; it measures the effectiveness of an intervention and is more relevant to public health (SACN, 2021). PP analysis includes only those participants who completed the study; it measures the efficacy of an intervention and, since it only includes data on completers, it could overestimate the intervention effect (SACN, 2021).
- 2.76 Primary studies included in SRs did not always report power calculations, making it difficult to interpret findings of null associations when confidence intervals were wide.
- 2.77 The evidence base on many topic areas was highly heterogeneous in terms of exposures, outcome measures, populations, settings, and study designs, which prevented the pooling of results into meta-analyses or other methods of quantitative synthesis.
- 2.78 Primary studies included in the SRs used different dietary assessment methods (for example, food frequency questionnaires, 24-hour recalls, food diaries). In most studies, dietary assessments were reported by a parent or caregiver of the child.

The reliability and validity of consumption estimates is uncertain since misreporting of food consumption (particularly underreporting by failing to report foods or drinks consumed or underestimating quantities) and changes to normal intakes during the recording period are known problems in dietary surveys (Mirmiran et al, 2006). Technical difficulties in the dietary assessment process, such as assumptions made in relation to food composition, recipes and portion sizes, quality and completeness of food and nutrient databases, can also affect the accuracy of consumption estimates.

- 2.79 The types of data reported included dichotomous (relative risks, odds ratios) and continuous (mean difference) outcome measures. Many primary studies included in the SRs used odds ratios (OR) rather than relative risk (RR) to estimate disease risk. The use of OR amplifies the risk estimate (in either direction) when the disease risk (for example, early childhood caries [ECC]) in the population is high ( $\geq 10\%$ ) (Ranganathan et al, 2015).
- 2.80 Where a measure of body size was the outcome (for example, body mass index), assessments were often performed and reported by the parent or caregiver of the child rather than by a trained practitioner. This decreases the reliability of outcome measurements.
- 2.81 Primary studies covered a wide range of time points, but the majority of studies had a 1 to 3-year follow-up period. Therefore, much of the evidence identified did not allow conclusions to be drawn about the longer term health effects of nutrient or dietary intake in children aged 1 to 5 years, or sustained effects of increasing children's acceptance or intake of certain foods (for example, vegetables).
- 2.82 Primary studies, particularly those conducted in high income countries (defined according to the [World Bank classification](#)), seldom considered whether the impact of dietary exposures on nutritional status (for example, vitamin D) or health outcomes differed among different ethnic groups.

## **General limitations of the evidence from dietary surveys**

- 2.83 The Diet and Nutrition Survey in Infants and Young Children (DNSIYC) was carried out in 2011. Dietary patterns may have changed significantly in the period since the data was collected.
- 2.84 Each NDNS fieldwork year collects data on approximately 150 to 160 children aged 18 to 60 months as part of a wider annual sample of 500 children aged 18 months to 18 years and is designed to be representative of the UK population. However, the sample of children that provide blood samples for status measures is much smaller, typically 15 to 20 per year.
- 2.85 An analysis conducted on the characteristics of NDNS participants indicated that

there were differences in the characteristics of children who gave a blood sample compared with the whole NDNS sample of children (see Annex 11 for details). For children aged 18 to 47 months, girls made up a marginally higher proportion of children who gave a blood sample compared with their proportion of the whole sample (52.9% versus 48.8% of the whole sample). The youngest children surveyed (aged 18 to 23 months) were underrepresented in the group who gave a blood sample compared with their proportion of the whole sample (9.4% versus 14.8% of the whole sample). White children were underrepresented in the group who gave a blood sample (75.6% vs 80.5% of the whole sample) as were Asian and Asian British children (6.7% vs 8.4% of the whole sample). For children aged 48 to 60 months, the proportion of children who gave a blood sample based on their age, sex and ethnic group roughly matched the age, sex and ethnic group breakdown of the whole sample.

- 2.86 Children who gave a blood sample were more likely to come from higher socioeconomic status households (where the Household Reference Person [HRP] worked in higher managerial and professional occupations).
- 2.87 Misreporting, and specifically underreporting, of food consumption and therefore underestimation of total dietary energy intake (TDEI) in self-reported dietary methods is a well-documented source of bias and is an important consideration when interpreting NDNS data. The NDNS rolling programme is one of the few national large-scale population surveys to include doubly labelled water (DLW) as an objective biomarker to validate energy intake estimated from reported food and drink consumption (see [chapter 3](#) for details). However, the latest available DLW sub-study of the NDNS rolling programme (collection years 2013 to 2015) only included children aged 4 years and older. For details on how potential misreporting of TDEI in the absence of DLW data in all the age groups of interest was investigated for this report and its implications, see [chapter 4](#).

## Structure of report

- 2.88 The structure of chapters 3 to 6 and 8 to 9 of this report are as follows:
- each chapter opens with a background section followed by evidence sections that describe the SR evidence identified on each topic area and the assessment of that evidence
  - the background sections of chapters 3 to 6 summarise current UK dietary recommendations and findings from DNSIYC and NDNS.
  - the background section of chapter 8 ([Excess weight and obesity](#)) describes the latest available data on overweight and obesity prevalence from national child measurement programmes in the UK (chapter 8).
  - the background section of chapter 9 ([Oral health](#)) describes the latest available data on dental health in young children in the UK.

# 3 Energy and macronutrients

## Energy

### Background

- 3.1 Energy is required for tissue maintenance and growth, to generate heat (thermogenesis), and for physical activity (Fleischer Michaelsen et al, 2003). In 2011, SACN set the energy requirements for all population groups (with the exception of pregnant women) as the level of dietary energy intake required to maintain a healthy body weight in otherwise healthy people at existing levels of physical activity (SACN, 2011a). Allowances were made for any additional physiological needs. For example, during infancy and childhood, the energy requirement must also meet the needs for healthy growth and development (SACN, 2011a). Weight gain is a sensitive indicator of the adequacy of energy intake in young children (Fleischer Michaelsen et al, 2003).
- 3.2 There is some evidence that infants have an intrinsic ability to self-regulate their energy intake according to requirements by responding to internal cues of satiety (Peters et al, 2012). This ability has also been demonstrated in children up to the age of 5 years in short-term studies (usually done in a single day) that measure the impact on total dietary energy intake (TDEI) when the energy content of foods offered to the child is changed (Rogers et al, 2016). However, this ability to adjust TDEI to meet requirements appears to diminish between the ages of 11 and 15 months (Brugailleres et al, 2019). Experimental research has shown that by the time they enter primary school, children do not fully adjust their TDEI and continue to eat when offered larger portion sizes regardless of how full they are (see [Effect of portion sizes on food or energy intake](#)).
- 3.3 An impaired ability to self-regulate energy intake may tip the balance between TDEI and energy that is expended and increase the risk of excess weight gain. A recent longitudinal experimental study demonstrated that children at ages 11 and 15 months with the greatest ability to self-regulate their energy intake experienced the lowest gains in Body Mass Index (BMI) z-score between ages 11 and 15 months and had the lowest BMI z-score at age 2 years (Brugailleres et al, 2019).

### Current recommendations for energy intake in the UK

- 3.4 In 2011, SACN published [revised dietary reference values \(DRVs\) for energy](#), which replaced the previous DRVs for energy set by the Committee on Medical Aspects of Food Policy (COMA) in 1991 (DH, 1991). For dietary energy, DRVs are

set at the average reference value, the Estimated Average Requirement (EAR). SACN has set revised EAR values for dietary energy for all age groups, including young children aged 1 to 5 years (SACN, 2011a).

## Comparison between SACN (2011) and COMA (1991) energy reference values

- 3.5 Table 3.1 presents the energy reference values for young children [derived by SACN in 2011](#) (SACN, 2011a) compared with summary values reported by COMA in 1991 (DH, 1991). SACN's energy reference values are 11% to 22% lower compared with the COMA 1991 values. Although some of the variance can be explained by slight differences in the body weights used to calculate values in the 2 reports, it is principally due to the different methodologies employed by the committees to calculate the energy reference values.
- 3.6 In 2011, SACN calculated energy reference values for children aged 1 to 18 years using a factorial approach which assumes that habitual total energy expenditure (TEE) is representative of energy requirements (EAR) and based on the assumption that TEE (or EAR) is equal to basal metabolic rate (BMR) x physical activity level (PAL). In children, an allowance for the energy needed for growth is also applied when calculating requirements. TEE values were based on a dataset of all published doubly labelled water (DLW) studies of children aged over one year; DLW is considered to be the most accurate method of measuring TEE in free-living people (SACN, 2011a). For all studies that did not report BMR, BMR values were estimated using the Henry equations (Henry, 2005) and PAL values were then derived from TEE and BMR.
- 3.7 In contrast, and in the absence of sufficient TEE data for children aged 1 to 10 years, COMA based its reference values on dietary energy intake data.

**Table 3.1. SACN energy reference values for children aged 1 to 6 years compared with values reported by COMA (1991)**

Age	COMA (1991) <sup>1</sup>	COMA (1991) <sup>1</sup>	SACN (2011)	SACN (2011)	Change (%)	Change (%)
	Boys (MJ per day)	Girls (MJ per day)	Boys (MJ per day)	Girls (MJ per day)	Boys (MJ per day)	Girls (MJ per day)
1 to 3 years	5.2	4.9	4.1	3.8	-20	-22
4 to 6 years	7.2	6.5	6.2	5.8	-14	-11

<sup>1</sup> Source: (DH, 1991).

## TDEI and BMI in young children in the UK

- 3.8 TDEI in children aged 12 to 60 months from the Diet and Nutrition Survey of Infants and Young Children (DNSIYC) and the National Diet and Nutrition Survey (NDNS) (years 2008 to 2019) are presented in Table 3.2.

**Table 3.2. TDEI of children aged 12 to 60 months in the UK (DNSIYC and NDNS years 2008 to 2019)<sup>1</sup>**

Age	EAR (MJ per day)	EAR (MJ per day)	Energy intake (MJ per day) <sup>2</sup> mean (SD)	Energy intake (MJ per day) <sup>2</sup> mean (SD)	% participants above EAR Boys	% participants above EAR Girls
	Boys	Girls	Boys	Girls		
12 to 18 months	3.2	3.0	4.2 (0.9)	4.0 (0.9)	88	88
18 to 23 months	3.2	3.0	4.5 (0.9)	4.1 (0.8)	96	87
24 to 35 months	4.2	3.9	4.7 (1.0)	4.4 (0.9)	69	69
36 to 47 months	4.9	4.5	4.9 (1.0)	4.8 (1.2)	47	58
48 to 60 months	5.8	5.4	5.7 (1.1)	5.1 (1.1)	43	37

Abbreviations: EAR, energy average requirement; MJ, megajoule; SD, standard deviation.

<sup>1</sup> Data source: DNSIYC 2011 (Lennox et al, 2013) for children aged 12 to 18 months and from NDNS years 2008 to 2019 for children aged 18 to 60 months.

<sup>2</sup> Number of participants in each age group for energy intake: 641 boys and 634 girls (12 to 18 months); 141 boys and 129 girls (18 to 23 months); 299 boys and 255 girls (24 to 35 months); 277 boys and 244 girls (36 to 47 months); 235 boys and 219 girls (48 to 60 months).

- 3.9 The dietary surveys indicated that approximately 90% of children aged 12 to 24 months and 70% of children aged 24 to 35 months had reported TDEI above the EAR for dietary energy. By age 36 to 47 months, approximately half of children had reported intakes above the EAR. By age 48 to 60 months less than half of children had reported intakes above the EAR. However, some caution should be taken when interpreting these findings given known problems with underreporting of dietary energy intake in dietary surveys (chapter 2, paragraph 2.78).
- 3.10 Time trend analysis of NDNS data (years 2008 to 2019) in children aged 18 to 36 months indicated a decrease in TDEI of  $-10$  kcal/day per year (95% CI  $-16$  to  $-0.5$  kcal/day/year) for the 11-year period (Bates et al, 2020). No time trend data were available for the other age groups.
- 3.11 At the same time, the latest available findings from child measurement programmes in England and Scotland for the school year 2021 to 2022 indicated

that the prevalence of overweight and obesity combined in children aged 4 to 5 years was 22.3% and 24.1%, respectively. The prevalence of obesity in England and Scotland (at 10.1% and 11.7%, respectively) decreased from that in the school year 2020 to 2021 when measurements were taken during the beginning of the COVID-19 pandemic but remained higher than before the pandemic. In Wales, limited data from the school year 2020 to 2021 also indicated that the prevalence of obesity (approximately 18%) had increased compared with the pre-pandemic school year of 2018 to 2019 (no comparable data are available for Northern Ireland). Data from these measurement programmes also indicated that deprivation is a major risk factor for obesity in childhood (see chapter 8 for details).

## TDEI, body weight and deprivation

- 3.12 TDEI by index of multiple deprivation (IMD), a broad measure of deprivation (see Glossary), in children aged 18 to 60 months from NDNS (years 2008 to 2019) is presented in Table 3.3. Children’s body weight (kg) by IMD for the same age group is presented in Table 3.4.

**Table 3.3. TDEI by IMD quintile in children aged 18 to 60 months in England (NDNS years 2008 to 2019)<sup>1</sup>**

Energy (MJ/day)	IMD quintile 1 (least deprived)	IMD quintile 2	IMD quintile 3	IMD quintile 4	IMD quintile 5 (most deprived)
Mean (95%CI)	4.90 (4.78 to 5.03)	4.78 (4.66 to 4.89)	4.91 (4.79 to 5.04)	4.83 (4.70 to 4.95)	4.67 (4.54 to 4.80)
Number of participants	210	211	182	234	277

Abbreviations: IMD, index of multiple deprivation; MJ, megajoule; TDEI, total dietary energy intake

<sup>1</sup> Data from NDNS years 2008 to 2019.

**Table 3.4. Body weight by IMD quintile in children aged 18 to 60 months in England (NDNS years 2008 to 2019)<sup>1</sup>**

Age	Body weight (kg)	IMD quintile 1 (least deprived)	IMD quintile 2	IMD quintile 3	IMD quintile 4	IMD quintile 5 (most deprived)
18 to 47 months	Mean (95%CI)	14.8 (14.3, 15.2)	14.1 (13.7, 14.5)	14.6 (14.1, 15.1)	14.9 (14.3, 15.5)	14.5 (14.1, 14.8)
18 to 47 months	Number of participants	136	148	120	148	178
48 to 60 months	Mean (95%CI)	17.9 (17.3, 18.4)	18.9 (18.0, 19.9)	18.8 (17.8, 19.7)	18.1 (17.5, 18.8)	18.3 (17.7, 19.0)
48 to 60 months	Number of participants	55	47	46	64	64

Abbreviations: IMD, index of multiple deprivation.

<sup>1</sup> Data from NDNS years 2008 to 2019.

- 3.13 There was no clear relationship (indicated by overlapping confidence intervals) between TDEI and IMD or body weight and IMD. However, caution should be taken when interpreting the data due to the small number of participants included in the analyses, particularly in relation to body weight.
- 3.14 In contrast, child measurement programmes in England (the National Child Measurement Programme) and Scotland (the Child Health Surveillance Programme School system) indicated a strong direct relationship between increasing deprivation and higher obesity prevalence (see chapter 8 for details).

## **Main dietary sources of energy**

- 3.15 Main dietary sources of energy in children aged 12 to 60 months are presented in Table 3.5. Milk (excluding formula milks) was the largest source of TDEI at age 12 to 18 months (19% TDEI) followed by formula milks (10% TDEI). Milk still provided 10% TDEI at ages 48 to 60 months.
- 3.16 Cereals and cereal products were also an important source of energy: bread provided 7% TDEI in children aged 12 to 18 months and 10% in the older groups. Breakfast cereals provided 5% TDEI in children aged 12 to 18 months and 6% TDEI in the older groups. Pizza, rice and pasta provided 6% TDEI in the youngest age group and 7% to 8% in the older groups.
- 3.17 Commercially manufactured foods and drinks marketed specifically for children aged up to 36 months provided a greater proportion of TDEI in the youngest age

group (6%) compared with older age groups (1%) (for details see [Foods and drinks specifically marketed for infants and young children](#) in chapter 5).

- 3.18 Biscuits, buns, cakes, pastries, fruit pies and puddings provided 6% TDEI, 11% TDEI and 13% TDEI in children aged 12 to 18 months, 18 to 47 months and 48 to 60 months, respectively.
- 3.19 For children aged 12 to 18 months, meat, meat products and dishes provided 8% TDEI while 6% TDEI was provided by fruit.
- 3.20 For children aged 18 to 47 months, meat, meat products and dishes provided 11% TDEI while 3% to 6% TDEI was provided by fruit, potatoes, products and dishes; sugar, preserves and confectionery; yoghurt, fromage frais and dairy desserts; cheese, crisps and savoury snacks; and vegetables, products and dishes.
- 3.21 For children aged 48 to 60 months, meat, meat products and dishes provided 13% TDEI; fruit provided 6% TDEI; sugar, preserves and confectionery provided 5% TDEI; and potatoes, products and dishes also provided 5% TDEI.

**Table 3.5. Contribution (% TDEI) of food groups (food sources) to average daily TDEI (MJ per day) in children aged 12 to 60 months (DNSIYC and NDNS years 2016 to 2019). Population average including non-consumers.**

Food groups <sup>2,3,4</sup>	12 to 18 months <sup>1</sup>	12 to 18 months <sup>1</sup>	18 to 47 months	18 to 47 months	48 to 60 months	48 to 60 months
	%	kcal	%	kcal	%	kcal
Milk <sup>5</sup>	19.0	187	15.0	159	10.1	125
Formula milks <sup>6</sup>	9.8	90	1.1	11	0	0
Meat, meat products and dishes	7.7	76	10.7	112	13.2	160
Bread	6.9	68	9.8	104	9.8	120
Commercially manufactured foods and drinks marketed specifically for infants and young children	6.2	58	1.0	11	0.7	8
Biscuits, buns, cakes, pastries, fruit pies, puddings	6.1	60	10.6	114	12.7	156
Pizza, pasta, rice, products and dishes	5.5	53	6.8	71	7.5	87
Fruit	5.7	56	5.6	60	5.8	69
Yoghurt, fromage, fraises and dairy desserts <sup>7</sup>	5.1	49	3.5	38	2.9	35
Breakfast cereals	5.1	49	6.2	63	6.0	76
Potatoes, potato products and dishes	3.8	37	4.2	45	5.0	63
Vegetables, vegetable products and dishes	2.7	26	2.8	29	3.2	38
Butter and fat spreads	2.5	25	2.9	32	3.3	41
Cheese <sup>7</sup>	2.5	25	3.2	33	2.2	27
Fish, fish products and dishes	2.0	19	2.4	25	1.8	22
Sugar, preserves and confectionery	2.0	19	3.6	38	5.1	64
Breast milk	1.8	16	0	0	0	0
Eggs, egg products and dishes	1.4	13	1.8	19	1.5	18
Crisps and savoury snacks	1.2	11	3.0	32	2.8	35
Soup	0.7	6	0.8	8	0.4	6
Savoury sauces, pickles, gravies and condiments	0.6	6	0.9	9	1.1	13
Fruit juice and smoothies	0.5	5	1.3	14	1.0	12
Sugar-sweetened beverages <sup>8</sup>	0.4	4	0.4	4	0.5	7.7
Ice cream <sup>7</sup>	0.4	4	1.0	11	2.0	25
Nuts and seeds	0.1	1	0.6	7	0.7	9
Number of participants	1275	1275	354	354	114	114

Abbreviations: TDEI, total dietary energy intake.

<sup>1</sup> Data in children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013) otherwise data from NDNS years 2016 to 2019.

<sup>2</sup> Food groups are ordered by largest to smallest % contribution in the youngest age group.

<sup>3</sup> Food groups that contribute less than 0.5% of energy intake in all age groups are not presented.

<sup>4</sup> Average % contribution for each food group has been calculated from the % contribution for each individual. Non-consumers are included in the average.

<sup>5</sup> Milk includes cream and non-dairy alternatives to milk.

<sup>6</sup> Formula milks include milks marketed to children over the age of 1 year, also known as 'toddler milks' and 'growing-up milks' (see Glossary).

<sup>7</sup> Includes non-dairy alternatives

<sup>8</sup> Includes carbonated drinks, concentrates and ready to drink products with added sugars.

## Limitations of the survey evidence

- 3.22 Misreporting, and specifically underreporting, of food consumption and therefore underestimation of total dietary energy intake (TDEI) in self-reported dietary methods is a well-documented source of bias and is an important consideration when interpreting NDNS data. The NDNS rolling programme is one of the few national large-scale population surveys to include doubly labelled water (DLW) as an objective biomarker to validate energy intake estimated from reported food and drink consumption (see chapter 3, paragraph 3.6, for more details). However, the latest available DLW sub-study of the NDNS rolling programme (collection years 2013 to 2015) only included children aged 4 years and older. For details on how potential misreporting of TDEI in the absence of DLW data in all the age groups was investigated for this report and its implications, see [Limitations of the evidence on micronutrients](#) in chapter 4.

## Systematic review evidence identified on dietary energy intake and health

- 3.23 Three systematic reviews (SRs) without meta-analyses (MAs) (Dougkas et al, 2019; Parsons et al, 1999; Rouhani et al, 2016) were identified that included studies that examined the relationship between dietary energy intake (TDEI or energy intake from certain foods or drinks) or the energy density of the whole diet and body composition (BMI or body fat). An additional 3 SRs without MAs (Mikkelsen et al, 2014; Osei-Assibey et al, 2012; Ward et al, 2015) were identified that included studies that examined the impact of portion sizes on children's food or energy intake.
- 3.24 Details of the 6 SRs included in this section can be found in Annex 5 (Tables A5.1, A5.3 and A5.5). Quality assessment of the SRs using the AMSTAR 2 tool can be found in Annex 8 (Tables A8.2, A8.4 and A8.8). Additional data extracted on the primary studies can be found in Annex 9 (Table A9.1).
- 3.25 The criteria used to grade the evidence are provided in chapter 2 (Table 2.4, paragraphs 2.54 to 2.59). Summary tables of the evidence grading process for this section are provided in Annex 10 (Tables A10.1, A10.2 and A10.36).

## Limitations of the systematic review evidence on dietary energy intake

- 3.26 Of the 3 SRs included in this section (Dougkas et al, 2019; Parsons et al, 1999; Rouhani et al, 2016), only 1 SR (Rouhani et al, 2016) sought to address the relationship between dietary energy intake or density and body composition. For

the other 2 SRs, TDEI or energy intake or density was neither an exposure nor included in the search terms. Therefore, their literature searches were unlikely comprehensive for this topic area, which is a potential source of bias.

- 3.27 Most of the primary studies included in the 6 SRs were small and may not have been adequately powered to sufficiently examine the relationship between dietary energy intake or energy density and obesity outcomes. As children grow at different rates, studies of energy intake in young children need to be large enough to accommodate the full range of body sizes and ages, and to adjust for these.
- 3.28 Primary studies did not always adjust for baseline body size and physical activity (after accounting for sex and age), which are key factors associated with differences in TDEI among individuals (Willett et al, 1997).
- 3.29 Many of the studies were conducted in the 1990s indicating a need for more current research in this area.

## Effect of portion sizes on food or energy intake

- 3.30 Three SRs (Mikkelsen et al, 2014; Osei-Assibey et al, 2012; Ward et al, 2015) examined whether reducing portion sizes of meals and snack foods in preschool settings could be an effective strategy to reduce children's food and energy intake. All of the primary studies included in the SRs were short term (<6 months). None examined the effect of manipulating portion sizes on children's weight status even though 2 of the 3 SRs (Mikkelsen et al, 2014; Osei-Assibey et al, 2012) sought to examine strategies designed to prevent weight gain or obesity in preschool and school-aged children.
- 3.31 Ward et al (2015) (AMSTAR confidence rating: moderate) included a pre-post study (see Glossary) (in 40 participants, preschool age not defined) that reported that children's intake of snack foods (during a designated snack time) was greater when teachers enabled the children to select how much food they could eat compared with when the children were offered a standard portion of the snack food (mean difference in portions of snack food eaten: 0.87;  $p < 0.01$ ).
- 3.32 Osei-Assibey et al (2012) (AMSTAR confidence rating: low) included 2 within-subject crossover studies (see Glossary) and 1 non-randomised controlled trial (non-RCT) that all reported an increase in food or energy intake when children were offered larger portions compared with when offered smaller portions. One within-subject crossover study (in 35 participants, aged 2 to 5 years) reported that doubling an age-appropriate portion size of macaroni and cheese served as part of a school lunch increased intake (g) by 25% ( $\pm$  SEM 7%;  $p < 0.001$ ) and energy intake (kcal) by 15% ( $\pm$  SEM 5%;  $p < 0.01$ ). The other within-subject crossover study (in 17 participants, aged 3 to 5 years) reported that children offered a larger portion of snack foods consumed more energy than when offered a smaller portion (energy intake 99.0 kcal for large portion; 84.2 kcal for small portion;  $p < 0.05$ ). The non-RCT (in 32 participants, aged 3 to 6 years) also reported that children increased their energy intake when served larger portions of food at lunchtime compared with when served smaller portions. However, this effect was only seen in the older children (aged 4 to 6 years, mean age 5 years) (effect size not reported [NR];  $p < 0.002$ ).
- 3.33 Mikkelsen et al (2014) (AMSTAR confidence rating: low) included 2 quasi-experimental studies in children aged 1 to 5 years. One study (in 235 participants, aged 2 to 7 years) reported that when children were served a standard portion of food (chicken nuggets) during a school lunch, their intake was greater than when they were offered the choice to select from a number of smaller portions (quantitative findings NR). However, food intake was measured at a canteen level rather than at an individual level. The other study (in 77 participants, aged 2 to 5 years) reported that reducing the energy density of a dish (macaroni and cheese)

served as part of a school lunch by 30% reduced children's energy intake from the dish by 25% and total lunch energy intake by 18%, even though children consumed more of the lower energy version of the dish than the regular version (quantitative findings NR).

## **Dietary energy intake and BMI**

- 3.34 Rouhani et al (2016) (AMSTAR 2 confidence rating: critically low) included 1 prospective cohort study (PCS) that examined the relationship between the consumption of energy dense foods (EDF) and BMI in children aged 1 to 5 years. EDF included sugar-sweetened beverages (SSBs, see Glossary), crisps, hamburgers, pizzas, cakes, chocolate and sweets. The PCS (in 589 participants) reported no association between consumption of EDF (average daily frequency of consumption) at age 2 years and BMI z-score at age 4 years (quantitative findings NR). The study adjusted for children's age at baseline and maternal age, pre-pregnancy BMI and education.
- 3.35 Parsons et al (1999) (AMSTAR 2 confidence rating: critically low) included 3 PCS (reported in 4 publications) that examined the relationship between energy intake (presumed TDEI) and BMI or change in BMI over time in children aged 1 to 5 years. Results from the 3 PCS were conflicting.
- 3.36 One PCS (in 146 participants) reported no association between TDEI at ages 3 to 5 years and change in BMI 2 years later (quantitative findings NR). The study adjusted for sex, age, baseline BMI, physical activity and parental weight status.
- 3.37 A second PCS (in 112 participants) reported that higher TDEI at age 2 years was associated with higher BMI at age 8 years ( $r=0.20$ ;  $p=0.049$ ). The relationship remained after adjusting for socioeconomic status (SES) ( $r=0.20$ ;  $p=0.044$ ). Additional analysis on data from the same cohort of participants showed that the increase in daily TDEI between ages 4 to 6 years was greatest in the children in the highest tertile for BMI at age 8 years compared with children in the other 2 tertiles for BMI ( $p=0.01$ ). However, there was no association between daily TDEI before age 4 years and BMI at age 8 years. The analysis was not adjusted for potential confounding factors and the study had a low participant retention rate (40%), which is a potential source of bias.
- 3.38 The third PCS (in 37 participants), with the longest follow-up duration, reported that TDEI (per kg of body weight) at ages 3 to 4 years was inversely correlated with BMI at age 15 years in girls only (correlation coefficient  $-0.73$ ;  $p<0.0118$ ; 10 participants). The study was small and did not adjust for potential confounding factors.

## Dietary energy intake and body fat

- 3.39 Dougkas et al (2019) (AMSTAR 2 confidence rating: low) included 1 PCS that examined the relationship between energy intake from milk (unspecified) in children aged 1 to 5 years and body fat. The PCS (in 49 participants) reported that a greater increase in energy consumed from milk at ages 3 to 5 years was associated with a 0.01cm (SE 0.004) decrease in waist circumference measured 3 years later (p=0.04). The study adjusted for TDEI at age 3 years and change in waist circumference from ages 3 to 5 years.
- 3.40 Parsons et al (1999) (AMSTAR 2 confidence rating: critically low) included an additional PCS (in 37 participants) in children aged 1 to 5 years, which reported that TDEI (per kg of body weight) at ages 3 to 4 years was inversely associated with body fat mass index at age 15 years in girls only (correlation coefficient -0.77; p<0.009; 10 participants). The study was small and did not adjust for potential confounding factors.

## Summary: dietary energy intake and body composition

- 3.41 The evidence identified from SRs on dietary energy intake and body composition is summarised in Table 3.6.

**Table 3.6. Summary of the evidence on dietary energy intake and obesity outcomes**

Exposure	Outcome	Direction of effect or association <sup>1</sup>	Certainty of evidence
Portion sizes	Food and energy intake (in the short term less than 6 months)	↑	Moderate
Dietary energy intake or energy density of the whole diet	Body Mass Index (BMI)	Inconsistent	Insufficient
Dietary energy intake or energy density of the whole diet	Body fat	Not applicable	Insufficient

<sup>1</sup> Direction of association for reported outcomes: ↑increase

- 3.42 The available evidence from SRs on dietary energy intake in children aged 1 to 5 years is from 6 SRs without MAs, 1 given a moderate confidence rating, 3 given a

low confidence rating and 2 given a critically-low confidence rating using the AMSTAR 2 tool.

- 3.43 Evidence from 6 intervention studies included in 3 SRs by Ward et al (2015), Mikkelsen et al (2014) and Osei-Assibey et al (2012) suggests that larger portion sizes of snacks and meals provided in preschool settings are associated with higher food and energy intake (in grams or energy) in the short term (less than 6 months). The evidence was graded 'moderate' rather than 'adequate' due to the non-randomised design of the studies, small sample sizes, lack of reported confidence intervals, and lack of information on study power, publication bias and confounding. No evidence from SRs was identified on the longer-term impact on TDEI from increasing portion sizes or the impact of increasing portion sizes on children's body composition or weight status.
- 3.44 Evidence from 4 PCS included in the SRs by Rouhani et al (2016) and Parsons et al (1999) on the association between TDEI in children aged 1 to 5 years and BMI in childhood and adolescence was inconsistent. The evidence was graded 'insufficient' due to the poor quality of the SRs, small sample sizes of the PCS and inadequate accounting for confounding factors. In addition, as Parsons et al (1999) did not include search terms for dietary energy intake in its search strategy, its literature search was unlikely to be comprehensive for dietary energy intake as an exposure, which is a potential source of bias.
- 3.45 There was 'insufficient' evidence from SRs to enable conclusions to be drawn on the relationship between dietary energy intake and body fat measures in children aged 1 to 5 years as there were fewer than 3 primary studies included in the SRs that examined this relationship.

# Macronutrients

## Background

- 3.46 The energy and nutrient density of the diet, including the balance of macronutrients (and micronutrients) need to be considered as the diets of young children are further diversified beyond 1 year of age.
- 3.47 Macronutrients (carbohydrate, dietary fat and protein) contribute to an individual's dietary energy intake (SACN, 2011a). Individuals who consume greater amounts of any one macronutrient are likely to also consume a greater amount of food and drink and therefore energy. The major factors that are associated with differences in energy requirements and thus intakes among individuals are differences in body size and physical activity (SACN, 2011a).
- 3.48 In RCTs, diets are often designed to examine the health effects of single macronutrients without changing the total energy content (that is, isoenergetic diets) by substituting the macronutrient of interest for other sources of energy (other macronutrients) (Willett et al, 1997). In observational studies, the principal means of separating out the health effects of a specific macronutrient is to statistically correct for its possible effect through its contribution to TDEI.
- 3.49 However, it may be informative to consider the health effects of a specific macronutrient both with and without correction for the effects of TDEI (Tomova et al, 2022). Epidemiological studies have suggested that higher TDEI may mediate the effects of sugar-sweetened beverages (SSBs) (see Glossary) and fruit juice on obesity and related cardiometabolic outcomes (Crowe-White et al, 2016; Malik & Hu, 2011). Studies that did not adjust for TDEI tended to report stronger associations than those that did (Malik & Hu, 2011), implying that adjusting for TDEI removes any effects that are mediated by energy intake. Evidence from SACN's report 'Carbohydrates and Health' indicated that children do not adequately compensate for the energy they consume from SSBs by reducing energy consumption from foods (SACN, 2015).
- 3.50 Therefore, this report considered findings that were adjusted for TDEI separately from findings not adjusted for TDEI, when data was available.

## Limitations of the systematic review evidence on macronutrients

- 3.51 The primary studies included in the SRs identified for this section were highly heterogeneous in their methods and approaches. Macronutrient intakes were either expressed as absolute amounts (grams per day) or as a proportion of TDEI

(% TDEI) and there was no standard definition for 'low' or 'high' intakes of dietary fat, protein or carbohydrates.

- 3.52 Many SRs did not distinguish between or discuss the implications of findings from primary studies that adjusted for TDEI against those that did not (see paragraphs 3.48 and 3.49).
- 3.53 Primary studies did not always adequately account for children's body size at baseline. A child who is larger at baseline may consume more food and drink (and more energy overall) than a smaller child. Therefore, the possibility of reverse causation, where body size drives food and drink consumption rather than the other way around, cannot be ruled out. The impact of intakes of different macronutrients on health outcomes may also differ in children with healthy weight at baseline versus children living with overweight or obesity.
- 3.54 Other potential confounding factors that were not always accounted for by SRs or primary studies when outcomes related to energy balance included physical activity levels, parental weight status and SES.
- 3.55 Many primary studies did not report power calculations. Findings of null associations with wide confidence intervals should therefore be interpreted with caution.

# Carbohydrates

## Classification of carbohydrates

- 3.56 Carbohydrates are a major source of energy in the diet and include a range of compounds, all containing carbon, hydrogen and oxygen. The primary classification of carbohydrates is based on chemistry, that is, the character of individual monomers, degree of polymerisation (DP) and type of linkage ( $\alpha$  or  $\beta$ ) (FAO and WHO, 1998). This classification divides carbohydrates into 3 main groups: sugars, including mono- and disaccharides (DP 1 to 2); oligosaccharides (DP 3 to 9); and polysaccharides (DP >9).
- 3.57 The 3 principal monosaccharides, glucose, fructose and galactose, are the building blocks of di-, oligo-, and polysaccharides. These hexoses (6-carbon sugars) can be found in honey and fruits (the disaccharide sucrose, made up of glucose and fructose units, is also found in fruits). Galactose in combination with glucose is found in milk as lactose. Polyols (also known as sugar alcohols) include hydrogenated mono- and disaccharides used as sugar replacers. Oligosaccharides are also widely used in the food industry to modify the texture of food products. Starch is a polysaccharide of glucose monomers and is the principal carbohydrate in most diets.
- 3.58 Dietary fibre includes constituents of plant cell walls, such as cellulose, and is the most diverse of the carbohydrate groups. The SACN report '[Carbohydrates and health](#)' (SACN, 2015) defines dietary fibre as all carbohydrates that are neither digested nor absorbed in the small intestine and have a degree of polymerisation of 3 or more monomeric units, plus lignin.
- 3.59 The chemical classification of carbohydrates does not allow a simple translation into nutritional effects since each class of carbohydrates has overlapping physiological properties and effects on health.
- 3.60 Carbohydrates can also be classified according to their digestion and absorption in the small intestine. Digestible carbohydrates are absorbed and digested in the small intestine. Non-digestible carbohydrates are resistant to hydrolysis in the small intestine and reach the large intestine where they are at least partially fermented by bacteria present in the large intestine.
- 3.61 The following terms are used in this report to describe carbohydrates:
- free sugars — all added sugars in any form; all sugars naturally present in fruit and vegetable juices, concentrates, smoothies, purées and pastes, powders, extruded fruit and vegetable products and similar products in which the structure has been broken down; all sugars in drinks (except for dairy-based drinks); and lactose and galactose added as ingredients. This definition excludes sugars naturally present in milk and dairy products, fresh and most

types of processed fruit and vegetables, and in cereal grains, nuts and seeds (Swan et al, 2018).

- starch — polymer of glucose, found in foods such as rice, bread, pasta and potatoes
- dietary fibre — defined in paragraph 3.58.

3.62 For more details on carbohydrates, please refer to the SACN reports on [‘Carbohydrates and Health’](#) (SACN, 2015) and [‘Lower carbohydrate diets for adults with type 2 diabetes’](#) (SACN, 2021).

## **Current recommendations for carbohydrate intake in the UK**

3.63 In its report [‘Carbohydrates and health’](#) (SACN, 2015), SACN evaluated evidence assessing whether intakes of specific carbohydrates are a factor in the risk for developing cardiovascular disease, obesity, type 2 diabetes mellitus and colorectal cancers. Based on the evidence, dietary recommendations were made for total carbohydrates, free sugars, starch and sugars contained within the cellular structure of food, milk sugars, and dietary fibre in the context of an energy intake that is appropriate to maintain a healthy weight (SACN, 2015).

3.64 For children aged 2 years and older, SACN recommended that:

- total carbohydrate intake should be maintained at a population average of approximately 50% TDEI
- the population average intake of free sugars should not exceed 5% TDEI
- sugar-sweetened beverages (SSBs) (see Glossary) should be minimised
- the average population intake of dietary fibre for children aged 2 to 5 years should approximate 15g per day.

3.65 No recommendations were made for carbohydrate intake for children aged under 2 years due to the absence of evidence in this age group. However, from about age 6 months, gradual diversification of the diet to provide increasing amounts of whole grains, pulses, vegetables and fruit was encouraged (SACN, 2015).

3.66 Table 3.7 shows the dietary reference values (DRVs) for total carbohydrates, free sugars and dietary fibre for children aged 2 to 6 years converted into daily gram amounts using the TDEI values for this age group from SACN (2011a). Data for children aged 1 year were not available.

**Table 3.7. Dietary Reference Values (DRVs) for total carbohydrates, free sugars and dietary fibre for children aged 2 to 6 years in grams per day**

Type of carbohydrate	Boys aged 2 to 3 years	Girls aged 2 to 3 years	Boys aged 4 to 6 years	Girls aged 4 to 6 years
Total carbohydrates (grams per day) <sup>1</sup> [at least]	145	134	198	184
Free sugars (grams per day) <sup>2</sup> [less than]	15	13	20	18
Dietary fibre (grams per day) [at least]	15	15	15 <sup>3</sup> 20 <sup>4</sup>	15 <sup>3</sup> 20 <sup>4</sup>

Source: PHE (2016a).

<sup>1</sup> Calculated using the energy figures from (SACN, 2011a). The % for which to calculate grams of total carbohydrate per day (50% TDEI) was obtained from (SACN, 2015).

<sup>2</sup> Calculated using the energy figures from (SACN, 2011a). The % for which to calculate grams of free sugars per day (5% food energy) was recommended in (SACN, 2015).

<sup>3</sup> Applies at age 4 years.

<sup>4</sup> Applies at age 5 to 6 years.

## Carbohydrate intakes in the UK

3.67 Total carbohydrate intake in children in the UK aged 12 to 60 months from DNSIYC and NDNS (years 2016 to 2019) are presented in Table 3.8. Mean intake ranged from 49.0% TDEI in the 12 to 18 months age group to 51.3% TDEI in the 48 to 60 months age group. It should be noted that the DRV of 50% TDEI applies to children aged 2 years (24 months) and over.

**Table 3.8. Total carbohydrate intake in children aged 12 to 60 months in the UK (DNSIYC and NDNS years 2016 to 2019)<sup>1</sup>**

Age	Grams per day Mean (SD)	% TDEI <sup>2</sup> Mean (SD)	Number of participants
12 to 18 months	126 (29)	49.0 (5.8)	1275
18 to 47 months	138 (36)	49.1 (5.9)	306
48 to 60 months	168 (44)	51.3 (5.4)	102

Abbreviations: TDEI, total dietary energy intake; SD, standard deviation.

<sup>1</sup> Data in children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013) otherwise data from NDNS years 2016 to 2019 (Bates et al, 2020).

<sup>2</sup> TDEI, total dietary energy intake. Total energy is equivalent to food energy as no alcohol is consumed by children of this age.

- 3.68 Time trend analysis for the age group 18 to 36 months indicated a significant decrease in % TDEI from total carbohydrate of  $-0.2$  percentage points per year (95% CI  $0.3\%$  to  $0.0\%$ ) over an 11-year period (2008 to 2019) (Bates et al, 2020). No time trend data was available for the other age groups.
- 3.69 Free sugars intake in children aged 12 to 60 months is presented in Table 3.9. Free sugars intake for the 12 to 18 month age group was not originally reported in DNSIYC because the survey predated the definition of 'free sugars' (instead, the survey reported intakes of 'non-milk extrinsic sugars', see Glossary). For this report, intake of free sugars for the 12 to 18 month age group was calculated. Sugar intakes for the 2 older age groups (18 to 47 months; 48 to 60 months) were reported as 'free sugars' in the NDNS years 2016 to 2019.
- 3.70 Mean intake of free sugars was double the maximum recommendation of 5% TDEI in children aged 12 to 47 months and more than double the maximum recommendation in children aged 48 to 60 months. The vast majority of children in all age groups had intakes above the 5% recommendation.
- 3.71 Time trend analysis of NDNS data (years 2008 to 2019) in children aged 18 to 36 months indicated a decrease in the % TDEI from free sugars of  $-0.3$  percentage points per year (95% CI  $-0.4\%$  to  $-0.2\%$ ) for the 11-year period (Bates et al, 2020). No time trend data were available for the other age groups.

**Table 3.9. Free sugars intake in children aged 12 to 60 months in the UK (DNSIYC and NDNS years 2016 to 2019)<sup>1</sup>**

Age	Free sugars <sup>2</sup> grams per day <sup>4</sup>	Free sugars <sup>2</sup> % TDEI <sup>3,4</sup>	% participants exceeding DRV <sup>5</sup>	Number of participants
12 to 18 months	25.2 (data not available)	9.9 (data not available)	80	1275
18 to 47 months	27.9 (15.8)	9.7 (4.6)	85	306
48 to 60 months	38.9 (19.3)	11.7 (4.6)	97	102

Abbreviations: DRV, dietary reference value; NMES, non-milk extrinsic sugars; SD, standard deviation; TDEI, total dietary energy intake.

<sup>1</sup> Data in children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013) otherwise data from NDNS years 2016 to 2019 (Bates et al, 2020).

<sup>2</sup> Free sugars intake for the age group 12 to 18 months was not originally reported in DNSIYC but have been calculated for this report. Sugar intakes for the 2 older age groups (18 to 47 months; 48 to 60 months) were reported as free sugars in the NDNS years 2016 to 2019.

<sup>3</sup> TDEI is equivalent to food energy as no alcohol is consumed by children of this age.

<sup>4</sup> Mean (SD).

<sup>5</sup> DRV:  $\leq 5\%$  total dietary energy. The DRVs for free sugars and fibre apply to children from the age of 2 years. However, for the purposes of reporting the age group 1.5 to 3 years, the recommendation has been applied to the whole group, including those aged under 2 years. The DRV for free sugars has been applied to free sugars intake in the 12 to 18 month age group for illustrative purposes.

- 3.72 Dietary fibre intake in children aged 12 to 60 months is presented in Table 3.10. NDNS (years 2016 to 2019) used the definition of dietary fibre recommended by SACN (2015)(see paragraph 3.58) that is chemically determined using prevailing Association of Official Agricultural Chemists (AOAC) methods. DNSIYC used a narrower definition of dietary fibre (non-starch polysaccharides [NSP]) that predated SACN (2015) (DH, 1991; DH, 1994b).
- 3.73 Mean intake of AOAC fibre was 10.4 grams per day in children aged 18 to 47 months, and 12.6 grams per day in children aged 48 to 60 months. Eighty eight percent of children aged 18 to 47 months and 72% of children aged 48 to 60 months had fibre intakes below the DRV.
- 3.74 Time trend analysis of NDNS data (years 2008 to 2019) in children aged 18 to 36 months indicated no annual change in dietary fibre intake (0.0 percentage point change per year 95% CI -0.1 to 0.0) for the 11-year period (Bates et al, 2020). No time trend data were available for the other age groups.

**Table 3.10. Dietary fibre intake in children aged 12 to 60 months in the UK (DNSIYC and NDNS 2016 to 2019)<sup>1</sup>**

Age	NSP <sup>2,3</sup> grams per day	AOAC fibre <sup>2,3</sup> grams per day	% participants below DRV <sup>4</sup>	Number of participants
12 to 18 months	7.3 (2.7)	Not calculated	Not calculated	1275
18 to 47 months	Not calculated	10.4 (3.5)	88	306
48 to 60 months	Not calculated	12.6 (4.7)	72	102

Abbreviations: AOAC, Association of Official Agricultural Chemists; NSP, non-starch polysaccharides; SD, standard deviation.

<sup>1</sup> Data in children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013) otherwise data from NDNS years 2016 to 2019 (Bates et al, 2020).

<sup>2</sup> NSP comprise cellulose and non-cellulose polysaccharides (e.g. pectins, glucans, arabinogalactans, arabinoxylans, gums and mucilages) (DH, 1991; DH, 1994b). SACN (2015) recommended a broader definition of dietary fibre to include all carbohydrates that are neither digested nor absorbed in the small intestine and have a degree of polymerisation of 3 or more monomeric units, plus lignin. The broader definition of dietary fibre is measured by AOAC methods and is colloquially known as 'AOAC fibre'. AOAC fibre intakes are typically about a third higher than NSP intakes.

<sup>3</sup> Mean (SD).

<sup>4</sup> DRV: fibre intake should approximate 15g per day for children aged 2 to 5 years. The DRV for fibre applies to children from the age of 2 years. However, for the purposes of reporting the age group 1.5 to 3 years, the recommendation has been applied to the whole group, including those aged under 2 years.

## Carbohydrate intakes and deprivation

- 3.75 Intake of carbohydrates (by type) by IMD (see Glossary) in children aged 18 to 60 months from NDNS (years 2008 to 2019) are presented in Table 3.11. For total

carbohydrates and free sugars, there was no clear relationship between intake and IMD (as indicated by overlapping confidence intervals).

- 3.76 Dietary fibre intake was lowest (10.3 grams per day) in quintile 5 (most deprived) and highest (11.7 grams per day) in quintile 1 (least deprived). The confidence intervals indicate that dietary fibre intake was significantly higher in quintiles 1, 2 and 3 compared with quintile 5.

**Table 3.11. Carbohydrate intakes by IMD quintile in children aged 18 to 60 months in England (NDNS years 2008 to 2019)<sup>1</sup>**

Intakes Mean (95% CI)	IMD quintile 1 (least deprived)	IMD quintile 2	IMD quintile 3	IMD quintile 4	IMD quintile 5 (most deprived)
Total carbohydrate % TDEI <sup>2</sup>	51.3 (50.6 to 52.0)	50.1 (49.4 to 50.8)	50.6 (49.9 to 51.2)	50.7 (50.1 to 51.3)	50.3 (49.7 to 50.9)
Free sugars % TDEI <sup>2</sup>	12.4 (11.7 to 13.2)	11.6 (10.9 to 12.2)	11.9 (11.2 to 12.5)	12.1 (11.5 to 12.6)	11.8 (11.2 to 12.3)
Dietary fibre grams per day	11.7 (11.3 to 12.1)	11.2 (10.8 to 11.6)	11.2 (10.8 to 11.6)	11.0 (10.6 to 11.4)	10.3 (9.9 to 10.7)
Number of participants	210	211	182	234	277

Abbreviations: IMD, index of multiple deprivation; TDEI, total dietary energy intake.

<sup>1</sup> Data from NDNS 2008 to 2019 (Bates et al, 2020).

<sup>2</sup> TDEI is equivalent to food energy as no alcohol is consumed by children of this age.

## Main dietary sources of carbohydrates

### Total carbohydrates

- 3.77 The main dietary sources of carbohydrates in children aged 12 to 60 months are presented in Table 3.12. Milk, bread and fruit were the largest contributors to carbohydrate intake in children aged 12 to 18 months, while bread, and biscuits, buns, cakes, pastries, fruit pies and puddings were the largest contributors to carbohydrate intake in children aged 18 to 60 months.

**Table 3.12. Food group contributors to average total carbohydrate intake in children aged 12 to 60 months (DNSIYC and NDNS years 2016 to 2019)<sup>1</sup>.**

**Population average including non-consumers**

Contribution of food groups <sup>2,3,4</sup> to total carbohydrate intake	12 to 18 months %	12 to 18 months grams per day	18 to 47 months %	18 to 47 months grams per day	48 to 60 months %	48 to 60 months grams per day
Milk <sup>5</sup>	11.5	13.9	9.1	12.0	6.9	11.1
Bread	10.8	13.6	14.7	20.5	14.3	23.9
Fruit	10.4	13.4	9.9	14.0	9.9	16.5
Formula milks <sup>6</sup>	9.1	11.4	1.1	1.4	0.0	0.0
Commercially manufactured foods and drinks specifically marketed for infants and young children	8.3	10.5	1.4	1.9	0.9	1.5
Breakfast cereals	7.9	9.9	9.2	12.3	8.8	15.1
Pizza, pasta, rice, products and dishes	7.8	10.0	9.2	12.7	9.5	15.5
Biscuits, buns, cakes, pastries, fruit pies, puddings	7.3	9.3	12.2	17.5	13.7	23.2
Yoghurt, fromage frais and dairy desserts <sup>5</sup>	5.5	6.9	3.8	5.3	3.0	5.1
Potatoes, potato products and dishes	5.4	6.8	5.8	7.8	6.2	10.5
Vegetables, vegetable products and dishes	3.0	3.8	3.0	4.2	3.4	5.7
Meat, meat products and dishes	3.0	3.8	4.3	5.8	5.0	8.1
Sugar, preserves and confectionery	2.3	3.0	4.4	6.2	6.5	11.2
Breast milk	1.5	1.7	0.0	0.0	0.0	0.0
Crisps and savoury snacks	1.1	1.4	2.9	4.0	2.6	4.4
Fish, fish products and dishes	1.1	1.3	1.4	1.9	0.9	1.5
Fruit juice and smoothies	1.0	1.3	2.4	3.6	1.8	3.2
Sugar-sweetened beverages <sup>7</sup>	0.8	1.1	0.7	1.1	1.0	2.0
Soup	0.6	0.8	0.7	0.8	0.4	0.8
Savoury sauces pickles gravies and condiments	0.4	0.6	0.6	0.8	0.9	1.5
Ice cream <sup>5</sup>	0.4	0.5	1.1	1.6	2.2	3.7
Low calorie soft drinks <sup>8</sup>	0.2	0.2	0.5	0.7	0.4	0.6
Cheese <sup>5</sup>	0.1	0.1	0.5	0.6	0.4	0.5
Number of participants	1275	1275	306	306	102	102

<sup>1</sup> Data in children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013) otherwise data from NDNS years 2016 to 2019 (Bates et al, 2020).

<sup>2</sup> Food groups are ordered by largest to smallest % contribution in the youngest age group.

<sup>3</sup> Food groups that contribute less than 0.5% of total carbohydrate intake in all age groups are not presented

<sup>4</sup> Average % contribution for each food group has been calculated from the % contribution for each individual. Non-consumers are included in the average.

<sup>5</sup> Includes dairy alternatives.

<sup>6</sup> Formula milks include milks marketed to children over the age of 1 year, also known as 'toddler milks' and 'growing-up milks' (see Glossary).

<sup>7</sup> Includes carbonated drinks, concentrates and ready to drink products with added sugars.

<sup>8</sup> Includes low calorie, diet, no added sugar, sugar-free drinks. Excludes mineral water.

## Free sugars

- 3.78 The main dietary sources of free sugars in children aged 12 to 60 months are presented in Table 3.13.
- 3.79 In children aged 12 to 18 months, formula milks (18.1%) followed by yoghurt, fromage frais and dairy desserts (17.7%) and biscuits, buns, cakes, pastries, fruit pies and puddings (14.6%), were the largest contributors to free sugars intake. [Foods and drinks specifically marketed for infants and young children](#) aged up to 36 months were also a major contributor to free sugars intake (12.9%) in this age group. Biscuits, buns, cakes, pastries, fruit pies and puddings were the largest contributors in children aged 18 to 47 months (22.7%) and 48 to 60 months (25.5%).
- 3.80 In children aged 18 to 47 months, fruit juice and smoothies contributed nearly 11% to free sugars intake while sugar-sweetened beverages contributed less than 3%. Breakfast cereals provided 7% to 8% of free sugars intake in children aged 18 to 60 months.

**Table 3.13. Food group contributors to average free sugars intake in children aged 12 to 60 months (DNSIYC and NDNS years 2016 to 2019).<sup>1</sup> Population average including non-consumers.**

Contribution of food groups <sup>2,3,4</sup> to free sugars intake	12 to 18 months free sugars <sup>5</sup> %	12 to 18 months free sugars <sup>5</sup> grams per day	18 to 47 months free sugars %	18 to 47 months free sugars grams per day	48 to 60 months free sugars %	48 to 60 months free sugars grams per day
Formula milks <sup>7</sup>	18.1	6.8	2.8	0.8	0.0	0.0
Yoghurt fromage frais and dairy desserts <sup>6</sup>	17.7	3.5	11.8	2.9	7.5	2.9
Biscuits, buns, cakes, pastries, fruit pies, puddings	14.6	3.2	22.7	6.3	25.5	9.2
Commercially manufactured foods and drinks marketed specifically for infants and young children	12.9	3.6	2.4	0.7	1.5	0.7
Sugar preserves and confectionery	10.8	2.5	17.7	5.1	22.6	9.4
Fruit juice and smoothies	4.5	1.2	10.5	3.6	6.6	3.1
Breakfast cereals	3.9	0.7	8.0	1.8	6.9	2.4
Sugar-sweetened beverages <sup>8</sup>	2.4	0.7	2.8	1.1	3.8	2.0
Vegetables, vegetable products and dishes	2.2	0.4	1.6	0.3	1.7	0.5
Meat, meat products and dishes	2.0	0.3	2.5	0.6	3.0	0.8
Fruit	1.8	0.5	0.9	0.3	0.1	0.1
Pizza, pasta, rice, products and dishes	1.8	0.3	2.0	0.5	1.3	0.4
Bread	1.7	0.3	1.7	0.4	1.3	0.4
Ice cream <sup>6</sup>	1.5	0.3	3.9	1.1	6.8	2.5
Low calorie soft drinks <sup>9</sup>	1.2	0.2	3.0	0.6	2.3	0.6
Savoury sauces pickles gravies and condiments	0.8	0.1	1.4	0.4	2.2	0.8
Soup	0.6	0.1	0.3	0.1	0.3	0.2
Beverages dry weight	0.5	0.1	1.4	0.5	3.0	1.5
Milk <sup>6</sup>	0.5	0.1	1.2	0.4	2.6	0.9
Crisps and savoury snacks	0.3	0.0	0.5	0.1	0.4	0.2
Number of participants	1275	1275	306	306	102	102

<sup>1</sup> Data in children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013) otherwise data from NDNS years 2016 to 2019 (Bates et al, 2020).

<sup>2</sup> Food groups are ordered by largest to smallest % contribution in the youngest age group.

<sup>3</sup> Food groups that contribute less than 0.5% of intake in all age groups are not presented.

<sup>4</sup> Average % contribution for each food group has been calculated from the % contribution for each individual. Non-consumers are included in the average.

<sup>5</sup> Free sugars intake for the age group 12 to 18 months was not originally reported in DNSIYC but have been calculated for this report. Sugar intakes for the 2 older age groups (18 to 47 months; 48 to 60 months) were reported as free sugars in the NDNS years 2016 to 2019.

<sup>6</sup> Includes non-dairy alternatives.

<sup>7</sup> Formula milks include milks marketed to children over the age of 1 year, also known as 'toddler milks' and 'growing-up milks' (see Glossary).

<sup>8</sup> Includes carbonated drinks, concentrates and ready to drink products with added sugars.

<sup>9</sup> Includes low calorie, diet, no added sugar, sugar-free drinks. Excludes mineral water.

## **Dietary fibre**

- 3.81 The main dietary sources of dietary fibre in children aged 12 to 60 months are presented in Table 3.14. Vegetables (and vegetable products and dishes), fruit, bread and breakfast cereals were the largest contributors to dietary fibre intakes in children in all the age groups. Commercially manufactured foods and drinks aimed at children aged up to 36 months also contributed, on average, nearly 10% of dietary fibre intakes in children aged 12 to 18 months.

**Table 3.14. Food group contributors to average dietary fibre intake in children aged 12 to 60 months (DNSIYC and NDNS years 2016 to 2019).<sup>1</sup> Population average including non-consumers.**

Contribution of food groups <sup>2,3,4</sup> to dietary fibre <sup>5</sup> intake	12 to 18 months NSP %	12 to 18 months NSP grams per day	18 to 47 months AOAC fibre %	18 to 47 months AOAC fibre grams per day	48 to 60 months AOAC fibre %	48 to 60 months AOAC fibre grams per day
Vegetables, vegetable products and dishes	14.5	1.1	14.0	1.6	16.3	2.2
Fruit	14.2	1.1	15.7	1.7	14.2	1.9
Bread	11.6	0.8	14.7	1.5	14.1	1.7
Breakfast cereals	11.2	0.8	10.7	1.1	10.3	1.4
Commercially manufactured foods and drinks marketed specifically for infants and young children	9.8	0.7	1.6	0.2	1.1	0.1
Formula milks <sup>6</sup>	7.6	0.7	1.1	0.1	0.0	0.0
Potatoes, potato products dishes	7.4	0.5	7.9	0.8	8.4	1.0
Pizza, pasta, rice, products and dishes	6.7	0.5	7.1	0.7	7.1	0.9
Meat, meat products and dishes	6.3	0.4	8.3	0.8	9.1	1.1
Biscuits, buns, cakes, pastries, pies puddings	4.3	0.3	7.4	0.7	8.4	1.0
Soup	1.5	0.1	1.7	0.2	0.8	0.1
Fish, fish products and dishes	1.3	0.1	1.3	0.1	0.9	0.1
Yoghurt, fromage frais and dairy desserts <sup>7</sup>	1.0	0.1	0.8	0.1	0.6	0.1
Crisps and savoury snacks	0.8	0.0	1.9	0.2	2.0	0.2
Savoury sauces pickles gravies and condiments	0.6	0.0	0.6	0.1	0.8	0.1
Sugar, preserves and confectionery	0.5	0.0	1.8	0.2	2.6	0.3
Eggs, egg products and dishes	0.2	0.0	0.3	0.0	0.1	0.0
Fruit juice and smoothies	0.2	0.0	0.6	0.1	0.5	0.1
Nuts and seeds	0.2	0.0	0.8	0.1	0.8	0.1
Milk <sup>7</sup>	0.1	0.0	0.4	0.1	0.4	0.1
Ice cream <sup>7</sup>	0.0	0.0	0.3	0.0	0.7	0.1
Cheese <sup>7</sup>	0.0	0.0	0.5	0.0	0.4	0.0
Number of participants	1275	1275	306	306	102	102

Abbreviations: AOAC, Association of Official Agricultural Chemists; NSP, non-starch polysaccharides.

1 Data in children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013) otherwise data from NDNS years 2016 to 2019 (Bates et al, 2020).

2 Food groups are ordered by largest to smallest % contribution in the youngest age group.

3 Food groups that contribute less than 0.5% of intake in all age groups are not presented.

4 Average % contribution for each food group has been calculated from the % contribution for each individual. Non-consumers are included in the average.

5 NSP comprise cellulose and non-cellulose polysaccharides (e.g. pectins, glucans, arabinogalactans, arabinoxylans, gums and mucilages) (DH, 1991; DH, 1994b). SACN (2015) recommended a broader definition of dietary fibre to include all carbohydrates that are neither digested nor absorbed in the small intestine and have a degree of polymerisation of 3 or more monomeric units, plus lignin. The broader definition of dietary fibre is measured by AOAC methods and is colloquially known as 'AOAC fibre' AOAC fibre intakes are typically about a third higher than NSP intakes.

6 Formula milks include milks marketed to children over the age of 1 year, also known as 'toddler milks' and 'growing-up milks' (see Glossary).

7 Includes non-dairy alternatives.

## **Systematic review evidence identified on carbohydrate intake and health**

- 3.82 2 SRs without MAs (Hörnell et al, 2013; Parsons et al, 1999) included primary studies that examined the health impact of total carbohydrate intake. However, as carbohydrate intake was not included in the search strategies or terms of these 3 SRs, the literature searches of these SRs were not comprehensive for carbohydrate intake as an exposure.
- 3.83 SR evidence on free sugars intake and oral health is covered in chapter 9 ([Free sugars intake and development of dental caries](#)). SR evidence on the health impact of different sources of free sugars, namely, [sugar-sweetened beverages](#) (SSB) and [100% fruit juice](#) are covered in chapters 6 and 0.
- 3.84 No evidence from SRs was identified on the health impact of dietary fibre in children aged 1 to 5 years.
- 3.85 Key outcomes were measures of body composition (BMI, BMI z-score, weight-for-height z-score, body fat) and weight status (overweight or obesity); and cognitive development.
- 3.86 The majority of primary studies included in the SRs were conducted in high income countries (HICs) (defined according to the [World Bank classification system](#)).
- 3.87 Details of the SRs can be found in Annex 5 (Tables A5.1). Quality assessment of the SRs using the AMSTAR 2 tool can be found in Annex 8 (Tables A8.2). Additional data extracted on the primary studies can be found in Annex 9 (Tables A9.1 to A9.3). The criteria used to grade the evidence are provided in chapter 2 (Table 2.4, paragraphs 2.54 to 2.59). Summary tables of the evidence grading process for this section are provided in Annex 10 (Tables A10.2 to A10.5 and Table A10.36).

## **Limitations of the systematic review evidence on carbohydrates**

- 3.88 The evidence identified on the health impact of total carbohydrate intake in young children is from SRs that did not primarily consider this as part of their research question. Therefore, the literature searches conducted by these SRs were unlikely comprehensive for total carbohydrate intake as an exposure, which is a potential source of bias.
- 3.89 Most SRs did not discuss the implications of findings adjusted for TDEI against those that were not adjusted for TDEI when outcomes relating to or resulting from effects on energy balance were investigated (paragraph 3.49).

## **Carbohydrates and body composition or weight status**

### **Total carbohydrate intake and body composition**

- 3.90 The SACN report '[Carbohydrates and Health](#)' (SACN, 2015) found no evidence of an association between total carbohydrate intake (as % TDEI) and BMI or body fatness in children aged 5 years and older (including adolescents).
- 3.91 For this report, 2 SRs without MAs were identified that included studies that examined the relationship between total carbohydrate intake in children aged 1 to 5 years and BMI (Hörnell et al, 2013; Parsons et al, 1999).
- 3.92 Overall, the PCS included in the SRs that adjusted their findings for TDEI reported no association between total carbohydrate intake and BMI, whereas those that did not adjust for TDEI reported an inverse association between total carbohydrate intake and BMI.
- 3.93 Hörnell et al (2013) (AMSTAR 2 confidence rating: moderate) included 1 PCS (in 70 participants) that reported that mean total carbohydrate intake (as % TDEI) at ages 2 to 8 years was inversely associated with BMI at age 8 years (quantitative findings NR), unadjusted for TDEI. The study adjusted for multiple key confounding factors (sex, baseline child BMI, parental BMI and a measure of sedentary behaviour).
- 3.94 Parsons et al (1999) (AMSTAR 2 confidence rating: critically low) included 2 additional PCS that examined the relationship between total carbohydrate intake and BMI in children aged 1 to 5 years. Both PCS (in a total of 258 participants) reported no association between total carbohydrate intake (as % TDEI) in children aged 2 to 5 years and BMI measured 2 and 6 years later, adjusted for TDEI. Both studies adjusted for multiple key confounding factors (sex, baseline child BMI and parental weight status). However, one study had a low participant retention rate (40%), which is a potential source of bias.

### **Total carbohydrate intake and body fat**

- 3.95 Parsons et al (1999) (AMSTAR 2 confidence rating: critically low) included 1 PCS that examined the relationship between total carbohydrate intake in children aged 1 to 5 years and body fat in later childhood. The PCS (112 participants) reported no association between total carbohydrate intake (as % TDEI) in children aged 2 years and body fat (skinfold measurements) 6 years later (quantitative findings NR). The study adjusted for TDEI, baseline child BMI, parental BMI and SES.

## Summary: total carbohydrate intake and body composition

- 3.96 The evidence identified from SRs on total carbohydrate intake and body composition in children aged 1 to 5 years is summarised in Table 3.15.

**Table 3.15. Summary of the evidence on carbohydrate intake and body composition**

Exposure	Outcome	Direction of association	Certainty of evidence
Total carbohydrate intake	Body Mass Index (BMI)	Inconsistent	Insufficient
Total carbohydrate intake	Body fat	Not applicable	Insufficient

- 3.97 The available evidence from SRs examining the relationship between total carbohydrate intake in children aged 1 to 5 years and body composition is from 2 SRs without MAs, 1 given a moderate confidence rating using the AMSTAR 2 tool, the other given a critically low confidence rating.
- 3.98 Evidence from 3 PCS included in the SRs by (Hörnell et al, 2013) and (Parsons et al, 1999) on the relationship between total carbohydrate intake and BMI was inconsistent. As carbohydrate intake was neither an exposure nor included in the search terms of either SR, their literature searches were unlikely comprehensive for total carbohydrate intake as an exposure, which is a potential source of bias. For this reason and given the uncertain role of TDEI in the relationship between total carbohydrate intake and BMI (see paragraphs 3.48 and 3.49), the evidence from SRs was graded 'insufficient'.
- 3.99 There was also 'insufficient' evidence from SRs to enable conclusions to be drawn on any relationship between carbohydrate intake in children aged 1 to 5 years and body fat as there were fewer than 3 primary studies included in the SRs that examined this relationship.

## Dietary fat

- 3.100 Dietary fats include all fats and oils from plants or animals that are edible. Fats in food are predominantly in the form of triacylglycerols (also called triglycerides), which take the form of 3 fatty acids (chains of carbon, hydrogen and oxygen) that are esterified to a glycerol backbone (SACN, 2019). Fatty acids constitute the main components of these lipids and are required as a source of energy and for metabolism and structure (FAO, 2010).
- 3.101 For more details on the classification of fatty acids, digestion, absorption and metabolism of dietary fat, see the [SACN report 'Saturated Fats and Health'](#) (SACN, 2019).

### Current recommendations for dietary fat intake in the UK

- 3.102 The DRVs for dietary fat are presented in Table 3.16. These currently do not apply before age 2 years and apply in full from age 5 years (DH, 1994a). A flexible approach is currently recommended to the timing and extent of dietary change for individual children between 2 and 5 years. However, it is recommended that by the age of 5 years, children should be consuming a diet based on the recommendations for adults (DH, 1994a).
- 3.103 For the purposes of assessing the nutritional intake of young children in the UK, the recommendations in children aged 5 years and older have been applied to children aged under 5 years. Table 3.17 shows the DRVs for dietary fats (DH, 1991; DH, 1994b; SACN, 2019) in children aged 4 to 6 years that have been converted into daily gram amounts using the TDEI values for this age group from SACN (2011a). Data in children aged 1 to 3 years are not available.

**Table 3.16. UK government dietary recommendations for dietary fat<sup>1</sup> for adults and children aged 5 years and older**

Dietary fat	DRV
Total fats <sup>4</sup>	33% total dietary energy (population average)
Saturated fatty acids (saturated fats) <sup>3</sup>	No more than 10% of total dietary energy (population average)
Monounsaturated fatty acids (MUFA) <sup>4</sup>	No specific recommendations for MUFA <sup>5</sup>
n-6 polyunsaturated fatty acids (n-6 PUFA) <sup>4</sup>	6% total dietary energy (population average) <sup>6</sup>
Linoleic acid <sup>2</sup>	Provide at least 1% of total energy
Long chain n-3 PUFA <sup>7</sup>	Increase from 0.2 grams per day to 0.45 grams per day <sup>8</sup>
Alpha linolenic acid (ALA) <sup>2</sup>	Provide at least 0.2% of total energy
Trans fats <sup>4</sup>	Provide no more than about 2% of dietary energy (population average)

<sup>1</sup> Values are expressed as proportions of either total (dietary) energy or dietary energy, depending on the source report.

<sup>2</sup> From DH (1991).

<sup>3</sup> From SACN (2019).

<sup>4</sup> From (DH, 1994a)

<sup>5</sup> To note that DH (1991) recommended that cis-MUFA (principally oleic acid) should continue to provide on average 12% of dietary energy for the population.

<sup>6</sup> To note that (DH, 1994a) recommends no further increase in average intakes of n-6 PUFA and recommends that the proportion of the population consuming excess of about 10% energy should not increase.

<sup>7</sup> From [SACN Advice on fish consumption benefits and risks](#) (SACN/COT, 2004). SACN endorsed the population recommendation (including pregnant women) to eat at least two portions of fish per week, of which one should be oily. Two portions of fish per week, one white and only oily, contain approximately 0.45g per day long chain n-3 PUFA.

<sup>8</sup> To note that DH (DH, 1994a) recommended 'an increase in the population average consumption of long chain n-3 PUFA from about 0.1g per day to about 0.2g per day (1.5g per week)'.

**Table 3.17. DRVs for dietary fat for children aged 4 to 6 years in grams per day<sup>1</sup>**

Type of dietary fat	Boys aged 4 to 6 years	Girls aged 4 to 6 years
Total fat (grams per day) [Less than]	58	54
Saturated fatty acids (grams per day) [Less than]	17	15
Polyunsaturated fatty acids (grams per day)	11	10
Monounsaturated fatty acids (grams per day)	21	20

<sup>1</sup> Source from PHE (2016a), except the values for saturated fatty acids that have been recalculated based on SACN (2019). Fat figures were calculated using the energy figures from (SACN, 2011a). The percentages for which to calculate grams per day of total fat (35% food energy); saturated fatty acids (10% total energy); polyunsaturated fatty acids (6.5% food energy) and monounsaturated fatty acids (13% food energy) were obtained from (DH, 1991) and (SACN, 2019).

## Dietary fat intakes in the UK

### Total fat

- 3.104 Total fat intake in children aged 12 to 60 months in the UK from DNSIYC and NDNS (years 2016 to 2019) is presented in Table 3.18. Mean intake of total fat was highest in children aged 12 to 18 months (35.4% TDEI) and lowest in children aged 48 to 60 months (33.7% TDEI). Although the DRV for total fat (see [Current recommendations for dietary fat intake in the UK](#)) applies in full from age 5 years, and does not apply before age 2 years, it is notable that 69% of children aged 12 to 47 months had intakes above it.
- 3.105 Time trend analysis of NDNS data (years 2008 to 2017) in children aged 18 to 36 months indicated no significant change in mean total fat intake (% TDEI) (0.1 percentage point change per year; 95% CI -0.1 to 0.2) for the 9-year period (Bates et al, 2019). No time trend data were available for the other age groups.

**Table 3.18. Total fat intake in children aged 12 to 60 months in the UK (DNSIYC and NDNS years 2016 to 2019)<sup>1</sup>**

Age	Grams per day <sup>2</sup>	% TDEI <sup>2,3</sup>	% participants above DRV <sup>4</sup>	Number of participants
12 to 18 months	38.2 (10.6)	35.4 (5.0)	(69)	1275
18 to 47 months	41.5 (11.5)	35.3 (4.9)	(69)	306
48 to 60 months	46.1 (13.8)	33.7 (4.7)	(53)	102

Abbreviations: DRV; dietary reference value; TDEI, total dietary energy intake.

<sup>1</sup> Data in children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013), otherwise data from NDNS years 2016 to 2019

<sup>2</sup> Mean (SD).

<sup>3</sup> TDEI is equivalent to food energy as no alcohol is consumed in children of this age.

<sup>4</sup> DRV: ≤33% total energy. The DRV does not apply before 2 years of age and applies in full from age 5 years (DH, 1994a). To indicate this limited applicability of the DRV the figures in this column are stated in parenthesis.

### **Saturated fatty acids (saturated fats)**

- 3.106 Saturated fatty acids (saturated fats) intake in children aged 12 to 60 months is presented in Table 3.19. Mean intake of saturated fats was 16.3% TDEI in children aged 12 to 18 months, 14.8% in children aged 18 to 47 months and 13.5% in children aged 48 to 60 months. Although the DRV for saturated fats (see [Current recommendations for dietary fat intake in the UK](#)) applies in full from age 5 years, and does not apply before age 2 years, it is notable that >90% of children aged 12 to 60 months had intakes above the DRV.
- 3.107 Time trend analysis of NDNS data (years 2008 to 2019) in children aged 18 to 36 months showed no change in saturated fat intakes (% TDEI) (0.0 percentage point change per year; 95% CI -0.1 to 0.1) for the 11-year period (Bates et al, 2020). No time trend data were available for the other age groups.

**Table 3.19 Saturated fat intakes in children aged 12 to 60 months in the UK (DNSIYC and NDNS 2016 to 2019)<sup>1</sup>**

Age	Grams per day <sup>2</sup>	% TDEI <sup>2,3</sup>	% participants exceeding DRV <sup>4</sup>	Number of participants
12 to 18 months	17.5 (5.8)	16.3 (3.6)	(95)	1275
18 to 47 months	17.5 (6.1)	14.8 (3.6)	(91)	306
48 to 60 months	18.6 (6.9)	13.5 (3.0)	(91)	102

Abbreviations: DRV, dietary reference value; TDEI, total dietary energy intake

<sup>1</sup> Data in children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013), otherwise data from NDNS years 2016 to 2019 (Bates et al, 2020).

<sup>2</sup> Mean (SD).

<sup>3</sup> TDEI is equivalent to food energy as no alcohol is consumed in children of this age.

<sup>4</sup> DRV: ≤10% total energy from saturated fats. The DRV does not apply before 2 years of age and applies in full from age 5 years (DH, 1994a). To indicate this limited applicability of the DRV the figures in this column are stated in parenthesis.

## Monounsaturated fatty acids

- 3.108 Mean intake of cis monounsaturated fatty acids (cis MUFA) in children aged 12 to 60 months was approximately 12% TDEI (see Table 3.20). There is no specific UK recommendation for cis MUFAs.
- 3.109 Time trend analysis of NDNS data (years 2008 to 2019) in children aged 18 to 36 months showed an increase in the percentage dietary energy intake from cis MUFA of 0.1 percentage points per year (95% CI 0.0% to 0.1%; p<0.05) for the 11-year period (Bates et al, 2020). No time trend data were available for the other age groups.

**Table 3.20 Cis monounsaturated fatty acids (cis MUFA) intakes in children aged 12 to 60 months in the UK (DNSIYC and NDNS 2016 to 2019)<sup>1</sup>**

Age	Grams per day <sup>2</sup>	% TDEI <sup>2,3</sup>	Number of participants
12 to 18 months	12.4 (3.7)	11.5 (2.2)	1275
18 to 47 months	14.0 (4.0)	12.0 (2.2)	306
48 to 60 months	16.2 (5.1)	11.9 (2.1)	102

Abbreviations: TDEI, total dietary energy intake.

<sup>1</sup> Data in children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013), otherwise data from NDNS 2016 to 2019 (Bates et al, 2020).

<sup>2</sup> Mean (SD).

<sup>3</sup> TDEI is equivalent to food energy as no alcohol is consumed in children of this age.

## Polyunsaturated fatty acids

- 3.110 Mean intake of cis n-3 polyunsaturated fatty acids (cis n-3 PUFA) in children aged 12 to 60 months ranged from 0.7% to 0.8% TDEI, and mean intake of cis n-6 polyunsaturated fatty acids (cis n-6 PUFA) ranged from approximately 4% to 6% TDEI (see Table 3.21).
- 3.111 Time trend analysis of NDNS data (years 2008 to 2019) in children aged 18 to 36 months indicated an increase in % TDEI from PUFA intake of 0.01 (n-3 PUFA) and 0.1 (n-6 PUFA) percentage points per year (n-3: 95% CI 0.01 to 0.02; n-6: 95% CI 0.0 to 0.1; all  $p < 0.05$ ) for the 11-year period (Bates et al, 2020). No time trend data were available for the other age groups.

**Table 3.21 Cis n-3 and n-6 polyunsaturated fatty acids (cis n-3 and n-6 PUFA) intakes in children aged 12 to 60 months in the UK (DNSIYC and NDNS years 2016 to 2019)<sup>1</sup>**

PUFA intake	Age	Grams per day <sup>2</sup>	% TDEI <sup>2,3</sup>	Number of participants
cis n-3	12 to 18 months	0.7 (0.3)	0.7 (0.2)	1275
cis n-3	18 to 47 months	0.9 (0.5)	0.8 (0.4)	306
cis n-3	48 to 60 months	1.1 (0.4)	0.8 (0.3)	102
cis n-6	12 to 18 months	4.0 (1.5)	3.7 (1.2)	1275
cis n-6	18 to 47 months	5.1 (2.0)	4.3 (1.4)	306
cis n-6	48 to 60 months	6.0 (2.2)	4.5 (1.3)	102

Abbreviations: TDEI, total dietary energy intake.

<sup>1</sup> Data in children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013) otherwise data from NDNS 2016 to 2019 (Bates et al, 2020).

<sup>2</sup> Mean (SD).

<sup>3</sup> TDEI is equivalent to food energy as no alcohol is consumed in children of this age.

## Trans fatty acids

- 3.112 Mean intake of trans fatty acids in children aged 12 to 60 months was 0.5% TDEI in each age group (Table 3.22). No children in any age group exceeded the recommendation of no more than 2% TDEI from trans fatty acids.
- 3.113 Time trend analysis of NDNS data (years 2008 to 2017) in children aged 18 to 36 months indicated a decrease in the % TDEI from trans fatty acids of -0.03 percentage points per year (95% CI -0.03 to -0.02;  $p < 0.05$ ) for the 9-year period (Bates et al, 2020). No time trend data were available for the other age groups.

**Table 3.22 Trans fat intakes in children aged 12 to 60 months in the UK (DNSIYC and NDNS years 2016 to 2019)<sup>1</sup>**

Age	Grams per day <sup>2</sup>	% TDEI <sup>2,3</sup>	Number of participants
12 to 18 months	0.6 (0.3)	0.5 (0.2)	1275
18 to 47 months	0.6 (0.3)	0.5 (0.2)	306
48 to 60 months	0.7 (0.3)	0.5 (0.2)	102

Abbreviations: TDEI, total dietary energy intake.

<sup>1</sup> Data in children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013) otherwise data from NDNS 2016 to 2019 (Bates et al, 2020).

<sup>2</sup> Mean (SD).

<sup>3</sup> TDEI is equivalent to food energy as no alcohol is consumed in children of this age.

## Dietary fat intakes and deprivation

- 3.114 Dietary fat intakes by IMD (see Glossary) in children aged 18 to 60 months are presented in Table 3.23. Data from NDNS (years 2016 to 2019) indicated that there was no relationship between total fat and saturated fat intakes and IMD quintile. The confidence intervals indicate that cis MUFA and cis PUFA intakes were significantly lower in quintile 1 (least deprived) compared to quintile 5 (most deprived).

**Table 3.23 Dietary fat intakes by IMD quintile in children aged 18 to 60 months in England (NDNS years 2008 to 2019)<sup>1</sup>**

Intakes % TDEI <sup>2</sup>	IMD quintile 1 (least deprived)	IMD quintile 2	IMD quintile 3	IMD quintile 4	IMD quintile 5 (most deprived)
Total fat Mean (95% CI)	33.6 (33.0 to 34.2)	34.4 (33.7 to 35.0)	33.8 (33.3 to 34.4)	34.2 (33.6 to 34.7)	34.5 (33.9 to 35.0)
Saturated fats Mean (95% CI)	14.6 (14.2 to 15.0)	14.8 (14.4 to 15.2)	14.3 (13.9 to 14.6)	14.1 (13.8 to 14.5)	14.2 (13.8 to 14.5)
Cis MUFA Mean (95% CI)	11.1 (10.9 to 11.4)	11.5 (11.3 to 11.8)	11.6 (11.4 to 11.8)	11.8 (11.5 to 12.0)	12.0 (11.7 to 12.2)
Cis n-3 PUFA Mean (95% CI)	0.72 (0.69 to 0.75)	0.72 (0.69 to 0.75)	0.73 (0.70 to 0.76)	0.75 (0.72 to 0.78)	0.80 (0.76 to 0.85)
Cis n-6 PUFA Mean (95% CI)	3.96 (3.80 to 4.12)	4.05 (3.90 to 4.19)	4.04 (3.89 to 4.20)	4.29 (4.14 to 4.45)	4.31 (4.17 to 4.45)
Number of participants	210	211	182	234	277

Abbreviations: IMD, index of multiple deprivation; TDEI, total dietary energy intake.

<sup>1</sup> Data from NDNS 2008 to 2019 (Bates et al, 2020).

<sup>2</sup> TDEI is equivalent to food energy as no alcohol is consumed by children of this age.

## Main dietary sources of dietary fat

### Total fat

- 3.115 The main dietary sources of total dietary fat in children aged 12 to 60 months are presented in Table 3.24. Milk (27.0%) followed by formula milks (12.4%) were the largest contributors to total fat intake in the youngest age group (age 12 to 18 months) while in the 2 older age groups, meat, meat products and dishes; and biscuits, buns, cakes, pastries, fruit pies and puddings made substantial contributions. In the oldest group meat, meat products and dishes (19.2%) was the largest food group contributor to total fat intake.

**Table 3.24 Food group contributors to average total dietary fat intake in children aged 12 to 60 months (DNSIYC and NDNS years 2016 to 2019)<sup>1</sup>. Population average including non-consumers.**

Contribution of food groups <sup>2,3,4</sup> to total fat intake	12 to 18 months %	12 to 18 months grams per day	18 to 47 months %	18 to 47 months grams per day	48 to 60 months %	48 to 60 months grams per day
Milk <sup>5</sup>	27.0	10.7	20.7	8.9	12.4	5.9
Formula milks <sup>6</sup>	12.4	4.3	1.4	0.5	0.0	0.0
Meat, meat products and dishes	10.0	3.9	14.2	5.8	19.2	8.7
Butter and fat spreads	7.0	2.8	8.3	3.5	9.7	4.6
Biscuits, buns, cakes, pastries, fruit pies and puddings	6.0	2.3	11.0	4.5	14.4	6.5
Cheese <sup>5</sup>	5.2	2.1	6.2	2.5	4.3	2.1
Yoghurt, fromage frais and dairy desserts <sup>5</sup>	3.9	1.4	2.8	1.2	2.4	1.1
Commercially manufactured foods and drinks marketed specifically for infants and young children	3.6	1.2	0.6	0.3	0.5	0.2
Pizza, pasta, rice, products and dishes	2.7	1.0	3.9	1.6	4.5	1.9
Breast milk	2.6	1.0	0.0	0.0	0.0	0.0
Potatoes, potato products and dishes	2.6	1.0	3.2	1.3	4.5	2.1
Eggs, egg products and dishes	2.5	1.0	3.1	1.3	2.8	1.3
Fish, fish products and dishes	2.3	0.9	2.8	1.1	1.9	0.9
Sugar preserves and confectionery	2.1	0.8	3.7	1.5	4.5	2.1
Bread	2.0	0.8	3.2	1.3	3.4	1.5
Breakfast cereals	1.7	0.6	2.7	1.1	2.4	1.2
Vegetables, vegetable products and dishes	1.7	0.7	2.0	0.8	2.4	1.0
Crisps and savoury snacks	1.6	0.6	4.0	1.7	4.1	1.9
Savoury sauces, pickles, gravies and condiments	1.0	0.4	1.6	0.6	1.5	0.7
Fruit	0.7	0.3	1.1	0.4	0.9	0.4
Soup	0.5	0.2	0.6	0.2	0.4	0.2
Ice cream <sup>5</sup>	0.4	0.2	1.1	0.5	2.1	1.0
Nuts and seeds	0.2	0.1	1.3	0.6	1.4	0.8
Number of participants	1275	1275	306	306	102	102

1 Data in children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013) otherwise data from NDNS years 2016 to 2019 (Bates et al, 2020).

2 Food groups are ordered by largest to smallest % contribution in the youngest age group.

3 Food groups that contribute less than 0.5% of intake in all age groups are not presented.

4 Average % contribution for each food group has been calculated from the % contribution for each individual. Non-consumers are included in the average.

5 Includes non-dairy alternatives.

6 Formula milks include milks marketed to children over the age of 1 year, also known as 'toddler milks' and 'growing-up milks' (see Glossary).

### **Saturated fatty acids (saturated fats)**

3.116 The main dietary sources of saturated fatty acids (saturated fats) in children aged 12 to 60 months are presented in Table 3.25. Milk contributed 34.3% of saturated fat intake in children aged 12 to 18 months and formula milks provided a further 11.1%. In the 2 older age groups, milk remained the highest contributor to saturated fat intake, followed by meat (including meat products and dishes) and biscuits, buns, cakes, pastries, fruit pies and puddings.

**Table 3.25 Food group contributors to average saturated fat intake in children aged 12 to 60 months (DNSIYC and NDNS 2016 to 2019)<sup>1</sup>. Population average including non-consumers.**

Contribution of food groups <sup>2,3,4</sup> to saturated fat intake	12 to 18 months %	12 to 18 months grams per day	18 to 47 months %	18 to 47 months grams per day	48 to 60 months %	48 to 60 months grams per day
Milk and cream <sup>5</sup>	34.3	6.6	28.5	5.4	18.6	3.6
Formula milks <sup>6</sup>	11.3	1.6	1.2	0.1	0.0	0.0
Meat, meat products and dishes	8.3	1.4	11.8	1.9	16.4	2.9
Cheese <sup>5</sup>	7.1	1.3	9.0	1.6	6.3	1.3
Butter and fat spreads	6.2	1.1	8.3	1.5	10.5	2.1
Biscuits, buns, cakes, pastries, fruit pies and puddings	6.0	1.0	11.8	2.0	16.6	2.9
Yoghurt, fromage frais and dairy desserts <sup>5</sup>	5.6	0.9	4.2	0.7	3.6	0.7
Commercially manufactured foods and drinks marketed specifically for infants and young children	2.9	0.4	0.5	0.1	0.3	0.0
Breast milk	2.8	0.4	0.0	0.0	0.0	0.0
Sugar, preserves and confectionery	2.6	0.5	4.4	0.7	5.8	1.1
Pizza, pasta, rice, products and dishes	2.3	0.4	3.9	0.6	4.5	0.8
Eggs, egg products and dishes	1.8	0.3	2.4	0.4	2.1	0.4
Potatoes, potato products and dishes	1.4	0.2	1.4	0.2	1.7	0.3
Fish, fish products and dishes	1.2	0.2	1.4	0.2	0.9	0.2
Breakfast cereals	1.2	0.2	2.4	0.4	2.0	0.4
Bread	1.0	0.2	2.0	0.3	2.2	0.4
Vegetables, vegetable products and dishes	0.9	0.2	1.0	0.2	1.3	0.2
Ice cream <sup>5</sup>	0.6	0.1	1.8	0.3	3.7	0.8
Fruit	0.5	0.1	0.8	0.1	0.5	0.1
Crisps and savoury snacks	0.4	0.1	1.0	0.2	1.1	0.2
Soup	0.4	0.1	0.5	0.1	0.3	0.1
Nuts and seeds	0.2	0.1	0.6	0.1	0.8	0.1
Number of participants	1275	1275	306	306	102	102

<sup>1</sup> Data in children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013) otherwise data from NDNS years 2016 to 2019 (Bates et al, 2020).

<sup>2</sup> Food groups are ordered by largest to smallest % contribution in the youngest age group.

<sup>3</sup> Food groups that contribute less than 0.5% of intake in all age groups are not presented.

<sup>4</sup> Average % contribution for each food group has been calculated from the % contribution for each individual. Non-consumers are included in the average.

<sup>5</sup> Includes non-dairy alternatives.

<sup>6</sup> Formula milks include milks marketed to children over the age of 1 year, also known as 'toddler milks' and 'growing-up milks' (see Glossary).

## **Monounsaturated fatty acids**

- 3.117 The main dietary sources of cis MUFA in children aged 12 to 60 months are presented in Annex 11 (Table A11.3). Milk and formula milks contributed over 45% to cis MUFA intake in children aged 12 to 18 months. In children aged 18 to 47 months, meat (including meat products and dishes) was the highest contributor to cis MUFA intake, followed by milk and biscuits, buns, cakes, pastries, fruit pies and puddings. In the oldest children (aged 48 to 60 months), meat (including meat products and dishes) was the highest contributor to cis MUFA intake.

## **Polyunsaturated fatty acids**

- 3.118 The main dietary sources of cis n-3 PUFA and cis n-6 PUFA in children aged 12 to 60 months are presented in Annex 11 (Tables A11.4 and A11.5, respectively). For children aged 12 to 18 months, formula milks, butter and fat spreads, meat (including meat products and dishes) and milk were the largest contributors to n-3 PUFA intake. In the 2 older age groups, meat (including meat products and dishes) and butter and fat spreads were the largest contributors to n-3 PUFA intake while for the oldest age group (47 to 60 months), biscuits, buns, cakes, pastries, pies and puddings was also a large contributor (>10%).
- 3.119 The main contributors to n-6 PUFA intake for the youngest age group (12 to 18 months) were similar to that for cis n-3 PUFA. In the 2 older age groups, meat (including meat products and dishes) followed by biscuits, buns, cakes, pastries, pies and puddings were major contributors to n-6 PUFA intake.

## **Trans fatty acids**

- 3.120 The main dietary sources of trans fatty acids in children aged 12 to 60 months are presented in Annex 11 (Table A11.6). Milk, meat (including meat products and dishes) and cheese were the largest contributors to intakes of trans fatty acids. In the oldest age group (age 48 to 60 months), butter and fat spreads as well as biscuits, buns, cakes, pastries, fruit pies and puddings were also major contributors to intake of trans fatty acids.

## Systematic review evidence identified on dietary fat intake and health

- 3.122 Two SRs without MAs (Naude et al, 2018; Voortman et al, 2015a) were identified that examined the health impact of total fat or PUFA intake in young children. Two other SRs without MAs (Hörnell et al, 2013; Parsons et al, 1999) included studies that examined the health impact of total fat intake but dietary fat intake was not included in the search strategies or terms of these 2 SRs, and therefore the literature searches of these SRs were not comprehensive for dietary fat intake as an exposure which is a potential source of bias.
- 3.123 No new evidence from SRs was identified on the health impact of saturated fats between the publication of the SACN report '[Saturated fats and health](#)' (SACN, 2019) and the cut-off date for consideration of evidence for this report (November 2022). Evidence related to saturated fat intake in children included in SACN's 2019 report has therefore been reproduced in this chapter. The evidence in children from SACN (2019) was drawn exclusively from 1 SR with MA (Te Morenga & Montez, 2017). Te Morenga & Montez (2017) included 8 RCTs in children aged 2 to 16 years in its analyses, of which 1 RCT included children aged 1 to 5 years only. As subgroup analyses by age were not conducted, the % weighting of the MAs from the RCT in children aged 1 to 5 years has been reported, if available.
- 3.124 No evidence from SRs was identified on the health impact of monounsaturated fatty acids (MUFA) or trans fatty acids in children aged 1 to 5 years.
- 3.125 Key outcomes were measures of body composition (BMI, body weight and body fat), blood lipids, blood pressure and linear growth.
- 3.126 The majority of primary studies included in the identified SRs were conducted in high income countries (HICs) (defined according to the [World Bank classification system](#)).
- 3.127 Details of the SRs can be found in Annex 5, Table A5.1. Quality assessment of the SRs using the AMSTAR 2 tool can be found in Annex 8 (Table A8.2). The criteria used to grade the evidence are provided in chapter 2 (Table 2.4, paragraphs 2.54 to 2.59). Additional data extracted from the primary studies can be found in Annex 9 (Table A9.3 to A9.6). The criteria used to grade the evidence are provided in chapter 2 (Table 2.4, paragraphs 2.54 to 2.58). Summary tables of the evidence grading process for this section are provided in Annex 10 (Table A10.4 and Table A10.36).

## **Dietary fat intake and body composition or weight status**

### **Total fat intake and BMI or body weight**

- 3.128 Two SRs without MAs (Naude et al, 2018; Parsons et al, 1999) were identified that examined the relationship between total fat intake and body weight or BMI in children.
- 3.129 Naude et al (2018) (AMSTAR 2 confidence rating: high) included 6 PCS in children aged 1 to 5 years. The SR authors divided the studies into those performed over the shorter term (1 to 3-year follow up) and those performed over the longer term (6 to 14 years). Of the 6 PCS, 4 were in the shorter term and 2 were in the longer term.

#### **Shorter term studies**

- 3.130 The outcomes examined in the 4 shorter term PCS were body weight (1 study), BMI (2 studies) or both (1 study).
- 3.131 The 2 PCS that examined body weight (in a total of 955 participants) reported no association between total fat intake (as % TDEI) and change in body weight. One study (in 215 participants) reported that the mean difference in change in body weight after 2 years between children with lower fat intakes ( $\leq 30\%$  TDEI) compared with children with higher fat intakes ( $>30\%$  TDEI) at ages 3 to 4 years was 0.2kg per year (95% CI  $-0.26$  to  $0.66$ kg per year). The study reported that adjusting for TDEI and key confounding factors (age, sex and baseline body weight) did not alter the results in a substantive way and therefore presented only unadjusted results. The other study (in 740 participants) reported no difference in weight gain from age 7 months to 36 months between children with higher fat intakes ( $>28.7\%$  TDEI) at baseline compared with children with lower fat intakes ( $<28.7\%$  TDEI). The study did not adjust for TDEI or any potential confounding factors and there was a significant imbalance in participant numbers between groups.
- 3.132 Of the 3 PCS that examined change in BMI as an outcome, 1 PCS (in 146 participants) reported that every 1% increase in dietary energy intake from total fat at age 3 to 5 years was associated with an increase in BMI 2 years later (beta coefficient  $0.034\text{kg/m}^2$ ; 95% CI NR;  $p=0.05$ ). The study adjusted for TDEI, and several key confounding factors (sex, age, baseline BMI and physical activity and parental BMI).
- 3.133 The other 2 PCS reported no association between total fat (as % TDEI) and change in BMI. One of these PCS (in 215 participants) reported a mean difference in change in BMI of  $0.02\text{kg/m}^2$  per year (95% CI  $-0.26$  to  $0.30$ ;  $p>0.05$ ) between children with lower fat intakes ( $\leq 30\%$  energy) at age 3 to 4 years compared with children with higher fat intakes ( $>30\%$  energy). The study reported that adjusting

for TDEI and key confounding factors (age, sex and baseline BMI) did not alter the results in a substantive way and therefore presented only unadjusted results. For the other PCS (in 133 participants), quantitative findings were NR. The study adjusted for TDEI, and key confounding factors (sex, ethnicity, baseline BMI, physical activity, and parental weight status).

### **Longer-term studies**

- 3.134 The 2 longer term studies included in Naude et al (2018) examined change in BMI as an outcome. One PCS (in 52 participants) reported that for every 1g increase in total fat intake from ages 2 to 8 years, BMI increased by 0.01kg/m<sup>2</sup> at age 8 years (95% CI NR; p=0.039). The study adjusted for baseline BMI and sedentary behaviour, among other potential confounding factors, but not TDEI. The other PCS (in 112 participants) reported that children in the lower fat intake group (mean 32% TDEI) at age 3 years reduced their BMI z-score by 0.13 while those in the higher fat intake group (mean 40% TDEI) increased their BMI z-score by 0.04 (95% CI and p-value NR) in unadjusted analyses.
- 3.135 Parsons et al (1999) (AMSTAR 2 confidence rating: critically low) included 1 additional PCS (in 112 participants) in children aged 1 to 5 years that reported no association between total fat intake (as % TDEI) in children aged 2 years and BMI 6 years later (correlation coefficient 0.02; p=0.77). The study adjusted for TDEI, baseline child BMI, parental BMI and SES but had a low participant retention rate (40%) by the end of the study which is a potential source of bias.

### **Total fat intake and body fat**

- 3.136 Two SRs without MAs examined the relationship between total fat intake and body fat (Naude et al, 2018; Parsons et al, 1999).
- 3.137 Naude et al (2018) (AMSTAR 2 confidence rating: high) included 1 PCS (in 53 participants) that reported that a 1 unit increase in total fat intake (grams per day) in children aged 2 years was associated with an increase in % body fat (beta coefficient 0.62%; SE 0.26; p=0.02) and total body fat (beta coefficient 179g; SE 70.1; p=0.01) 4 years later, adjusted for TDEI. The study adjusted for baseline child BMI, sex, parental BMI and protein and MUFA intakes (grams per day).
- 3.138 Parsons et al (1999) (AMSTAR 2 confidence rating: critically low) included 1 PCS (in 112 participants) in children aged 1 to 5 years that reported no association between total fat intake in children aged 2 years and subscapular skinfold (correlation coefficient 0.02; p=0.79) or triceps skinfold (correlation coefficient -0.05; p=0.65) at age 8 years, adjusted for TDEI. The study adjusted for baseline BMI, parental BMI and SES but had a low participant retention rate (40%) by the end of the study which is a potential source of bias.

## Summary: total fat intake and body composition or weight status

- 3.139 The evidence identified from SRs on total fat intake or body composition and weight status in children aged 1 to 5 years is summarised in Table 3.26.

**Table 3.26 Summary of the evidence on the relationship between total fat intake and body composition or weight status**

Exposure	Outcome	Direction of association	Certainty of evidence
Total fat intake	Change in Body Mass Index (BMI) or body weight (shorter term)	No association	Limited
Total fat intake	BMI or change in BMI (longer term)	Not applicable	Insufficient
Total fat intake	Body fat	Not applicable	Insufficient

- 3.140 The available evidence from SRs examining the relationship between total fat intake and body composition or weight status in children aged 1 to 5 years is from 2 SRs without MAs, 1 given a high confidence rating using the AMSTAR 2 tool and the other given a critically low rating.
- 3.141 Evidence from 4 PCS included in the SR by Naude et al (2018) suggests that there is no association between total fat intake in children aged 1 to 5 years and change in BMI or body weight in the shorter term (1 to 3 years). The evidence was graded 'limited' due to wide confidence intervals around the effect estimates and the uncertain role of TDEI in this relationship.
- 3.142 There was 'insufficient' evidence from SRs to enable conclusions to be drawn on any relationship between total fat intake in children aged 1 to 5 years and BMI in the longer term (6 to 14 years). This was due to the inconsistency in the findings from PCS and the uncertain role of TDEI in this relationship. Furthermore, one of the SRs (Parsons et al, 1999) that informed this evidence base did not include dietary fat intake in its search terms or strategy. Therefore, its literature search would not have been comprehensive for dietary fat intake as an exposure which is a potential source of bias.
- 3.143 There was also 'insufficient' evidence from SRs to enable conclusions to be drawn on any relationship between total fat intake in children aged 1 to 5 years and body fat as fewer than 3 primary studies included in the SRs examined these relationships.

## **Saturated fat intake and body composition or weight status**

- 3.144 The SACN report on [‘Saturated fats and Health’](#) included 1 MA in children (Te Morenga & Montez, 2017). It reported no effect of reducing saturated fats on BMI, body weight and waist circumference. Of the 4 RCTs (1419 participants) included in the MA, only 1 RCT was conducted in children aged 1 to 5 years (% weighting of the MA NR).
- 3.145 No additional SRs were identified on saturated fats and body composition or weight status outcomes in children aged 1 to 5 years.

## **PUFA intake and body composition or weight status**

- 3.146 One SR without MA (Voortman et al, 2015a) examined the relationship between intakes of PUFA (including n-3 PUFA) and body composition or weight status in children aged up to 5 years.

## **PUFA intake and overweight**

- 3.147 Voortman et al (2015a) (AMSTAR 2 confidence rating: low) included 2 PCS that examined the relationship between PUFA intake and overweight in children aged 1 to 5 years. One PCS (in 3610 participants) reported that a 1 SD increase in PUFA intake (energy-adjusted grams per day) at age 14 months was associated with a 23% lower odds of overweight at age 4 years (OR 0.77; 95% CI 0.62 to 0.96;  $p < 0.05$ ). The study adjusted for sex, birth weight, intakes of saturated fats and MUFA (units unclear), age at introduction of solid foods, parental BMI and several measures of SES.
- 3.148 The other PCS (in 147 participants) reported no difference in PUFA intakes (as % TDEI) at age 1 year between children with a BMI greater than versus less than the 90<sup>th</sup> centile (defined as overweight in the study) at age 5 years ( $p = 0.06$ ) in unadjusted analyses.

## **PUFA intake and body fat**

- 3.149 Voortman et al (2015a) included 1 PCS (in 53 participants) that examined the relationship between PUFA intake in children aged 1 to 5 years and body fat. The PCS reported no association between PUFA intake (grams per day) at age 2 to 5 years and % body fat at age 5 to 6 years. The study adjusted for sex, child BMI (age unspecified), child intakes of other macro- and micronutrients (units unclear) and parental BMI.

## **n-3 PUFA and BMI**

- 3.150 Voortman et al (2015a) included 2 RCTs and 1 PCS that examined the relationship between intakes of n-3 PUFA and BMI in children aged 1 to 5 years.

- 3.151 Both RCTs (in a total of 233 participants) reported no effect of n-3 PUFA (fish oil) supplementation in children up to 5 years old on BMI in the shorter term (9 months) (effect size and 95% CI NR; p=0.85) or longer term (4.5 years) (quantitative findings NR).
- 3.152 The PCS (in 388 children) reported no association between n-3 PUFA (measured by plasma phospholipid concentrations, a biomarker of PUFA intake) at age 2 years and BMI z-score at ages 2, 6 and 10 years (quantitative findings NR). The study adjusted for birth weight, breastfeeding duration and maternal BMI.

### **n-3 PUFA and body fat**

- 3.153 No evidence from SRs was identified on the relationship between n-3 PUFA intake and body composition in children aged 1 to 5 years.

### **Summary: PUFA intake and body composition or weight status**

- 3.154 The evidence identified from SRs on PUFA intake and body composition or weight status in children aged 1 to 5 years is summarised in Table 3.27.

**Table 3.27 Summary of the evidence on PUFA intake and body composition or weight status**

Exposure	Outcome	Direction of association	Certainty of evidence
PUFA intake	Overweight	Not applicable	Insufficient
PUFA intake	Body fat	Not applicable	Insufficient
n-3 PUFA intake	Body Mass Index (BMI) or BMI z-score	Not applicable	Insufficient
n-3 PUFA intake	Body fat	Not applicable	No systematic review evidence identified

Abbreviations: PUFA, polyunsaturated fatty acids

- 3.155 The available evidence from SRs examining the relationship between intakes of PUFA or n-3 PUFA and body composition or weight status in children aged 1 to 5 years is from 1 SR without MA given a low confidence rating using the AMSTAR 2 tool.
- 3.156 There was insufficient evidence from SRs to enable conclusions to be drawn on any relationship between intakes of PUFA or n-3 PUFA in children aged 1 to 5 years and measures of body fatness as fewer than 3 primary studies included in the SRs examined these relationships.

## Dietary fat intake and other health outcomes

### Blood lipids

- 3.157 Dyslipidaemia is defined as an abnormal amount of lipids (triacylglycerols, cholesterol or phospholipids) in the blood while hyperlipidaemia is increased concentrations of lipids in the blood (SACN, 2019). In adults, hyperlipidaemia is associated with a number of metabolic diseases including cardiovascular disease and incident type 2 diabetes (Adult Treatment Panel III, 2001). In 2019, SACN endorsed the conclusions of its predecessor, the Committee on Medical Aspects of Food Policy (COMA) and the European Atherosclerosis Society Consensus Panel, that there is strong evidence that low density lipoprotein cholesterol (LDL-C) and other blood lipids are causally related to cardiovascular morbidity and mortality (FERENCE et al, 2017). Increased concentration of serum high density lipoprotein cholesterol (HDL-C) has been associated with reduced risk of CVD, although the benefits of interventions to raise serum HDL-C remain equivocal (Tariq et al, 2014).

### Total fat intake and blood lipids

- 3.158 No evidence from SRs was identified on the relationship between total fat intake and blood lipids in children aged 1 to 5 years.

### Saturated fat intake and blood lipids

- 3.159 The SACN report '[Saturated fats and Health](#)' (SACN, 2019) included 1 SR with MA in children aged 2 to 16 years (Te Morenga & Montez, 2017). Findings from this SR as described by SACN (2019) are presented below.
- 3.160 Reduced intake of saturated fats lowered serum total cholesterol (MD  $-0.16$  mmol/L, 95% CI  $-0.25$  to  $-0.07$ ,  $p=0.0004$ ;  $I^2=64\%$ , 7 RCTs, 2372 participants). Of the 7 RCTs included in the MA, 1 was conducted in children aged 1 to 5 years (11.7% weighting in the MA).
- 3.161 Reduced intake of saturated fats lowered serum LDL-C using a random-effects model (quantitative findings NR; 7 RCTs; 2004 participants). Of the 7 RCTs included in the MA, 1 study was conducted in children aged 1 to 5 years (14.7% weighting in the MA). The heterogeneity was above the cut-off of 75% ( $I^2=77\%$ ) pre-specified in SACN (2019) and therefore, the pooled estimate was NR.
- 3.162 There was no effect of reduced intake of saturated fats on serum HDL-C (quantitative findings NR; 6 RCTs; 1565 participants). Of the 6 RCTs included in the MA, 1 RCT was conducted in children aged 1 to 5 years (% weighting in the MA NR).
- 3.163 There was no effect of reduced intake of saturated fats on triacylglycerol (quantitative findings NR; 6 RCTs, 1565 participants). Of the 6 RCTs included in

the MA, 1 RCT was conducted in children aged 1 to 5 years (% weighting in the MA NR).

- 3.164 This evidence is consistent with evidence found in adults that lowering saturated fats or substituting saturated fats with PUFA, MUFA or a mixture of PUFA and MUFA lowers serum total cholesterol and LDL cholesterol but has no effect on serum HDL-C or triacylglycerol SACN (2019).
- 3.165 No additional SRs were identified on saturated fats and blood lipids in children.

### **PUFA intake and blood lipids**

- 3.166 One SR without MA (Voortman et al, 2015a) was identified that examined the relationship between PUFA intake and blood lipids (serum total cholesterol, LDL-C and HDL-C) in children aged 5 years and under.

### **Serum total cholesterol**

- 3.167 Voortman et al (2015a) (AMSTAR 2 confidence rating: low) included 2 PCS that examined serum total cholesterol in children aged 1 to 5 years. One PCS (in 127 participants) reported no association between total PUFA intake (as % TDEI) at age 6 months to 4 years and sex-adjusted serum total cholesterol in univariate regression analyses (quantitative findings NR). The other PCS (in 496 participants) reported no association between energy-adjusted PUFA intake (transformed to the natural logarithm to normalise the distribution of intake) at age 18 months and serum total cholesterol at age 31 months (quantitative findings NR) after adjusting for TDEI, energy-adjusted intakes of saturated fats and PUFA, starch, sugar and dietary fibre; and key confounding factors (sex, ethnicity).

### **Serum LDL cholesterol**

- 3.168 Voortman et al (2015a) included 1 PCS (in 127 participants) that examined serum LDL-C in children aged 1 to 5 years. It reported no association between total PUFA intake (as % TDEI) at age 6 months to 4 years and serum LDL-C at age 4 years (quantitative findings NR) in univariate regression analyses.

### **Serum HDL cholesterol**

- 3.169 Voortman et al (2015a) included 2 PCS that examined serum HDL-C in children aged 1 to 5 years. One PCS (in 496 participants) reported that every unit increase in energy-adjusted PUFA intake (transformed to the natural logarithm) at age 18 months was associated with a decrease in HDL-C ( $-0.15\text{mmol/l}$ ; 95% CI  $-0.29$  to  $-0.01\text{mmol/l}$ ;  $p=0.036$ ) at age 31 months in girls only. The study adjusted for TDEI, energy-adjusted intakes of saturated fats, PUFA, starch, sugar, dietary fibre, vitamin C and key confounding factors (sex and ethnicity).

- 3.170 The other PCS (in 127 participants) reported no association between PUFA intake (% TDEI) at ages 6 months to 4 years and serum HDL-C at age 4 years in either sex, in univariate analyses.

#### **Serum triacylglycerol**

- 3.171 No evidence from SRs was identified on the relationship between PUFA intake and triacylglycerol in children aged 1 to 5 years.

#### **n-3 PUFA intake and blood lipids**

##### **Serum total cholesterol and LDL cholesterol**

- 3.172 No evidence from SRs was identified on the relationship between n-3 PUFA intake and serum total cholesterol and LDL-C in children aged 1 to 5 years.

##### **Serum HDL cholesterol**

- 3.173 Voortman et al (2015a) included 1 RCT that examined serum HDL-C in children aged 1 to 5 years. The RCT (in 100 participants) reported no effect of n-3 PUFA (fish oil supplementation) at age 6 months to 5 years and serum HDL-C at age 8 years (quantitative findings NR).

##### **Serum triacylglycerol**

- 3.174 Voortman et al (2015a) included 1 RCT that examined serum triacylglycerol in children aged 1 to 5 years. The RCT (in 100 participants) reported no effect of n-3 PUFA (fish oil supplementation) at age 6 months to 5 years and serum triacylglycerol at age 8 years (quantitative findings NR).

#### **Summary: dietary fat intake and blood lipids**

- 3.175 The evidence identified from SRs on dietary fat intake and blood lipids in children aged 1 to 5 years is summarised in Table 3.28.

**Table 3.28 Summary of the evidence on dietary fat intake and blood lipids**

Exposure	Outcome	Direction of association	Certainty of evidence
Total fat	Blood lipids (all)	Not applicable	No systematic review evidence identified
Saturated fats	Blood lipids (all)	Not applicable	No additional evidence identified since the SACN report ' <a href="#">Saturated fats and health</a> '
PUFA intake	TC	Not applicable	Insufficient
PUFA intake	LDL-C	Not applicable	Insufficient
PUFA intake	HDL-C	Not applicable	Insufficient
PUFA intake	Triacylglycerol	Not applicable	No systematic review evidence identified
n-3 PUFA intake	TC	Not applicable	No systematic review evidence identified
n-3 PUFA intake	LDL-C	Not applicable	No systematic review evidence identified
n-3 PUFA intake	HDL-C	Not applicable	Insufficient
n-3 PUFA intake	Triacylglycerol	Not applicable	Insufficient

Abbreviations: HDL-C, high-density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; PUFA, polyunsaturated fatty acids; TC, total cholesterol.

- 3.176 No evidence from SRs was identified on the relationship between total fat intake and blood lipids in children aged 1 to 5 years.
- 3.177 The available evidence examining the relationship between intakes of PUFA or n-3 PUFA and blood lipids in children aged 1 to 5 years is from 1 SR without MA, given a low confidence rating using the AMSTAR 2 tool.
- 3.178 There was insufficient evidence from SRs to enable conclusions to be drawn on any relationship between intakes of PUFA or n-3 PUFA in children aged 1 to 5 years and blood lipids as fewer than 3 primary studies included in the SRs examined these relationships.

## Blood pressure

- 3.179 Hypertension is one of the most important modifiable risk factors for cardiovascular, cerebrovascular and renal disease (WHO, 2017). Blood pressure in childhood is strongly predictive of blood pressure in later life (Bao et al, 1995). The global prevalence of children (aged 19 years and under) with hypertension is estimated to be around 4%, with a higher prevalence in children with obesity (between 7 and 25%) and overweight (between 2 and 9%) compared with children with healthy weight (Song et al, 2019).

## Total fat intake and blood pressure

- 3.180 No evidence from SRs was identified on the relationship between total fat intake and blood pressure in children aged 1 to 5 years.

## Saturated fat intake and blood pressure

- 3.181 The SACN report '[Saturated fats and Health](#)' (SACN, 2019) included 1 MA in children (Te Morenga & Montez, 2017). Its findings as described in SACN (2019) are reproduced below.
- 3.182 There was no effect of reducing saturated fats on systolic blood pressure (SBP) (quantitative findings NR; 2 RCTs, 1106 participants). Of the 2 RCTs included in the MA, 1 RCT was conducted in children aged 1 to 5 years (25.6% weighting in the MA).
- 3.183 A reduction in saturated fats decreased diastolic blood pressure (DBP) (MD  $-1.45$ , 95% CI  $-2.34$  to  $-0.56$ ,  $p=0.001$ ;  $I^2=0\%$ ; 2 RCTs, 1106 participants). Of the 2 RCTs included in the MA, 1 RCT was conducted in children aged 1 to 5 years (57.2% weighting in the MA).
- 3.184 No additional SRs were identified on saturated fats and blood lipids in children.

## PUFA intake and blood pressure

- 3.185 One SR without MA was identified that examined the relationship between PUFA intake and blood pressure in children aged 5 years and under (Voortman et al, 2015a).
- 3.186 Voortman et al (2015a) (AMSTAR 2 confidence rating: low) included 1 PCS (in 2882 participants) in children aged 1 to 5 years that reported no association between PUFA intake ( $>8.6g$  per day vs  $<7g$  per day, adjusted for TDEI) at age 14 months and SBP (beta coefficient  $0.26$  mmHg; 95% CI  $-0.41$  to  $0.93$  mmHg;  $p$ -value NR) or DBP at age 6 years (beta coefficient  $0.10$  mmHg; 95% CI  $-0.46$  to  $0.66$  mmHg;  $p$ -value NR). The study adjusted for multiple key confounding factors (age, sex, ethnicity, birth weight, BMI at age 6 years, sedentary behaviour, maternal smoking and education).

## n-3 PUFA intake and blood pressure

- 3.187 Voortman et al (2015a) included 1 RCT (in 100 participants) that examined the relationship between n-3 PUFA intake and SBP and DBP in children aged 1 to 5 years. It reported no effect of n-3 PUFA (fish oil supplementation) at ages 6 months to 5 years and SBP or DBP at age 8 years.

## Summary: dietary fat intake and blood pressure

- 3.188 The evidence identified from SRs on dietary fat intake and blood pressure in children aged 1 to 5 years is summarised in Table 3.29.

**Table 3.29 Summary of the evidence on dietary fat intake and blood pressure**

Exposure	Outcome	Direction of effect or association	Certainty of evidence
Total fat intake	Blood pressure	Not applicable	No systematic review evidence identified
Saturated fat intake	Blood pressure	Not applicable	No additional systematic review evidence identified since the SACN report <a href="#">‘Saturated fats and health’</a>
PUFA intake	Blood pressure	Not applicable	Insufficient
n-3 PUFA intake	Blood pressure	Not applicable	Insufficient

Abbreviations: PUFA, polyunsaturated fatty acids

- 3.189 No evidence from SRs was identified on the relationship between total fat intake and blood pressure in children aged 1 to 5 years.
- 3.190 The available evidence examining the relationship between intakes of PUFA or n-3 PUFA in children aged 1 to 5 years and blood pressure is from 1 SR without MA, given a low confidence rating using the AMSTAR 2 tool.
- 3.191 There was ‘insufficient’ evidence from SRs to enable conclusions to be drawn on any relationship between intakes of PUFA or n-3 PUFA in children aged 1 to 5 years and blood pressure as fewer than 3 primary studies included in the SRs examined these relationships.

## **Linear growth**

- 3.192 In this report, linear growth denotes changes in a child's height or length. Outcome measures related to linear growth that are examined by the SRs identified for this section were change in height and age at peak linear growth velocity.

### **Total fat intake and linear growth**

- 3.193 Two SRs without MAs (Hörnell et al, 2013; Naude et al, 2018) examined the relationship between total fat intake and linear growth.
- 3.194 Naude et al (2018) (AMSTAR 2 confidence rating: high) included 2 PCS in children aged 1 to 5 years. Both studies (in a total of 955 participants) reported no association between total fat intake (% TDEI) in children aged under 5 years and linear growth measured 1 to 2 years later. While one study did not adjust for any potential confounding factors and had a significant imbalance in participant numbers between comparison groups, the other study reported that adjusting for key confounding factors (age, sex, ethnicity, baseline weight) and TDEI did not alter the results and therefore presented only unadjusted results.
- 3.195 Hörnell et al (2013) (AMSTAR 2 confidence rating: moderate) included 1 PCS (in 67 girls) in children aged 1 to 5 years that reported that 1 SD increase in total fat intake (adjusted for age and TDEI, expressed as logarithmic scale residuals) at ages 1 to 2 years was associated with earlier peak linear growth during adolescence (by 0.63 years;  $p < 0.05$ ). The study defined the age at peak linear growth velocity as the adolescent year in which a child experienced the most rapid growth in height. The study adjusted for age- and energy-adjusted intakes of animal and vegetable protein, BMI and age-specific height z-scores at ages 1 to 5 years. However, participants were born in the 1930s and 1940s when nutrition and lifestyle factors may have been different from today, potentially limiting the generalisability of this finding. The study also had a low participant retention rate (<60%) which is a potential source of bias.

### **Saturated fat intake and linear growth**

- 3.196 No evidence from SRs was identified on the relationship between saturated fat intake and linear growth in children aged 1 to 5 years.

### **PUFA intake and linear growth**

- 3.197 No evidence from SRs was identified on the relationship between PUFA intake and linear growth in children aged 1 to 5 years.

## **Summary: dietary fat intake and linear growth**

- 3.198 The evidence identified from SRs on dietary fat intake and linear growth in children aged 1 to 5 years is summarised in Table 3.30.

**Table 3.30 Summary of the evidence on dietary fat intake and linear growth**

Exposure	Outcome	Direction of association	Certainty of evidence
Total fat intake	Age at peak growth velocity	Not applicable	Insufficient
Total fat intake	Linear growth	Not applicable	Insufficient
Saturated fat intake	Linear growth	Not applicable	No systematic review evidence identified since the SACN report <a href="#">‘Saturated fats and health’</a>
PUFA intake	Linear growth	Not applicable	No systematic review evidence identified
n-3 PUFA intake	Linear growth	Not applicable	No systematic review evidence identified

Abbreviations: PUFA, polyunsaturated fatty acids.

- 3.199 The available evidence examining the relationship between dietary fat intake in children aged 1 to 5 years and linear growth outcomes is from 2 SRs without MAs, 1 given a high confidence rating using the AMSTAR 2 tool, the other given a moderate confidence rating.
- 3.200 There was ‘insufficient’ evidence from SRs to enable conclusions to be drawn on any relationship between total fat intake in children aged 1 to 5 years and linear growth as fewer than 3 primary studies included in the SRs examined this relationship.
- 3.201 No evidence from SRs was identified on the relationship between intake of saturated fats, PUFA or n-3 PUFA and linear growth in children aged 1 to 5 years.

## Protein

- 3.202 Proteins consist of amino acids joined by peptide bonds into polypeptide chains. These polypeptide chains are folded into a three-dimensional structure to form the protein. Of the 20 amino acids that build proteins in living organisms, 9 are classified essential as they cannot be synthesised in the human body. Dietary proteins are the source of essential amino acids and nitrogen (EFSA, 2015a).

### Current recommendations for protein intake in the UK

- 3.203 Dietary proteins are necessary for tissue growth and maintenance (EFSA, 2015a).
- 3.204 The current DRVs for protein in the UK were set by COMA in 1991 (DH, 1991). COMA set a reference nutrient intake (RNI) at 14.5 grams per day for children aged 1 to 3 years and 19.7 grams per day for children aged 4 to 6 years, not stratified by sex. The RNI is the amount likely to be sufficient for 97.5% of those in a population. If the mean intake of a population is above the RNI, it is likely that intakes are adequate. The DRVs were based on the recommendations published in a [report from the joint FAO/WHO/UNU expert consultation in 1985](#) (WHO, 1985).
- 3.205 In 2012, the [European Food Safety Authority \(EFSA\) published updated DRVs for protein](#), which were originally set in 1993 by the Scientific Committee for Food for the European Community (EFSA, 2015a). EFSA adopted the recommendations published in a [report by the WHO/FAO/UN joint expert consultation in 2007](#) (WHO, 2007b). It set a population reference intake (PRI), which is the intake of a nutrient that is likely to meet the needs of almost all healthy people in a population or 97.5% of the individuals in the population. The PRI is stratified by sex.
- 3.206 The DRVs set by DH (1991) and EFSA (2015a) are presented in Table 3.31, while the values from which the DRVs set by each body were derived are presented in Table 3.32. The table indicates that the COMA DRVs, which were derived from the 1985 WHO values, were overestimated (by between 20 to 30% for this age group).

**Table 3.31 COMA (1991) DRVs for protein for children aged 1 to 5 years compared with DRVs set by EFSA**

Age (years)	COMA (1991) RNI (grams per day) <sup>1</sup>	EFSA (2012)	
		PRI (grams per day) <sup>2</sup> Boys	PRI (grams per day) <sup>2</sup> Girls
1	14.5	12	11
1.5	14.5	12	11
2	14.5	12	12
3	14.5	13	13
4	19.7	15	14
5	19.7	16	16

Abbreviations: RNI, Reference Nutrient Intake; PRI, Population Reference Intake.

<sup>1</sup> Data from DH (1991). It is recommended that intake in adults should not exceed twice the RNI; no recommendations on high intakes were made for children. The RNI is based on a body weight of 12.5kg and 17.8kg for children aged 1 to 3 years and 4 to 5 years, respectively. Data are for boys and girls.

<sup>2</sup> Data from EFSA (2015a).

**Table 3.32 Safe level of protein intake<sup>1</sup> for children aged 1 to 5 years in the WHO 1985 and 2007 reports**

Age (years)	WHO 1985 <sup>2</sup>	WHO 2007 <sup>3</sup>
1	1.57	1.14
1.5	1.26	1.03
2	1.17	0.97
3	1.13	0.90
4	1.09	0.86
5	1.06	0.85

<sup>1</sup> In gram protein per kg body weight per day. The safe level of intake for a population is defined as the average protein requirement of the individuals in the population, plus twice the standard deviation (SD) (WHO, 2007b).

<sup>2</sup> Data from (WHO, 1985) on which the COMA DRVs for protein (DH, 1991) were derived.

<sup>3</sup> Data from (WHO, 2007b) on which the EFSA DRVs for protein (EFSA, 2015a) were derived.

## Protein intakes in the UK

3.207 Protein intake in children aged 12 to 60 months in the UK from DNSIYC and NDNS (years 2016 to 2019) is presented in Table 3.33.

**Table 3.33 Protein intakes in children aged 12 to 60 months in the UK (DNSIYC and NDNS 2016 to 2019)<sup>1</sup>**

Age	Grams per day <sup>2</sup>	% TDEI <sup>2,3</sup>	% of participants above RNI	Number of participants
12 to 18 months	37.7 (10.2)	15.6 (2.6)	99	1275
18 to 47 months	41.0 (10.0)	15.7 (2.8)	100	306
48 to 60 months	45.8 (14.8)	15.0 (3.0)	100	102

Abbreviations: RNI, reference nutrient intake; TDEI, total dietary energy intake.

<sup>1</sup> Data in children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013), otherwise data from NDNS 2016 to 2019 (Bates et al, 2020).

<sup>2</sup> Mean (SD).

<sup>3</sup> TDEI is equivalent to food energy as no alcohol is consumed in children of this age.

- 3.208 Mean protein intake in children aged 12 to 18 months was 37.7 grams per day, more than 2.5 times the RNI (14.5 grams per day) and more than 3 times the PRI (Table 3.31). In children aged 18 to 48 months, mean protein intake was 41.0 grams per day, close to 3 times the RNI, and 3 to 4 times the PRI. In children aged 48 to 60 months, mean protein intake was 45.8 grams per day, more than twice the RNI and around 3 times the PRI.
- 3.209 Time trend analysis of NDNS data (years 2008 to 2017) in children aged 18 to 36 months indicated no significant change in protein intakes (0.0 percentage point change per year 95% CI 0.0 to 0.1) for the 9-year period (Bates et al, 2019). No time trend data was available for the other age groups.

## **Protein intake and deprivation**

- 3.210 Protein intake by IMD (see Glossary) is presented in Table 3.34. Although there were small differences in mean protein intake (as % TDEI and in grams per day) between IMD quintiles, there was no evidence of any relationship between protein intakes and IMD quintile (as indicated by overlapping confidence intervals).

**Table 3.34 Protein intakes by IMD quintile in children aged 18 to 60 months in England (NDNS years 2008 to 2019)<sup>1</sup>**

Protein	IMD quintile 1 (least deprived)	IMD quintile 2	IMD quintile 3	IMD quintile 4	IMD quintile 5 (most deprived)
Grams per day Mean (95% CI)	43.7 (42.3 to 45.1)	43.7 (42.5 to 44.9)	45.4 (44.0 to 46.8)	43.0 (41.8 to 44.2)	41.9 (40.6 to 43.1)
% TDEI <sup>2</sup> Mean (95% CI)	15.1 (14.8 to 15.4)	15.6 (15.3 to 15.9)	15.6 (15.3 to 16.0)	15.2 (14.9 to 15.4)	15.3 (15.0 to 15.6)
Number of participants	210	211	182	234	277

Abbreviations: IMD, index of multiple deprivation; TDEI, total dietary energy intake.

<sup>1</sup> Data from NDNS years 2008 to 2019 (Bates et al, 2020).

<sup>2</sup> TDEI is equivalent to food energy as no alcohol is consumed by children of this age.

## Main dietary sources of protein

- 3.211 Different foods contain variable proportions of dietary proteins, which differ in their amino acid composition and essential amino acid content. This results in variability of dietary protein intake within and between populations (EFSA, 2015a).
- 3.212 Foods of animal origin with a high protein content are meat, fish, eggs, milk and dairy products while plant-based foods with a high protein content include legumes (such as peas, beans, lentils and soya), nuts and seeds, and bread and cereals. The essential amino acid content of plant proteins is usually lower than in animal proteins (EFSA, 2015a). Foods with high quality protein content have an optimal amino acid composition for human requirements and are highly digestible. Animal proteins tend to be considered as having higher protein quality than plant proteins (EFSA, 2015a).
- 3.213 The main dietary sources of protein in children aged 12 to 60 months in the UK are presented in Table 3.35. Milk (24%) followed by meat (including meat products and dishes) (17.0%) were the largest contributors to protein intake in children aged 12 to 18 months. In children aged 48 to 60 months, meat (including meat products and dishes) (27.0%) was the largest contributor to protein intake followed by milk (16.1%).

**Table 3.35 Food group contributors to average protein intake in children aged 12 to 60 months (DNSIYC and NDNS years 2016 to 2019)<sup>1</sup>. Population average including non-consumers.**

Contribution of food groups to protein intake <sup>2,3,4</sup>	12 to 18 months %	12 to 18 months Grams per day	18 to 47 months %	18 to 47 months Grams per day	48 to 60 months %	48 to 60 months Grams per day
Milk <sup>5</sup>	23.7	9.6	20.2	8.7	16.1	7.7
Meat, meat products and dishes	17.0	6.7	22.9	9.5	27.0	12.7
Yoghurt, fromage frais and dairy desserts <sup>5</sup>	7.0	2.6	4.5	1.9	3.9	1.7
Bread	6.7	2.5	9.4	3.8	9.7	4.3
Formula milks <sup>6</sup>	6.6	2.0	0.8	0.3	0.0	0.0
Commercially manufactured foods and drinks specifically marketed for infants and young children	5.4	1.8	0.7	0.3	0.5	0.2
Pizza, pasta, rice, products and dishes	4.6	1.7	5.7	2.3	7.1	3.0
Fish, fish products and dishes	4.2	1.6	4.7	1.9	4.1	2.0
Breakfast cereals	3.9	1.5	4.6	1.8	4.7	2.2
Cheese <sup>5</sup>	3.9	1.5	5.0	2.0	3.6	1.7
Vegetables, vegetable products and dishes	3.8	1.4	4.0	1.6	4.5	2.0
Biscuits, buns, cakes, pastries, fruit pies and puddings	3.1	1.1	5.0	2.0	6.2	2.6
Eggs, egg products and dishes	2.3	0.9	3.4	1.4	2.8	1.3
Potatoes, potato products and dishes	2.0	0.7	2.0	0.8	2.4	1.1
Fruit	1.9	0.7	2.1	0.8	2.2	1.0
Breast milk	1.1	0.3	0.0	0.0	0.0	0.0
Soup	1.0	0.4	1.0	0.4	0.5	0.3
Sugar preserves and confectionery	0.6	0.2	1.0	0.4	1.4	0.6
Savoury sauces, pickles, gravies and condiments	0.5	0.2	0.4	0.3	0.5	0.2
Crisps and savoury snacks	0.3	0.1	0.8	0.3	0.9	0.4
Ice cream <sup>5</sup>	0.2	0.1	0.5	0.2	0.8	0.4
Fruit juice and smoothies	0.1	0.0	0.4	0.2	0.3	0.1
Nuts and seeds	0.1	0.0	0.6	0.2	0.6	0.3
Number of participants	1275	1275	306	306	102	102

<sup>1</sup> Data in children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013), otherwise data from NDNS years 2016 to 2019 (Bates et al, 2020).

<sup>2</sup> Food groups are ordered by largest to smallest % contribution in the youngest age group.

<sup>3</sup> Food groups that contribute less than 0.5% of intake in all age groups are not presented.

<sup>4</sup> Average % contribution for each food group has been calculated from the % contribution for each individual. Non-consumers are included in the average.

<sup>5</sup> Includes non-dairy alternatives.

<sup>6</sup> Formula milks include milks marketed to children over the age of 1 year, also known as 'toddler milks' and 'growing-up milks' (see Glossary).

## **Systematic review evidence identified on protein intake and health**

- 3.214 Two SRs without MAs (Hörnell et al, 2013; Voortman et al, 2015b) were identified that examined the health impact of protein intake in children. An additional 2 SRs without MAs (Dougkas et al, 2019; Parsons et al, 1999) included primary studies that examined the health impact of protein intake. However, as protein intake was neither a primary exposure nor included in the search terms of these 2 SRs, the literature searches for these 2 SRs was not comprehensive for protein intake as an exposure which is a potential source of bias.
- 3.215 Key exposures were total protein intake (Hörnell et al, 2013; Parsons et al, 1999; Voortman et al, 2015b) and different sources of protein (animal, vegetable, meat, dairy) (Dougkas et al, 2019; Hörnell et al, 2013).
- 3.216 Key outcomes were:
- Body composition (BMI, body weight and body fat) or weight status
  - growth outcomes (age of adiposity rebound, peak linear growth velocity)
  - pubertal timing (timing of menarche or voice break, pubertal growth spurt)
  - blood lipids
  - bone health
  - neurodevelopment.
- 3.217 Details of the SRs included in this section can be found in Annex 5 (Table A5.1 and Table A5.3). Quality assessment of the SRs using the AMSTAR 2 tool can be found in Annex 8 (Tables A8.2 and A8.4). Additional data extracted on the primary studies can be found in Annex 9 (Tables A9.7 to A9.10). The criteria used to grade the evidence are provided in chapter 2 (Table 2.4, paragraphs 2.54 to 2.59). Summary tables of the evidence grading process for this section are provided in Annex 9 (Tables A10.5, A10.6 and A10.36).

### **Protein intake and body composition or weight status**

- 3.218 Evidence from RCTs and observational studies indicates that higher protein intake in infancy (for example, through infant formula feeding) promotes rapid weight gain and later risk of obesity (SACN, 2018). The association between higher protein intakes and rapid growth in the first year of life is thought to depend on the stimulating effect of protein intake on insulin-like growth factor 1 (IGF-1) which promotes increased muscle as well as fat mass (Hörnell et al, 2013).
- 3.219 Some researchers consider the first 5 years of life to be a 'critical period' for protein intake and later adiposity (Gunther et al, 2007).

- 3.220 This report examined the evidence from SRs on protein intake in children aged 1 to 5 years and body composition and weight status.

### **Total protein intake and later BMI and overweight**

- 3.221 Two SRs without MAs examined the relationship between total protein intake and BMI (Hörnell et al, 2013; Parsons et al, 1999).
- 3.222 Hörnell et al (2013) (AMSTAR 2 confidence rating: moderate) included 4 PCS in children aged 1 to 5 years. All 4 PCS (in a total of 547 participants) reported that higher total protein intake (as % TDEI) at ages 1 to 2 years was associated with increased BMI at ages 4 to 8 years. Two of the 4 PCS adjusted for TDEI. One PCS (in 203 participants) reported that children with consistently high protein intakes at ages 12 months and 18 to 24 months (median intake at ages 18 to 24 months: 13.8% TDEI) had a standardised BMI (BMI SDS) of 0.37 (95% CI 0.12 to 0.61) at age 7 years compared with a BMI SDS of 0.08 (95% CI -0.09 to 0.26) in children with lower protein intakes (median intake at ages 18 to 24 months: 13.3% TDEI) ( $p=0.04$  between-group difference). Analyses were adjusted for TDEI and multiple confounding factors including sex, baseline BMI SDS, parental weight status and SES.
- 3.223 Of the 4 PCS, 2 PCS also reported an association between higher total protein intake in early childhood and later overweight.
- 3.224 One PCS (in 203 participants) reported that consistently high protein intakes at ages 12 months and 18 to 24 months (median intake at ages 18 to 24 months: 13.8% TDEI) was associated with a more than 2-fold greater odds of being overweight at age 7 years compared with children with lower protein intakes (median intake at ages 18 to 24 months: 13.3% TDEI) (OR 2.39; 95% CI 1.14 to 4.99;  $p=0.02$ ). Overweight was defined as having a BMI >75<sup>th</sup> percentile of German reference curves. The analysis adjusted for TDEI, sex, baseline BMI SDS and SES.
- 3.225 The other PCS (in 147 participants) reported that children with overweight at age 5 years had a higher total protein intake at age 1 year compared with children with healthy weight (mean 22% versus 20% of total energy;  $p=0.024$ ). This relationship was supported by multivariate logistic analysis that demonstrated that total protein intake at age 1 year was associated with overweight at age 5 years (estimate of association NR;  $p=0.05$ ). The analysis adjusted for sex, weight and length at birth and at age 1 year, other macronutrients (% TDEI), parental age and weight status. Overweight was defined as having a BMI >90<sup>th</sup> percentile of age- and sex-adjusted curves created by (Rolland-Cachera et al, 1982)
- 3.226 Parsons et al (1999) (AMSTAR 2 confidence rating: critically low) included an additional PCS (in 112 participants) in children aged 1 to 5 years that reported that higher total protein intake (as % TDEI) at age 2 years was correlated with higher BMI at age 8 years (correlation coefficient 0.27;  $p=0.008$ ) after adjusting for TDEI,

baseline BMI, and parental BMI. However, the study had a low participant retention rate (40%) by the end of the study which is a potential source of bias.

### **Total protein intake and body fat**

- 3.227 Two SRs without MAs examined the relationship between total protein intake and body fat (Hörnell et al, 2013; Parsons et al, 1999).
- 3.228 Hörnell et al (2013) included 1 PCS (in 203 participants) that reported that children with consistently high total protein intakes (median intake at ages 18 to 24 months: 13.8% TDEI) at ages 12 months and 18 to 24 months had a more than 2-fold greater odds of having a % body fat over the 75<sup>th</sup> percentile of body fat reference curves (based on % body fat values measured by bioelectric impedance analysis in British children; (McCarthy et al, 2006)) at age 7 years compared with children with a consistently lower total protein intake (median intake at ages 18 to 24 months: 13.3% TDEI) (OR 2.28; 95% CI 1.06 to 4.88; p=0.03). The analysis adjusted for TDEI, sex, baseline BMI SDS, SES. Percentage body fat was calculated from multiple skinfold measurements.
- 3.229 Parsons et al (1999) included an additional PCS (in 112 participants) in children aged 1 to 5 years that reported no association between total protein intake (as % TDEI) at age 2 years and body fat (% and total body fat) at age 8 years, adjusted for TDEI and baseline BMI. However, there was a correlation with subscapular skinfold after adjusting for parental BMI (correlation coefficient 0.20; p=0.004). Body fatness was predicted by triceps and subscapular skinfolds. The study had a low participant retention rate (40%) by the end of the study which is a potential source of bias.

### **Animal protein intake and BMI**

- 3.230 Two SRs without MAs examined the relationship between animal protein intake and BMI or body weight (Hörnell et al, 2013; Parsons et al, 1999).
- 3.231 Hörnell et al (2013) included 1 PCS (in 203 participants) that reported that higher intake of animal protein (as % TDEI) at age 1 year was associated with increased BMI SDS at age 7 years (estimate of association NR; p=0.02). Additionally, protein intake from dairy rather than meat was associated with BMI SDS (estimate of association NR; p=0.02). The analysis adjusted for TDEI, baseline BMI SDS, dietary fat intake (% TDEI), breastfeeding, maternal overweight and education.
- 3.232 Dougkas et al (2019) (AMSTAR 2 confidence rating: low) included 1 additional PCS in children aged 1 to 5 years that considered the impact of protein from dairy sources. The PCS (in 3564 participants) reported that every 10g of dairy protein intake per day at age 1 year was associated with an increase of 0.07 SD in BMI (95% CI 0.02 to 0.11; p<0.05) and an increase of 0.07 SD in body weight (kg) (95% CI 0.03 to 0.012; p<0.05) 8 years later. However, there were no differences in the association between dairy protein intake and BMI/body weight, and the

association between non-dairy protein intake and BMI/body weight (quantitative findings NR).

### **Animal protein intake and body fat**

- 3.233 Hörnell et al (2013) included 1 PCS (in 203 participants) that reported that higher intake of animal protein (as % TDEI) at age 1 year was associated with increased % body fat at age 7 years (estimate of association NR;  $p=0.01$ ). Protein from dairy rather than meat or cereals tended to be associated with % body fat (estimate of association NR;  $p=0.07$ ). The study adjusted for TDEI, child baseline % body fat, dietary fat intake (as % TDEI), breastfeeding, maternal overweight and education.

### **Vegetable protein intake and BMI**

- 3.234 Hörnell et al (2013) included 1 PCS (described in paragraph 3.233) that reported no association between vegetable protein intake (as % TDEI) at age 1 year and BMI at age 7 years (quantitative findings NR) in adjusted analyses.

### **Vegetable protein intake and body fat**

- 3.235 Hörnell et al (2013) included 1 PCS (described in paragraph 3.233) that reported no association between vegetable protein intake (as % TDEI) at age 1 year and % body fat at age 7 years (quantitative findings NR) in adjusted analyses.

### **Summary: protein intake and body composition or weight status**

- 3.236 The evidence identified from SRs on protein intake and body composition or weight status is summarised in Table 3.36.

**Table 3.36 Summary of the evidence on protein intake and body composition or weight status**

Exposure	Outcome	Direction of association <sup>1</sup>	Certainty of evidence
Total protein intake	Body Mass Index (BMI)	↑	Moderate
Total protein intake	Overweight	Not applicable	Insufficient
Total protein intake	Body fat	Not applicable	Insufficient
Animal protein intake	BMI	Not applicable	Insufficient
Animal protein intake	Body fat	Not applicable	Insufficient
Vegetable protein intake	BMI	Not applicable	Insufficient
Vegetable protein intake	Body fat	Not applicable	Insufficient

<sup>1</sup> Direction of association for reported outcomes: ↑increase

- 3.237 The available evidence from SRs on the relationship between protein intake in children aged 1 to 5 years and body composition or weight status is from 2 SRs without MAs, one given a moderate confidence rating using the AMSTAR 2 tool, the other given a critically low confidence rating.
- 3.238 Evidence from 5 PCS included in the SR by Hörnell et al (2013) and Dougkas et al (2019) suggests that higher total protein intake in children aged 1 to 5 years is associated with higher BMI in childhood compared with lower total protein intake. However, the role of TDEI in this relationship is unclear. The evidence was graded 'moderate'. There was 'insufficient' evidence from SRs to enable conclusions to be drawn on any relationship between total protein intake in children aged 1 to 5 years and later overweight as fewer than 3 primary studies included in the SRs examined this relationship.
- 3.239 There was 'insufficient' evidence from SRs to enable conclusions to be drawn on the relationships between protein intake from animal or vegetable sources in children aged 1 to 5 years and body composition or weight status as fewer than 3 primary studies included SRs examined these relationships.

## **Protein intake and growth outcomes**

- 3.240 The growth outcomes examined in this section are timing of adiposity rebound and peak linear growth velocity.

### **Protein intake and timing of adiposity rebound (AR)**

#### **Total protein intake and timing of AR**

- 3.241 Several growth patterns in early childhood have been linked to later adiposity or risk of obesity. Between the ages of 4 and 8 years, children typically experience a period when their BMI reaches a minimum level before increasing again (Brisbois et al, 2012). This is known as 'adiposity rebound' (AR). Many researchers have defined 'early adiposity rebound' as occurring before the age of 5 years (Brisbois et al, 2012) and observational evidence indicates that early AR may be associated with obesity in adulthood (see chapter 7 for details).
- 3.242 One SR without MA (Hörnell et al, 2013) was identified that examined the relationship between total protein intake in children and timing of AR.
- 3.243 Hörnell et al (2013) (AMSTAR 2 confidence rating: moderate) included 2 PCS in children aged 1 to 5 years. Both PCS (in a total of 1085 participants) reported no association between total protein intake (grams per day or as % TDEI) in children aged under 2 years and timing of AR (quantitative findings NR). One PCS adjusted for maternal BMI, gestational age and breastfeeding duration, as well as TDEI. The other study adjusted for sex only.
- 3.244 One of the 2 PCS (in 313 participants) also reported that total protein intake at ages 1 to 2 years was directly associated with BMI SDS at AR, but in girls only (quantitative findings NR). The study adjusted for TDEI, maternal BMI, child gestational age and breastfeeding duration.

### **Protein intake and peak linear growth velocity (PLGV)**

- 3.245 No SRs were identified that examined the relationship between total protein intake (from all sources) in children aged 1 to 5 years and peak linear growth velocity (PLGV).

#### **Animal protein intake and PLGV**

- 3.246 Hörnell et al (2013) included 1 PCS that examined the relationship between animal protein intake in children aged 1 to 5 years and PLGV. The PCS (in 67 girls) reported that animal protein intake (adjusted for age and TDEI, expressed as log-scale residuals) at ages 3 to 5 years predicted greater PLGV (cm per year), defined in the study as the most growth in height attained in a single adolescent year (quantitative findings NR). The study adjusted for age- and energy-adjusted intakes of dietary fat and vegetable protein, BMI and age-specific height z-scores

at ages 1 to 5 years. However, participants were born in the 1930s and 1940s when nutrition and lifestyle factors may have been different from today, potentially limiting the generalisability of this finding. The study also had a low participant retention rate (<60%), which is a potential source of bias.

### Vegetable protein and PLGV

3.247 No evidence from SRs was identified on the relationship between vegetable protein intake and age at PLGV in children aged 1 to 5 years.

### Summary: protein intake and growth outcomes

3.248 The evidence identified from SRs on protein intake and growth outcomes is summarised in Table 3.37.

**Table 3.37 Summary of the evidence on protein intake and growth**

Exposure	Outcome	Direction of association	Certainty of evidence
Total protein intake	Age at adiposity rebound	Not applicable	Insufficient
Total protein intake	Body Mass Index (BMI) at adiposity rebound	Not applicable	Insufficient
Total protein intake	Peak linear growth velocity	Not applicable	No systematic review evidence identified
Animal protein intake	Peak linear growth velocity	Not applicable	Insufficient
Vegetable protein intake	Peak linear growth velocity	Not applicable	No systematic review evidence identified

3.249 The available evidence from SRs examining the relationship between protein intake in children aged 1 to 5 years and growth outcomes is from 1 SR without MA, given a moderate confidence rating using the AMSTAR 2 tool.

3.250 There was 'insufficient' evidence from SRs to enable conclusions to be drawn on any relationship between total protein intake and animal protein intake in children aged 1 to 5 years and growth outcomes as fewer than 3 primary studies included in the SRs examined this relationship.

3.251 No evidence from SRs was identified on vegetable protein intake and growth outcomes in children aged 1 to 5 years.

## **Protein intake and timing of puberty**

### **Protein intake and age of menarche or voice break**

- 3.253 The contribution of genetics to the timing of menarche is estimated to be about 57 to 82% (Yermachenko & Dvornyk, 2014). Despite the apparent major role of genetic factors in timing of menarche, multiple non-genetic determinants of the timing of menarche have also been proposed. This includes the existence of a 'critical period' in early childhood during which higher protein intake (such as through infant formula feeding) influences pubertal timing through promoting rapid weight gain in the first year of life and later risk of obesity (SACN, 2018). It is well established that body size is associated with age of menarche (Dossus et al, 2012). How long the critical period lasts, and the relative importance of protein intake in infancy compared with in young childhood, is unclear.
- 3.254 Epidemiological studies have linked earlier age of menarche (and later menopause) to the development of breast cancer through longer exposure to oestrogens (Collaborative Group on Hormonal Factors in Breast Cancer, 2012), which makes this a potential public health issue.
- 3.255 For this report, 1 SR without MA (Hörnell et al, 2013) was identified that examined the relationship between protein intake in young children and pubertal timing.

### **Total protein intake and age of menarche**

- 3.256 Hörnell et al (2013) (AMSTAR 2 confidence rating: moderate) included 1 PCS (in 3298 participants) that reported that total protein intake (grams per day) at ages 3 to 4 years was associated with reaching menarche by age 12 years and 8 months, a cut-off determined by the primary study authors (quantitative findings NR). However, it is unclear whether the analyses adjusted for confounding factors.

### **Animal protein intake and age of menarche or voice break**

- 3.257 Hörnell et al (2013) included 3 PCS that examined the relationship between animal protein intake in children aged 1 to 5 years and age of menarche or voice break.
- 3.258 All 3 PCS (in a total of 3457 participants) reported an inverse association between animal protein intake at ages 3 to 5 years and age of menarche or voice break, although in 1 PCS (in 92 participants) the association did not reach statistical significance (estimate of association NR;  $p=0.06$ ), and quantitative findings were not reported for a second PCS.
- 3.259 One of the 3 PCS (in 67 participants) reported that girls aged 3 to 5 years with an animal protein intake 1 standard deviation (SD) above the mean (approximately 8g per day) reached menarche 0.63 years earlier than girls with an animal protein intake 1 SD below the mean. The study adjusted for age- and energy-adjusted intakes of dietary fat and vegetable protein, BMI and age-specific height z-scores

at ages 1 to 5 years. However, participants were born in the 1930s and 1940s when nutrition and lifestyle factors may have been different from today, potentially limiting the generalisability of this finding. The study also had a low participant retention rate (<60%), which is a potential source of bias.

- 3.260 Two of the 3 PCS adjusted for TDEI or a measure of body size. For the third PCS, it is unclear whether the analyses reported in Hörnell et al (2013) were adjusted.
- 3.261 Two of the 3 PCS also examined the impact of protein intake from meat or dairy products. One of these PCS (in 92 participants) reported that protein intake from cows' milk rather than meat (% TDEI) at ages 3 to 4 years tended to be inversely associated with age of menarche or voice break (estimate of association NR;  $p=0.06$ ). The other PCS (in 3298 participants) reported that meat intake (portions per week) and not dairy intake (units NR) at age 3 years was associated with a greater odds of menarche by age 12 years and 8 months (quantitative findings NR).

#### **Vegetable protein intake and age of menarche or voice break**

- 3.262 Hörnell et al (2013) included 2 PCS that examined the relationship between vegetable protein intake and age of menarche or voice break. One PCS (in 67 participants) reported an association between higher vegetable protein intake (in grams per day) at ages 3 to 5 years and later age at menarche; and the other PCS (in 92 participants) reported an association between higher vegetable protein intake (in grams or as % TDEI) at age 3 to 5 years and later age at menarche or voice break. Quantitative findings were not reported for either study. Both studies adjusted for TEI or a measure of body size. The limitations of the study in 67 participants are described in paragraph 3.259.

#### **Protein intake and age of onset of pubertal growth spurt**

- 3.263 The age of onset of pubertal growth spurt is the age at which linear growth velocity is at its minimum before pubertal linear growth takes off (Gunther et al, 2010).

#### **Total protein intake and age of onset of pubertal growth spurt**

- 3.264 No evidence from SRs was identified on the relationship between total protein intake and age of onset of pubertal growth spurt.

#### **Animal protein intake and age of onset of pubertal growth spurt**

- 3.265 Hörnell et al (2013) included 1 PCS (in 112 participants) that reported that children in the highest tertile of animal protein intake (as % TDEI) at ages 3 to 4 years experienced an earlier onset of pubertal growth (mean age 9.0 years; 95% CI 8.7 to 9.3) than children in the lowest tertile of animal protein intake (mean age 9.7 years; 95% CI 9.4 to 10.0) ( $p<0.05$  for the difference between highest and lowest

tertiles). The analysis adjusted for TDEI, sex, breastfeeding duration, rapid weight gain in infancy, and parental education status.

### **Vegetable protein intake and age of onset of pubertal growth spurt**

- 3.266 Hörnell et al (2013) included 1 PCS (described in paragraph 3.265) that reported that children in the highest tertile of vegetable protein intake (as % TDEI) at ages 3 to 4 years experienced a later pubertal growth spurt (mean age 9.6; 95% CI 9.2 to 9.9) compared with children in the lowest tertile of vegetable protein intake (mean age 9.1; 95% CI 8.8 to 9.4) (p-trend across tertiles =0.01) in adjusted analyses.

### **Protein intake and age at peak linear growth velocity (PLGV)**

#### **Total protein intake and age at PLGV**

- 3.267 No evidence from SRs was identified on the relationship between total protein intake and age at PLGV.

#### **Animal protein intake and age at PLGV**

- 3.268 Hörnell et al (2013) included 1 PCS (in 112 participants) that reported that children in the highest tertile of animal protein intake (as % TDEI) at ages 3 to 4 years experienced PLGV at an earlier age (mean 12.0 years; 95% CI 11.7 to 12.3) compared with children in the lowest tertile of animal protein intake (mean 12.5 years; 95% CI 12.2 to 12.9; p<0.05 for difference). The analysis adjusted for TDEI, sex, breastfeeding duration, rapid weight gain in infancy, and parental education status.

#### **Vegetable protein intake and age at PLGV**

- 3.269 Hörnell et al (2013) included 1 PCS (described in paragraph 3.268) that reported that children in the highest tertile of vegetable protein intake (as % TDEI) at ages 3 to 4 years experienced PLGV at a later age (mean 12.6 years; 95% CI 12.3 to 13.0) compared with children in the lowest tertile of vegetable protein intake (mean 12.1 years; 95% CI 11.8 to 12.5) (p-trend = 0.02) in adjusted analyses.

### **Summary: protein intake and timing of puberty**

- 3.270 The evidence identified from SRs on protein intake and timing of puberty is summarised in Table 3.38.

**Table 3.38 Summary of the evidence on protein intake and timing of puberty**

Exposure	Outcome	Direction of association <sup>1</sup>	Certainty of evidence
Total protein intake	Age of menarche	Not applicable	Insufficient
Animal protein intake	Age of menarche or voice break	↓	Limited
Vegetable protein intake	Age of menarche or voice break	Not applicable	Insufficient
Total protein intake	Age of onset of pubertal growth	Not applicable	No systematic review evidence identified
Animal protein intake	Age of onset of pubertal growth	Not applicable	Insufficient
Vegetable protein intake	Age of onset of pubertal growth	Not applicable	Insufficient
Total protein intake	Age at peak linear growth velocity	Not applicable	No systematic review evidence identified
Animal protein intake	Age at peak linear growth velocity	Not applicable	Insufficient
Vegetable protein intake	Age at peak linear growth velocity	Not applicable	Insufficient

<sup>1</sup> Direction of association for reported outcomes: ↓inverse

- 3.271 The available evidence from SRs on protein intake in children aged 1 to 5 years and timing of puberty is from 1 SR without MA, given a moderate confidence rating using the AMSTAR 2 tool.
- 3.272 Evidence from 3 PCS included in the SR by Hörnell et al (2013) suggests that higher animal protein intake in children aged 1 to 5 years is associated with earlier menarche or voice break. The evidence was graded 'limited' given the small number and size of the PCS identified.
- 3.273 There was 'insufficient' evidence from SRs to enable conclusions to be drawn on any relationship between protein intake (total, animal or vegetable) in children aged 1 to 5 years and other outcomes related to timing of puberty fewer than 3 primary studies included in the SR examined these relationships.

## Protein intake and other health outcomes

### Protein intake and blood lipids

- 3.274 One SR without MA (Voortman et al, 2015b) was identified that examined the relationship between total protein intake in childhood and blood lipids.
- 3.275 Voortman et al (2015b) (AMSTAR 2 confidence rating: low) included 1 PCS (in 389 participants) in children aged 1 to 5 years that reported no association between total protein intake (grams per day) at age 18 months and serum total cholesterol, LDL cholesterol, HDL cholesterol or triacylglycerol at age 31 months. The analysis adjusted for TDEI and intakes of saturated fats and PUFA (it is unclear whether intakes of these macronutrients were expressed as % TDEI or in absolute amounts).
- 3.276 No evidence from SRs was identified on the relationship between sources of protein (animal or vegetable) and blood lipids in children aged 1 to 5 years.

### Summary: protein intake and blood lipids

- 3.277 The evidence identified from SRs on protein intake and blood lipids in children aged 1 to 5 years is summarised in Table 3.39.

**Table 3.39 Summary of the evidence on protein intake and blood lipids**

Exposure	Outcome	Direction of association	Certainty of evidence
Total protein intake	Blood lipids	Not applicable	Insufficient
Animal protein intake	Blood lipids	Not applicable	No systematic review evidence identified
Vegetable protein intake	Blood lipids	Not applicable	No systematic review evidence identified

- 3.278 The available evidence from SRs on protein intake in children aged 1 to 5 years and timing of puberty comes from 1 SR without MA, given a low confidence rating using the AMSTAR 2 tool.
- 3.279 There was 'insufficient' evidence from SRs to enable conclusions to be drawn on any relationship between protein intake (total, animal or vegetable) in children aged 1 to 5 years and blood lipids, as fewer than 3 primary studies included in the SRs examined these relationships.

3.280 No evidence from SRs was identified on the relationship between protein intake from animal or vegetable sources and blood lipids in children aged 1 to 5 years.

### **Protein intake and bone health**

3.281 Protein intake may have a stronger relationship with bone health in childhood compared with bone health in adulthood due to the involvement of amino acids and nutritionally-regulated hormones, such as Insulin Growth Factor-1, in the ossification process of bone growth (Darling et al, 2019; Millward, 2021; Switkowski et al, 2019).

3.282 For this report, 1 SR without MA (Hörnell et al, 2013) was identified that examined the relationship between total protein intake and bone health in children.

3.283 Hörnell et al (2013) included 1 PCS (in 52 participants) in children aged 1 to 5 years that reported that average longitudinal total protein intake (in grams, source unspecified) from the ages of 2 to 8 years was associated with higher bone mineral content and bone mineral density at age 8 years (estimate of association NR;  $p \leq 0.05$ ). However, it is unclear whether the analysis adjusted for potential confounding factors, such as intakes of other dietary constituents, particularly isoflavones in soy protein, dietary fat or iron in meat, and calcium; and physical activity (Darling et al, 2019).

3.284 No evidence from SRs was identified that examined the relationship between sources of protein intake (animal or vegetable) and bone health in children aged 1 to 5 years.

### **Summary: protein intake and bone health**

3.285 The evidence identified from SRs on protein intake and bone health in children aged 1 to 5 years is summarised in Table 3.40.

**Table 3.40 Summary of the evidence on protein intake and bone health**

Exposure	Outcome	Direction association	Certainty of evidence
Protein intake	Bone health	Not applicable	Insufficient
Animal protein intake	Bone health	Not applicable	No systematic review evidence identified
Vegetable protein intake	Bone health	Not applicable	No systematic review evidence identified

3.286 The available evidence from SRs on protein intake in children aged 1 to 5 years and bone health comes from 1 SR without MA, given a moderate confidence rating using the AMSTAR 2 tool.

- 3.287 There was ‘insufficient’ evidence from SRs to enable conclusions to be drawn on any relationship between protein intake in children aged 1 to 5 years and bone health, as fewer than 3 primary studies included in the SR examined this relationship.
- 3.288 No evidence from SRs was identified on the relationship between protein intake from animal or vegetable sources and bone health in children aged 1 to 5 years.

### **Protein intake and neurodevelopment**

- 3.289 Protein is among several nutrients that are of particular importance for pre- and postnatal brain development. Protein is involved in forming the anatomical structure of the brain, neurotransmitter function, and mitochondrial health, which supports energy-taxing processes of the brain (Georgieff et al, 2018). The role of protein in brain development is closely associated with its role in supporting adequate growth prenatally and in early infancy (Georgieff et al, 2018). Pre-clinical and human studies have demonstrated that protein deficiency in early life results in life-long brain dysfunction (Georgieff et al, 2018).
- 3.290 For this report, 1 SR without MA (Hörnell et al, 2013) was identified that included studies that examined the relationship between protein intake and neurodevelopment.

### **Total protein intake and neurodevelopment**

- 3.291 Hörnell et al (2013) (AMSTAR 2 confidence rating: moderate) included 1 PCS (in 496 participants) in children aged 1 to 5 years that reported that higher total protein intake (as % TDEI) at age 4 years predicted favourable performance on gross motor function and perception tests at age 5 years in boys only (quantitative findings NR). Analyses were stratified by sex but were not adjusted for other potential confounding factors, such as socioeconomic status.

### **Summary: protein intake and neurodevelopment**

- 3.292 The evidence identified from SRs on protein intake and neurodevelopment in children aged 1 to 5 years is summarised in Table 3.41.

**Table 3.41 Summary of the evidence on protein intake and neurodevelopment**

Exposure	Outcome	Direction of association	Certainty of evidence
Total protein intake	Neurodevelopment	Not applicable	Insufficient

- 3.293 The available evidence from SRs on protein intake in children aged 1 to 5 years and neurodevelopment comes from 1 SR without MA, given a moderate confidence rating using the AMSTAR 2 tool.
- 3.294 There was 'insufficient' evidence from SRs to enable conclusions to be drawn on any relationship between total protein intake in children aged 1 to 5 years and neurodevelopment as fewer than 3 primary studies included in the SRs examined these relationships.

# 4 Micronutrients

## Background

- 4.1 The SACN report [‘Feeding in the first year of life’](#) (SACN, 2018) identified iron, vitamin A and vitamin D as key micronutrients of concern due to potential deficiency or excess during infancy.
- 4.2 In relation to iron, SACN (2018) concluded that iron status at birth is the most important determinant of iron status throughout infancy. For healthy, term infants of appropriate weight born with adequate iron stores, exclusive breastfeeding during the first 6 months of life provides sufficient dietary iron. However, a diverse complementary diet is needed to meet the increased iron requirements of infants beyond the age of 6 months (SACN, 2018). SACN (2018) also concluded that there was substantial evidence that consumption of unmodified cows’ milk as a main drink by infants before their first birthday is associated with lower iron status and that iron supplements in infancy are not protective against future iron deficiency but may have a detrimental effect on linear growth (SACN, 2010; SACN, 2018).
- 4.3 In relation to vitamin D, SACN recommends Safe Intakes (see Glossary) for infants and children aged up to 4 years in the range of 8.5 to 10 µg per day (340 to 400 IU per day) for all infants from birth up to 1 year and 10 µg per day (400 IU per day) beyond age 1 year (SACN, 2016).
- 4.4 SACN (2018) noted that ample vitamin A is supplied by the average UK diet, but a risk of exceeding the tolerable upper limit (TUL) was identified for some infants who habitually consume dietary supplements containing vitamin A in addition to large amounts of fortified foods, including formula (see Glossary). Vitamin A is also included in Healthy Start vitamins for children under 4 years (see Annex 1, Table A1.2 for details).
- 4.5 As a continuation of SACN (2018), the aim of this chapter was to address whether micronutrient intakes and status in children aged 1 to 5 years in the UK were adequate, and if not, which age or population groups were most at risk and why.
- 4.6 While a wide range of micronutrients could have been considered, SACN chose to focus on iron, zinc and vitamins A and D. As described later in this chapter, national dietary surveys in the UK (the Diet and Nutrition Survey of Infants and Young Children [DNSIYC] and the National Diet and Nutrition Survey [NDNS]) have shown that there are proportions of children (greater than 5%) who may be at risk of inadequate intakes of these micronutrients (for DNSIYC and NDNS data on all nutrients that were surveyed, see Annex 11, Table A11.3). Concerns around the adequacy of intakes are supported by NDNS data on the iron, vitamin A and

vitamin D status (blood markers) of children aged 1 to 5 years (there is no equivalent status data or suitable biomarker for zinc which adds uncertainty to estimates of the proportion of children at risk of zinc insufficiency). Additional analysis of NDNS data also indicated that children from lower socioeconomic backgrounds and certain ethnic groups may be more at risk of micronutrient deficiency.

- 4.7 This chapter provides an overall assessment of intake levels, dietary contributors to intakes and status measures for iron, zinc, vitamin A and vitamin D in children aged 1 to 5 years followed by an assessment of the systematic review (SR) evidence identified on the health impact of each of these micronutrients for this age group.
- 4.8 In addition to the above micronutrients, the committee also considered whether there was any new evidence on the short and long-term health impact of high sodium (salt) intakes in children aged 1 to 5 years given the paucity of evidence in this age group when SACN last reviewed recommendations for salt intake in 2003 (SACN, 2003).
- 4.9 The committee also noted that consideration of vitamin C intake in children aged 1 to 5 years was warranted because in the UK, it is recommended that all children aged 6 months to 5 years are given vitamin supplements containing vitamins A, C and D (see Annex 1, Table A1.1). Vitamins A, C and D are also provided by the Healthy Start vitamin scheme in England, Wales and Northern Ireland, while vitamin D is provided under the Scottish Vitamins Scheme in Scotland (see Annex 1, Table A1.2).
- 4.10 The committee also recognised that the shift towards adopting plant-based diets (including vegetarian or vegan diets) may raise additional nutrients of concern, such as calcium, iodine and vitamin B12, even if there was currently a lack of data from dietary surveys to link plant-based dietary patterns with inadequate nutritional intake and status in young children.
- 4.11 SACN has previously recommended that a public health approach to achieving adequate nutritional status should emphasise the importance of a healthy balanced diet that includes a variety of foods containing nutrients such as iron (SACN, 2010). However, for nutrients that are required in quantities greater than can be obtained from the diet alone (for example, vitamin D and folate), risk management strategies should be identified.

## **Limitations of the evidence on micronutrients**

- 4.12 The limitations described in paragraphs 4.13 to 4.19 relate to the NDNS data used in this chapter while those described in paragraph 4.21 relate to the SR evidence that was identified on micronutrients.

- 4.13 Each NDNS fieldwork year collects data on approximately 150 to 160 children aged 18 to 60 months as part of a wider annual sample of 500 children aged 18 months to 18 years and is designed to be representative of the UK population. However, the sample of children that provide blood samples for status measures is much smaller, typically 15 to 20 per year.
- 4.14 An analysis conducted on the characteristics of NDNS participants indicated that there were differences in the characteristics of children who gave a blood sample compared with the whole NDNS sample of children (see Annex 11, Tables A11.11 to A11.14 for details).
- 4.15 For children aged 18 to 47 months, girls made up a marginally higher proportion of children who gave a blood sample compared with their proportion of the whole sample (52.9% versus 48.8% of the whole sample). The youngest children surveyed (aged 18 to 23 months) were underrepresented in the group who gave a blood sample compared with their proportion of the whole sample (9.4% versus 14.8% of the whole sample). White children were underrepresented in the group who gave a blood sample (75.6% vs 80.5% of the whole sample) as were Asian and Asian British children (6.7% vs 8.4% of the whole sample).
- 4.16 For children aged 48 to 60 months, the proportion of children who gave a blood sample based on their age, sex and ethnic group roughly matched the age, sex and ethnic group breakdown of the whole sample.
- 4.17 Children aged 18 to 60 months who gave a blood sample were more likely to come from higher socioeconomic status households (where the Household Reference Person [HRP] worked in higher managerial and professional occupations).
- 4.18 Misreporting of food consumption, specifically underreporting, and therefore underestimation of total dietary energy intake (TDEI) (known as underreporting) in self-reported dietary methods is a well-documented source of bias and is an important consideration when interpreting NDNS data. To assess the level of underreporting of TDEI, the ratio of reported TDEI to basal metabolic rate (BMR) (TDEI:BMR) was calculated for each child (Annex 11, Table A11.15). The analysis indicated evidence of underreporting of TDEI, particularly among the children with intakes below the lower reference nutrient intake (LRNI) for iron, zinc and vitamin A compared with the children with intakes at or above the LRNI and the reference nutrient intake (RNI) (see Glossary, 'Dietary Reference Values'). Underreporting of TDEI has been defined as TDEI:BMR of less than 1.35 (in adults), with normal reporting of dietary intake as TDEI:BMR of 1.35 to 2.39 (Mirmiran et al, 2006; Sichert-Hellert et al, 1998). For children aged 18 to 47 months with intakes below the LRNI for zinc, vitamin A or iron, the reported TDEI:BMR ranged from 0.94 for zinc to 1.03 for vitamin A and 1.12 for iron. These values are not plausible and are therefore unlikely to represent habitual dietary intakes. However, the extent to which energy underreporting affects the assessment of vitamin and mineral intakes is not known.

- 4.19 Additionally, body weight z-scores (see Glossary) of children with intakes of iron, zinc or vitamin A below, at or above the LRNI for these micronutrients were compared in order to examine whether the children with intakes below the LRNI were physically smaller and therefore had lower energy requirements than the children with intakes at or above the LRNI (Annex 11, Table A11.15). Body weight z-scores of children with intakes below the LRNI for iron and zinc were generally smaller than children with intakes at or above the LRNI for these micronutrients. This indicates that the lower intakes reported in the former group of children may not have been solely due to underreporting.
- 4.20 The LRNI is set at the lowest 2.5<sup>th</sup> percentile of the distribution of nutrient requirements and represents a level below which intakes are almost certainly inadequate for most individuals (DH, 1991). Due to the evidence of underreporting of TDEI, particularly among the children with intakes below the LRNI for vitamin A, iron and zinc, it is difficult to be fully confident in the estimates of micronutrient intakes in this group.
- 4.21 The evidence identified from SRs to inform this chapter did not directly address the question of improving diets and health outcomes in the UK or other high income countries (HICs) (defined according to the [World Bank classification system](#)). There was a paucity of SR evidence identified on vitamin D and no SR evidence was identified on vitamin C. At the same time, the SR evidence that was identified on iron, zinc and vitamin A was drawn exclusively from supplementation and food fortification trials, many of which were designed for populations in low income (LICs), lower-middle (LMICs) or upper-middle income (UMICs) countries (defined according to the [World Bank classification system](#)). While findings from these trials can be useful in understanding health inequalities in HICs, they can also be confounded by the existence of multiple micronutrient deficiencies, infectious diseases (such as malaria) and levels of inflammation that are not seen in the UK, thereby limiting their generalisability to the UK context.

## Approach to grading the evidence for this chapter

- 4.22 Due to the limitations highlighted in paragraph 4.21, the committee decided that only SR evidence that was most relevant to the UK context should be graded (see [Grading of the evidence from systematic reviews](#) in chapter 2) and used to inform the conclusions of this chapter. Accordingly, evidence for the following population subgroups, interventions and health outcomes was graded if available:
- population stratification: children with adequate micronutrient status at baseline versus children with inadequate status at baseline
  - interventions: supplementation trials for vitamins A and D (given current UK government advice on supplementation in young children; see Annex 1, Table

A1.1) and fortification trials for iron, vitamin A, vitamin D (given mandatory or voluntary fortification of foods with these micronutrients in the UK)

- outcomes: micronutrient status measures, growth, cognitive development, morbidities (including diarrhoea, fever, vomiting, respiratory infection).

- 4.23 Evidence was graded if findings were stratified by intervention type and baseline nutritional status (paragraph 4.22). This is because the effectiveness of supplementation compared with fortification strategies to improve nutritional status and related health outcomes can be expected to differ (SACN, 2010). At the same time, the effectiveness of an intervention (supplementation or fortification) can be expected to differ depending on the baseline nutritional status of participants. For example, while supplementing children with a micronutrient deficiency may improve health outcomes, supplementing children with adequate micronutrient status may actually lead to adverse health outcomes (paragraph 4.126).
- 4.24 Evidence that was not graded has been summarised in this chapter as it can still offer insights into the physiological basis underpinning deficiency and deficiency-related health outcomes.
- 4.25 Details of the SRs included in this section can be found in Annex 5 (Table A5.2). Quality assessment of the SRs using the AMSTAR 2 tool can be found in Annex 8 (Table A8.3). Additional data extracted on the primary studies can be found in Annex 9 (Table A9.11 to A9.22). The criteria used to grade the evidence are provided in chapter 2 (Table 2.4, paragraphs 2.54 to 2.58). Summary tables of the evidence grading process for this section are provided in Annex 9 (Tables A9.8, A9.10 and A10.36).

## **Dietary contributors to iron, zinc and vitamin A intakes in children with intakes at or above dietary recommendations**

- 4.26 This section considers the dietary intake of children aged 18 to 60 months in the UK with intakes at or above dietary recommendations for the nutrients of concern (iron, zinc and vitamin A) and examines the main dietary contributors to iron, zinc and vitamin A intake for these children.
- 4.27 As vitamin D requirements cannot be met through the diet alone, there is no entry for vitamin D in this section.

### **Iron**

- 4.28 Dietary contributors to mean daily iron intake in children aged 18 to 47 months and aged 48 to 60 months with intakes at or above the RNI for iron, zinc and vitamin A collectively are presented in Table 4.1 and Table 4.2, respectively.
- 4.29 In both age groups, nearly 40% of iron intake came from breakfast cereals and bread (see paragraphs 4.76 and 4.77 for details on fortification in the UK). In the younger age group (age 18 to 47 months), formula milks (mainly follow-on formula and milks marketed to children over the age of 1 year, also known as 'toddler milks' and 'growing-up milks') contributed 10.7% to mean iron intake. In both age groups, sources of haem iron (see paragraph 4.53), which is almost entirely from foods of animal origin, contributed 9% to 10% of iron intake. Iron-containing dietary supplements contributed a further 7% to 9% in both age groups.

**Table 4.1 Contributors to mean daily iron intake in children aged 18 to 47 months with intakes at or above the RNI<sup>1</sup> for iron, zinc and vitamin A (NDNS years 2008 to 2019)**

Food group	% contribution <sup>2,3</sup>	mg per day
Breakfast cereals	22.2	2.0
Formula milks <sup>4</sup>	10.7	1.1
Bread	10.3	0.9
Meat, meat products and dishes	9.3	0.8
Vegetables, vegetable products and dishes	7.1	0.7
Dietary supplements	6.5	0.7
Biscuits, buns, cakes, pastries, fruit pies, puddings	6.5	0.6
Fruit	5.1	0.5
Pizza, pasta, rice, products and dishes	4.5	0.4
Potatoes, potato products and dishes	2.7	0.2
Eggs, egg products and dishes	2.4	0.2
Commercially manufactured foods and drinks specifically marketed for infants and young children	1.9	0.2
Fish, fish products and dishes	1.7	0.1
Fruit juice and smoothies	1.2	0.1
Confectionery	1.0	0.1
Soup	1.0	0.1
Number of participants	254	254

Abbreviations: RNI, reference nutrient intake

Data from NDNS years 2008 to 2019.

<sup>1</sup> RNI for iron (ages 1 to 3 years: 6.9mg per day; ages 4 to 6 years: 6.1mg per day); zinc (ages 1 to 3 years: 5.0mg per day; ages 4 to 6 years: 6.5mg per day); vitamin A (ages 1 to 6 years: 400 retinol equivalents µg per day).

<sup>2</sup> Food groups that contributed less than 1% to iron, zinc and vitamin A intakes are not presented.

<sup>3</sup> Average % contribution for each food group has been calculated from the % contribution for each individual. Non-consumers are included in the average.

<sup>4</sup> Formula milks include milks marketed to children over the age of 1 year, also known as 'toddler milks' and 'growing-up milks' (see Glossary).

**Table 4.2 Contributors to mean iron intake in children aged 48 to 60 months with intakes at or above the RNI<sup>1</sup> for iron, zinc and vitamin A (NDNS years 2008 to 2019)**

Food group	% contribution <sup>2,3</sup>	mg per day
Breakfast cereals	23.9	2.5
Bread	12.5	1.1
Meat, meat products and dishes	10.3	0.9
Biscuits, buns, cakes, pastries, fruit pies puddings	8.7	0.8
Dietary supplements	8.6	1.3
Vegetables, vegetable products and dishes	7.1	0.7
Pizza, pasta, rice products and dishes	5.7	0.6
Fruit	4.5	0.4
Eggs, egg products and dishes	2.4	0.2
Commercially manufactured foods and drinks specifically marketed for infants and young children	2.1	0.2
Potatoes, potato products and dishes	1.9	0.2
Fruit juice and smoothies	1.7	0.2
Confectionery	1.7	0.1
Fish, fish products and dishes	1.5	0.1
Yoghurt, fromage frais, dairy desserts <sup>4</sup>	1.2	0.1
Savoury sauces pickles gravies and condiments	1.0	0.1
Soup	1.0	0.1
Number of participants	71	71

Abbreviations: RNI, reference nutrient intake

Data from NDNS years 2008 to 2019.

<sup>1</sup> RNI for iron (ages 1 to 3 years: 6.9mg per day; ages 4 to 6 years: 6.1mg per day); zinc (ages 1 to 3 years: 5.0mg per day; ages 4 to 6 years: 6.5mg per day); vitamin A (ages 1 to 6 years: 400 retinol equivalents µg per day).

<sup>2</sup> Food groups that contributed less than 1% to iron, zinc and vitamin A intakes are not presented.

<sup>3</sup> Average % contribution for each food group has been calculated from the % contribution for each individual. Non-consumers are included in the average.

<sup>4</sup> Includes non-dairy alternatives.

## Zinc

4.30 Dietary contributors to mean zinc intake in children aged 18 to 47 months and aged 48 to 60 months with intakes at or above the RNI for zinc, iron and vitamin A collectively are presented in Table 4.3 and Table 4.4, respectively. Over a third of zinc intake in both age groups came from meat and milk. In the younger age group (age 18 to 47 months), infant formula contributed 10.1% to zinc intake while in the older age group (age 48 to 60 months), zinc-containing dietary supplements contributed nearly 11.7%.

**Table 4.3 Contributors to mean zinc intake in children aged 18 to 47 months with intakes at or above the RNI<sup>1</sup> for zinc, iron and vitamin A (NDNS years 2008 to 2019)**

Food group	% contribution <sup>2,3</sup>	mg per day
Meat, meat products and dishes	17.6	1.21
Milk <sup>4</sup>	15.5	1.04
Formula milks <sup>5</sup>	10.1	0.77
Bread	7.8	0.52
Dietary supplements	6.3	0.59
Breakfast cereals	5.2	0.34
Pizza, pasta, rice products and dishes	5.0	0.35
Cheese <sup>3</sup>	5.0	0.33
Vegetables, vegetable products and dishes	4.4	0.30
Biscuits, buns, cakes, pastries, puddings	4.1	0.27
Yoghurt, fromage frais and dairy desserts <sup>4</sup>	3.8	0.26
Fruit	2.6	0.18
Eggs, egg products and dishes	2.3	0.16
Potatoes, potato products and dishes	1.9	0.12
Fish, fish products and dishes	1.7	0.11
Commercially manufactured foods and drinks specifically marketed for infants and young children	1.6	0.12
Number of participants	254	254

Abbreviations: RNI, reference nutrient intake

Data from NDNS years 2008 to 2019.

<sup>1</sup> RNI for iron (ages 1 to 3 years: 6.9mg per day; ages 4 to 6 years: 6.1mg per day); zinc (ages 1 to 3 years: 5.0mg per day; ages 4 to 6 years: 6.5mg per day); vitamin A (ages 1 to 6 years: 400 retinol equivalents µg per day).

<sup>2</sup> Food groups that contributed less than 1% to zinc, iron and vitamin A intake are not presented.

<sup>3</sup> Average % contribution for each food group has been calculated from the % contribution for each individual. Non-consumers are included in the average.

<sup>4</sup> Includes non-dairy alternatives.

<sup>5</sup> Formula milks include milks marketed to children over the age of 1 year, also known as 'toddler milks' and 'growing-up milks' (see Glossary).

**Table 4.4 Contributors to zinc intake in children aged 48 to 60 months with intakes at or above the RNI<sup>1</sup> for zinc, iron, and vitamin A (NDNS years 2008 to 2019)**

Food Group	% contribution <sup>2,3</sup>	mg per day
Meat, meat products and dishes	19.3	1.59
Milk <sup>4</sup>	16.1	1.40
Dietary supplements	11.7	1.47
Bread	8.0	0.67
Pizza, pasta, rice, products and dishes	6.3	0.52
Cheese <sup>4</sup>	6.1	0.49
Breakfast cereals	5.1	0.42
Vegetables, vegetable products and dishes	4.5	0,38
Biscuits, buns, cakes, pastries, puddings	4.3	0.36
Yoghurt, fromage frais, dairy desserts <sup>4</sup>	3.7	0.30
Commercially manufactured foods and drinks specifically marketed for infants and young children	2.2	0.16
Fruit	2.1	0.18
Eggs, egg products and dishes	1.9	0.15
Potatoes, potato products and dishes	1.3	0.11
Fish, fish products and dishes	1.2	0.10
Number of participants	71	71

Abbreviations: RNI, reference nutrient intake

Data from NDNS years 2008 to 2019.

<sup>1</sup> RNI for iron (ages 1 to 3 years: 6.9mg per day; ages 4 to 6 years: 6.1mg per day); zinc (ages 1 to 3 years: 5.0mg per day; ages 4 to 6 years: 6.5mg per day); vitamin A (ages 1 to 6 years: 400 retinol equivalents µg per day).

<sup>2</sup> Food groups that contributed less than 1% to zinc, iron and vitamin A intake are not presented.

<sup>3</sup> Average % contribution for each food group has been calculated from the % contribution for each individual. Non-consumers are included in the average.

<sup>4</sup> Includes non-dairy alternatives.

## Vitamin A

- 4.31 Dietary contributors to mean vitamin A intake in children aged 18 to 47 months and aged 48 to 60 months with intakes at or above the RNI for vitamin A, iron and zinc, are presented in Table 4.5 and Table 4.6, respectively.
- 4.32 Carrots, milk and dietary supplements were the main contributors to vitamin A intake for both age groups. Meat, meat products and dishes, as well as formula milks were also major contributors in children aged 18 to 47 months.

**Table 4.5 Contributors to vitamin A intake in children aged 18 to 47 months with intakes at or above the RNI<sup>1</sup> for vitamin A, iron, and zinc (NDNS years 2008 to 2019)**

Food Group	% contribution <sup>2,3</sup>	µg per day
Carrots raw and cooked	15.5	155
Milk <sup>4</sup>	11.5	79
Dietary supplements	11.3	124
Meat, meat products and dishes	8.9	92
Formula milks <sup>5</sup>	8.5	66
Fat spreads	7.6	51
Vegetables, vegetable products and dishes (excl carrots)	6.8	50
Cheese <sup>4</sup>	4.8	33
Soup	3.5	33
Yoghurt, fromage frais and dairy desserts <sup>4</sup>	3.3	22
Biscuits, buns, cakes, pastries, fruit pies, puddings	3.2	22
Eggs, egg products and egg dishes	2.6	19
Pizza, pasta, rice, products and dishes	2.6	20
Commercially manufactured foods and drinks specifically marketed for infants and young children	2.0	19
Fruit	1.3	10
Soft drinks	1.1	7
Number of participants	254	254

Abbreviations: RNI, reference nutrient intake

Data from NDNS years 2008 to 2019.

<sup>1</sup> RNI for iron (ages 1 to 3 years: 6.9mg per day; ages 4 to 6 years: 6.1mg per day); zinc (ages 1 to 3 years: 5.0mg per day; ages 4 to 6 years: 6.5mg per day); vitamin A (ages 1 to 6 years: 400 retinol equivalents µg per day).

<sup>2</sup> Food groups that contributed less than 1% to vitamin A, zinc and iron intake are not presented.

<sup>3</sup> Average % contribution for each food group has been calculated from the % contribution for each individual. Non-consumers are included in the average.

<sup>4</sup> Includes non-dairy alternatives.

<sup>5</sup> Formula milks include milks marketed to children over the age of 1 year, also known as 'toddler milks' and 'growing-up milks' (see Glossary).

**Table 4.6 Contributors to vitamin A intake in children aged 48 to 60 months with intakes at or above the RNI<sup>1</sup> for vitamin A, iron, and zinc (NDNS years 2008 to 2019)**

Food group	% contribution <sup>2,3</sup>	µg per day
Carrots raw and cooked	19.9	226
Dietary supplements	17.1	202
Milk <sup>4</sup>	12.3	102
Vegetables, vegetable products and dishes (excluding carrots)	8.5	72
Butter and fat spreads	7.0	60
Cheese <sup>4</sup>	5.8	49
Meat, meat products and dishes	5.4	65
Biscuits, buns, cakes, pastries, fruit pies, puddings	4.5	35
Pizza, pasta, rice, products and dishes	3.5	30
Yoghurt, fromage frais, dairy desserts <sup>4</sup>	2.9	22
Eggs, egg products and dishes	2.1	17
Soup	1.7	20
Soft drinks	1.7	15
Fruit	1.4	12
Ice cream <sup>4</sup>	1.2	9
Commercially manufactured foods and drinks specifically marketed for infants and young children	1.1	12
Number of participants	71	71

Abbreviations: RNI, reference nutrient intake

Data from NDNS years 2008 to 2019.

<sup>1</sup> RNI for iron (ages 1 to 3 years: 6.9mg per day; ages 4 to 6 years: 6.1mg per day); zinc (ages 1 to 3 years: 5.0mg per day; ages 4 to 6 years: 6.5mg per day); vitamin A (ages 1 to 6 years: 400 retinol equivalents µg per day).

<sup>2</sup> Food groups that contributed less than 1% to vitamin A, zinc and iron intake are not presented.

<sup>3</sup> Average % contribution for each food group has been calculated from the % contribution for each individual. Non-consumers are included in the average.

<sup>4</sup> Includes non-dairy alternatives.

4.33 Table 4.1 to 4.6 show that the differences in dietary contributors to each micronutrient was greater between the micronutrients than between age groups (18 to 47 months and 48 to 60 months). That is, the largest contributors to each micronutrient were different between the micronutrients. But for each micronutrient, the contributors to that micronutrient were similar between the 2 age

groups. In addition, for each micronutrient, there was a clear main contributor to intake (for example, breakfast cereals for iron, carrots and milk for vitamin A and meat and milk for zinc).

- 4.34 Milk and dairy products contributed substantially to intakes of all 3 micronutrients (with the exception for iron in the older age group), with possible implications for children who avoid dairy due to restrictive diets or intolerance.
- 4.35 For children aged 18 to 47 months, formula milks (mainly follow-on formula and milks marketed to children over the age of 1 year, also known as 'toddler milks' and 'growing-up milks') and dietary supplements were also large contributors to intakes of all 3 micronutrients.
- 4.36 It is currently recommended that children aged 6 months to 5 years are given supplements containing vitamin A (and vitamins C and D) except when they consume more than 500ml of formula milk per day because formula milk (see Glossary) is fortified with vitamins A, C and D and other nutrients. Children who consume both formula milk and dietary supplements may be at risk of excess intakes of these micronutrients (COT, 2017).
- 4.37 Dietary supplements also contributed substantially to intakes of the 3 micronutrients in the older age group (9% to 17%).

# Iron

## Physiological requirements

- 4.38 From around 1 year of age, a diverse diet is needed to meet the increasing iron requirements of young children (SACN, 2018). For children aged over 3 years, iron is required to meet the needs for an expanding red cell mass, for growth, and to replace basal loss (SACN, 2010).

## Assessment of iron status

- 4.39 The term 'iron status' is used to describe whether an individual has too little, enough, or too much iron in their body for their needs as well as to indicate the possible risk of deficiency or excess (SACN, 2010). Iron deficiency (ID) is a state in which there is insufficient iron to maintain the normal physiological function of tissues, including the blood, brain and muscles (WHO/CDCP, 2004). Infants and young children are at particular risk of ID and subsequent anaemia due to the increased requirements associated with this period of rapid growth (McCarthy et al, 2017).
- 4.40 ID is conventionally considered to develop in 3 stages: iron depletion, iron-deficient erythropoiesis, and iron deficiency anaemia (IDA), a combination of ID and anaemia (Domellöf et al, 2014). In the first stage (iron depletion), body stores are reduced, which is typically measured using serum ferritin (corrected for high-sensitivity C-reactive protein [CRP], or other markers of inflammation). As iron depletion progresses, transferrin saturation decreases while soluble transferrin receptors increase. In the third stage, blood haemoglobin concentration is reduced, and red cell morphology is affected; the mean cell volume (MCV) also decreases while the red cell distribution width increases. A low MCV is not specific to iron deficiency as low values can indicate the presence of thalassaemia, a blood disorder, or anaemia due to inflammation (WHO/CDCP, 2004).
- 4.41 Serum ferritin and haemoglobin concentrations are commonly cited markers of iron status, but the thresholds to indicate deficiency have been much debated (for details see [SACN report 'Iron and Health'](#)). Serum ferritin concentration reflects systemic ferritin depots. Low serum ferritin concentrations represent low depots but may not represent a functional deficiency of iron (SACN, 2018).
- 4.42 There are many biomarkers of iron status, including transferrin saturation, soluble transferrin receptor, reticulocyte haemoglobin and hepcidin concentrations, but all have limitations in terms of their sensitivity and specificity (SACN, 2010) and the reference ranges and cut-offs for the different biomarkers are poorly defined in

young children Domellöf et al (2014). For more details, see SACN (2010), as well as Domellöf et al (2014), Hernell et al (2015) and McCarthy et al (2017).

## **Assessment criteria for IDA in young children**

- 4.43 Although no single marker of iron metabolism is considered ideal for the assessment of iron deficiency (or excess), in this report, a combination of haemoglobin (functional iron) and serum ferritin (iron depots) were considered to be the most useful indicators in agreement with international practice.
- 4.44 For children aged 6 to 60 months, the World Health Organization (WHO) recommends that haemoglobin concentrations below 110g/l indicate the presence of anaemia while serum ferritin values below 12µg/l indicate depleted iron stores (WHO, 2001c). However, serum ferritin is also an acute phase protein, which means that its concentration can rise during states of inflammation or infection, which can lead to potentially underestimating micronutrient deficiency in a population (Namaste et al, 2019). Methods of accounting for this in the presence of infection for children aged under 5 years include increasing the threshold for serum ferritin to <30µg/l (WHO/CDCP, 2004) or adjusting the concentrations of serum ferritin (or other iron biomarkers whose concentrations are affected by inflammation) by concentrations of markers of inflammation, such as CRP (Namaste et al, 2019). Presence of inflammation is usually defined as a CRP concentration of 5mg/l or higher (Namaste et al, 2019).
- 4.45 For this report, WHO cut-off values were used (SACN, 2010) as it is not within the scope of this risk assessment to review these markers and cut-offs.

## **Prevalence of ID and IDA in the UK**

- 4.46 ID is the most common micronutrient deficiency in the world (Domellöf et al, 2014), with ID prevalence in young European children ranging from 3 to 48% (Eussen et al, 2015). Prevalence of IDA is below 5% in European children aged 1 to 3 years, while approximately 25% of preschool children globally have IDA.
- 4.47 Table 4.7 presents NDNS data of iron status (ID, anaemia, IDA) of children aged 12 to 60 months in the UK. As the presence of infection or inflammation can result in elevated serum ferritin concentrations, an analysis was undertaken to assess whether levels of inflammation in the NDNS sample significantly affected mean values of the overall sample (Annex 10, Table A10.13). The analysis showed that excluding children with high CRP from the analysis had little impact on the overall prevalence of anaemia or IDA, suggesting that in this population of young children in health, adjustment for CRP levels was not necessary.
- 4.48 Table 4.7 shows that nearly 25% of children aged 18 to 47 months had ID, which is a finding of potential concern. However, the prevalence of IDA was much lower.

The prevalence of ID appears to increase with age while the prevalence of IDA appears to decrease with age. However, the small numbers of children aged 48 to 60 months with IDA precludes a more detailed analysis of this group and ability to draw firm conclusions.

**Table 4.7 Iron status (plasma ferritin, ID, anaemia, IDA) in children aged 12 to 60 months in the UK (DNSIYC and NDNS years 2008 to 2019)**

Age	Haemoglobin (g/l) <sup>1,2</sup>	Plasma ferritin (µg/l) <sup>1,3</sup>	% ID (plasma ferritin below 12µg/l)	% anaemia (Hb below 110g/l)	% IDA (% below thresholds for ferritin and Hb)
12 to 18 months <sup>4</sup>	117 (10)	28.3 (18.8)	11	15	2
18 to 47 months <sup>5</sup>	120 (82)	24.5 (18.7)	23.9	9.0	3.3
48 to 60 months <sup>5</sup>	123 (80)	29.1 (22.6)	20.0	7.2	[0.0] <sup>6</sup>

Abbreviations: Hb, haemoglobin; ID, iron deficiency; IDA, iron deficiency anaemia; SD, standard deviation

<sup>1</sup> Mean (SD).

<sup>2</sup> Number of participants: 325 (12 to 18 months), 140 (18 to 47 months), 58 (48 to 60 months).

<sup>3</sup> Number of participants: 298 (12 to 18 months), 117 (18 to 47 months), 53 (48 to 60 months).

<sup>4</sup> Data from DNSIYC 2011 (DH, 2013).

<sup>5</sup> Data from NDNS years 2008 to 2019.

<sup>6</sup> Data for a variable with a cell size between 30 to 49 are presented in square brackets.

## **Non-dietary determinants of iron status**

- 4.49 Non-dietary risk factors for ID and IDA in European infants and toddlers include low birth weight, early cord clamping, male sex and low socioeconomic status (Domellöf et al, 2014).
- 4.50 Iron status at birth is the most important determinant of iron status throughout infancy; cord blood ferritin concentrations are correlated with ferritin concentrations until at least 2 years of age (Georgieff et al, 2002; Hay et al, 2007).
- 4.51 Factors associated with lower iron status at birth include low birthweight, maternal IDA, and other indicators of pregnancy risk including maternal obesity, smoking and gestational hypertension (SACN, 2018).
- 4.52 In lower income countries, haemolysis caused by malaria (Fleming, 1981) (WHO, 2000) and intestinal blood loss caused by helminthiasis (Crompton & Nesheim, 2002; Roche & Layrisse, 1966) are also major causes of anaemia but are of less relevance to the UK population.

## **Dietary determinants of iron status**

### **Current recommendations for iron intake in the UK**

- 4.53 Dietary iron exists in 2 main forms: haem iron and non-haem iron. Haem iron is found almost entirely in food of animal origin, while non-haem iron is found in animal and plant tissues. The richest sources of non-haem iron include cereals, vegetables, nuts, eggs, fish and meat (SACN, 2010). Haem iron, if there is a systemic need for iron in the body, is absorbed more efficiently than non-haem iron and is considered more bioavailable (SACN, 2010).
- 4.54 Dietary determinants of iron status include adequate dietary iron intake, the form of iron ingested (haem or non-haem iron), and the presence of inhibitors and enhancers of iron absorption in meals (McCarthy et al, 2017).
- 4.55 SACN recommends that a public health approach to achieving adequate iron status should emphasise the importance of a healthy balanced diet that includes a variety of foods containing iron (SACN, 2010).
- 4.56 The current UK dietary reference values (DRVs) for iron for young children (Table 4.8) were set by the Committee on Medical Aspects of Food and Nutrition Policy (COMA) in 1991 and retained following a detailed review by SACN (2010). Intakes at or above the RNI will almost certainly meet the needs of 97.5% of the population while the LRNI represents a level below which intakes are almost certainly inadequate for most individuals. Intakes at the estimated average requirement (EAR) will meet the needs of approximately 50% of the population (SACN, 2010).

However, it should be noted that there are uncertainties in the iron DRVs (SACN, 2010).

**Table 4.8. DRVs for iron for children aged 1 to 6 years<sup>1</sup>**

Age	LRNI mg per day ( $\mu$ mol per day)	EAR mg per day ( $\mu$ mol per day)	RNI mg per day ( $\mu$ mol per day)
1 to 3 years	3.7 (65)	5.3 (95)	6.9 (120)
4 to 6 years	3.3 (60)	4.7 (80)	6.1 (110)

Abbreviations: DRV, dietary reference value; EAR, estimated average requirement; LRNI, Lower Reference Nutrient Intake; RNI, Reference Nutrient Intake

<sup>1</sup> Source: DH (1991) and SACN (2010).

## Iron intake in the UK

4.57 Iron intake data in children in the UK aged 12 to 60 months from DNSIYC and NDNS (years 2016 to 2019) are presented in Table 4.9.

**Table 4.9 Iron intake in children aged 12 to 60 months in the UK (DNSIYC and NDNS years 2016 to 2019)**

Age	Intake from diet and supplements Mean intake as % RNI	Intake from diet only Mean intake as % RNI	Intake from diet and supplements % below LRNI	Intake from diet only % below LRNI	Number of participants
12 to 18 months <sup>1</sup>	93	92	13	Data not available	1275
18 to 47 months <sup>2</sup>	88	84	11	11	306
48 to 60 months <sup>2</sup>	187	186	1	1	102

Abbreviations: LRNI, Lower Reference Nutrient Intake; RNI, Reference Nutrient Intake

<sup>1</sup> Data from DNSIYC 2011 (DH, 2013).

<sup>2</sup> Data from NDNS years 2016 to 2019

4.58 Data from both dietary surveys indicated that children in the 2 younger age groups may be most at risk of iron insufficiency: 13% of children aged 12 to 18 months and 11% of children aged 18 to 47 months had intakes below the LRNI.

4.59 While this raises concerns about the iron content and quality of solid foods (which should ensure adequate intake to replenish iron stores that are diminished during periods of exclusive breastfeeding), some caution should be taken when interpreting the data given concerns about the level of underreporting of TDEI in

the group of children with intakes below the LRNI (see paragraph 4.18), and uncertainties in the iron DRVs (SACN, 2011b).

- 4.60 Secondary analysis of NDNS data (years 2008 to 2019) was conducted to determine the characteristics of children (in 2 age groups: 18 to 47 months, and 48 to 60 months) with intakes below the LRNI for iron and those with intakes at or above the LRNI (Annex 11, Tables A11.17 to A11.20). Characteristics that were considered were age, sex, ethnicity and household socioeconomic status.
- 4.61 For children aged 18 to 47 months, girls made up a higher proportion of the children with intakes below the LRNI for iron (61%) compared with their proportion of the sample of children in this age group (49%). Asian or Asian British children made up 17% of the children with intakes below the LRNI, but only 8% of the whole sample. Children from households where the HRP had never worked (outside the home) made up 14% of the children with intakes below the LRNI, but only 6% of the whole sample. However, some caution should be taken when interpreting the findings because the total number of children with intakes below the LRNI was small (n=118).
- 4.62 At the same time, children from households where the HRP was in higher managerial and professional occupations were overrepresented in the group with intakes at or above the RNI (21% at or above the RNI versus 15% of the whole sample).
- 4.63 The number of children aged 48 to 60 months with intakes below the LRNI for iron was too small to enable a similar breakdown of characteristics in this group.
- 4.64 Time trend analysis of NDNS data (years 2008 to 2019) in children aged 18 to 36 months indicated a significant average annual reduction in daily iron intake (from food sources only) of  $-0.07\text{mg}$  (95% CI  $-0.11$  to  $-0.03\text{mg}$ ), equivalent to a reduction of  $0.8\text{mg}$  over the 11-year period (Bates et al, 2020). Over the same 11-year period, there was a significant increase in children with intakes (from food sources only) below the LRNI of 0.66 percentage points per year (95% CI 0.06 to 1.26 percentage points), equivalent to a reduction of 7 percentage points over the 11 years. This downward trend in iron intake raises concerns and potential implications of the movement towards the adoption of plant-based diets, which may have a lower bioavailable iron content than diets containing animal sources of iron.
- 4.65 No time trend data was available for the other age groups.

## **Iron intake and deprivation**

- 4.66 Iron intake by index of multiple deprivation (IMD) in children aged 18 to 60 months are presented in Table 4.10. The IMD is the official measure of relative deprivation in over 30,000 small areas or neighbourhoods in England (MHCLG, 2019). It

broadly defines deprivation to encompass a wide range of an individual's living conditions, including housing, education and training, and crime.

**Table 4.10 Iron intake (from diet only) by IMD quintile in children aged 18 to 60 months in England (NDNS years 2008 to 2019)<sup>1</sup>**

Iron mg/day	IMD quintile 1 (least deprived)	IMD quintile 2	IMD quintile 3	IMD quintile 4	IMD quintile 5 (most deprived)
Mean (95% CI)	6.5 (6.2 to 6.7)	6.3 (6.0 to 6.5)	6.5 (6.2 to 6.7)	6.5 (6.3 to 6.8)	6.3 (6.1 to 6.5)
Number of participants	210	211	182	234	277

Abbreviations: IMD, index of multiple deprivation

<sup>1</sup> Data from NDNS years 2008 to 2019.

4.67 The analysis did not indicate an obvious trend in intake across the IMD quintiles for children aged 18 to 60 months. However, another analysis of NDNS data (years 2012 to 2017) in children aged 18 to 36 months that used a narrower measure of household socioeconomic status (equivalised household income, see Glossary) indicated that every £10,000 increase in equivalised household income was associated with an average increase in iron intake (mg per day, from food sources only) of 0.16mg per day (95% CI 0.06 to 0.26mg per day) (Bates et al, 2019). The difference in findings between the IMD analysis and the analysis based on equivalised household income suggests that diet quality (at least with respect to iron intake) may be more closely linked with affordability of foods than other aspects of an individual's living environment.

## Dietary sources of iron

4.68 Dietary iron exists in 2 main forms: haem iron and non-haem iron (paragraph 4.53).

4.69 The main dietary contributors (including from dietary supplements) to mean iron intake in children in the UK with intakes below the LRNI for iron were compared with those in children with intakes above the LRNI. Detailed results of this analysis of NDNS data (years 2008 to 2019) are presented in Annex 11, Tables A11.21 and A11.22 in children aged 18 to 48 months. The contribution of these food groups to TDEI is also shown. For children aged 48 to 60 months, the number of children with intakes below the LRNI for iron was too small to be presented.

4.70 For children aged 18 to 47 months, the difference in the relative (% TDEI) and absolute (mg per day) contributions of food groups to iron intake between children with iron intakes at or above the LRNI compared with those with iron intakes below

the LRNI was most pronounced for breakfast cereals (Annex 11, Table A11.21). Breakfast cereals contributed 23.9% (1.59mg per day) to the iron intake of children at or above the LRNI compared with 17.4% (0.55mg per day) in children with an iron intake below the LRNI.

- 4.71 While children with iron intakes below the LRNI obtained a higher proportion of their iron intake from bread, and meat and meat products, their absolute intake of iron from these foods was lower than that in children with a mean iron intake at or above the LRNI. This may be accounted for by their lower TDEI, smaller body size or a greater tendency to underreport TDEI (see paragraph 4.18), or a combination of these factors.
- 4.72 For children aged 48 to 60 months, there were insufficient numbers of children to present results in those with intakes below the LRNI.

## **Systematic review evidence identified on iron and health outcomes**

### **Interventions to improve iron status**

- 4.73 Very few trials have been conducted that examine the effect of improving diets to improve iron status in children aged 1 to 5 years in HIC, including the UK.
- 4.74 Only 1 randomised controlled trial (RCT), included in 2 SRs identified for this report (Domellöf et al, 2013; Matsuyama et al, 2017), examined the effect of increasing meat intake on iron status in young children from a HIC. The RCT (in 225 participants, aged 12 to 20 months) reported that children in New Zealand without anaemia who were given a high red meat diet (approximately 56g per day containing 2.5mg iron) for 20 weeks had a greater change from baseline in mean serum ferritin concentration (adjusted for CRP) compared with the control group (whole cows' milk not fortified with iron) by the end of the intervention. There was no evidence of a difference in the change from baseline in haemoglobin concentration or body iron. Although red meat appeared to improve iron status, Matsuyama et al (2017) noted that the adherence rate in the group randomised to red meat was low, at only 3.4%. This was compared with nearly 90% adherence in the control group.
- 4.75 Observational evidence from Ireland suggests that cows' milk intake  $\geq 400$ ml per day in children aged 2 years is associated with an increased risk of low serum ferritin concentrations, after adjustment for daily iron intake (McCarthy et al, 2017). The mechanisms behind the effect of cows' milk on iron status are unclear. Possible explanations are its low iron content (approximately 0.5mg/l) and the presence of components that may inhibit iron absorption or cause occult intestinal

blood loss (McCarthy et al, 2017). Due to its influence on iron status, the European Society for Paediatric Gastroenterology, Hepatology and Nutrition Committee on Nutrition (ESPGHAN CoN) advises that consumption of cows' milk in young children should not exceed 500ml per day (Domellöf et al, 2014); while others suggest that this threshold may be too high and that dietary recommendations into the second year of life need to be re-examined (McCarthy et al, 2017).

## Iron fortification

- 4.76 Fortification of foods with iron (that is, the addition of iron to foods) has been the main approach used to improve the supply of iron in the UK diet (SACN, 2010). Iron has also been added to foods to replace iron lost during processing (restoration) and to ensure nutritional equivalence of products replacing common foods in the diet (for example, meat substitutes) (SACN, 2010).
- 4.77 In the UK, mandatory addition of iron to white and brown flour was introduced in 1953 as iron is lost during the processing of wheat flour, while many breakfast cereals are fortified on a voluntary basis (SACN, 2010).
- 4.78 The composition of infant formula and follow-on formula (see Glossary), including its iron content, is regulated in the UK ([Commission Delegated Regulation \(EU\) 2016/127](#), which was retained as UK law after the UK left the EU). For example, the iron content of infant formula made from cows' or goats' milk should be between 0.07 and 0.3mg per 100 KJ (0.3 to 1.3mg per 100 kcal).
- 4.79 For this report, 2 SRs with meta-analyses (MAs) (Athe et al, 2014; Matsuyama et al, 2017) and 1 SR without MA (Pratt, 2015) were identified that examined the effect of iron fortification on measures of iron status.
- 4.80 Most of the evidence was from trials that tested the effect of fortifying with iron together with other micronutrients (primarily zinc, vitamin A, vitamin C, vitamin D and folic acid). The most common food vehicles used for fortification were milk or formula, cereals, condiments and micronutrient powders (for example, Sprinkles). Interventions were mostly in the short term ( $\leq 12$  months) and conducted in upper-middle income (UMICs), lower-middle income (LMICs) or low-income countries (LICs).
- 4.81 Following the methodological approach outlined in paragraphs 4.22 and 4.23, the certainty of the evidence was graded if findings from the SRs were clearly stratified by the baseline nutritional status of participants. Evidence in participants with mixed or unknown or unreported nutritional status at baseline is described below but the certainty of this evidence was not graded.
- 4.82 Details of the SRs included in this section can be found in Annex 5, Table A5.1. Quality assessment of the SRs using the AMSTAR 2 tool can be found in Annex 7, Table A7.3. Additional data extracted on the primary studies can be found in Annex 8 (Table A8.12). The criteria used to grade the evidence are provided in

chapter 2 (Table 2.4, paragraphs 2.54 to 2.59). Summary tables of the evidence grading process for this section are provided in Annex 9 (Table A9.8, A9.9 and A10.36).

### **Haemoglobin concentration – fortification trials (iron and other micronutrients) in children with anaemia or high anaemia prevalence**

- 4.83 One SR without MA (Pratt, 2015) examined the effect of fortification with iron (and other micronutrients, mainly zinc, vitamin A, vitamin C and folic acid) on haemoglobin concentrations in children with anaemia or a high prevalence of anaemia from UMIC and LMIC. Anaemia was defined as a haemoglobin concentration less than 110g/l.
- 4.84 Pratt (2015) (AMSTAR 2 confidence rating: critically low) included 2 trials in children aged 1 to 5 years. Both studies performed per protocol (PP) analyses. One randomised trial (in 2666 participants, aged 36 months, 43 to 44% anaemia prevalence) reported that children who received solid foods fortified with 10mg iron (and zinc, vitamin A, vitamin C and folic acid) had an increased mean haemoglobin concentration after 4 months' intervention (quantitative findings not reported). However, as all comparison groups in this trial received iron (at different doses), there was effectively no control group. The other study, a cluster-RCT (in 2283 participants, aged 6 to 36 months, mean baseline haemoglobin concentration approximately 100g/l), reported that the mean haemoglobin concentration of children who received a micronutrient powder intervention (which included 12.5mg iron, as well as zinc, vitamin A, vitamin C and folic acid) increased by 7g/l (95% CI not reported) from baseline, while the mean haemoglobin concentration of children in the control group (no powder) decreased by 2g/l (95% CI not reported) ( $p < 0.001$  for the difference in change from baseline between groups). Analyses were adjusted for cluster effects.

### **Haemoglobin concentration – fortification trials (iron alone or with other micronutrients) in children (baseline status not reported)**

- 4.85 Two SRs with MAs (Athe et al, 2014; Matsuyama et al, 2017) examined the effect of fortification with iron (with or without other micronutrients, mainly zinc) on haemoglobin concentrations in children under age 5 years but did not report the baseline iron status of participants.
- 4.86 Matsuyama et al (2017) (AMSTAR 2 confidence rating: moderate) reported no difference in mean haemoglobin concentrations between children who received milk or formula fortified with iron (with or without zinc, vitamin D or vitamin C) and children who received non-fortified milk (MD 5.89g/l; 95% CI -0.25 to 12.02g/l;  $p = 0.06$ ;  $I^2$  not reported (NR); 8 RCTs, participants NR). However, the confidence

interval was wide, and the degree of heterogeneity was not reported. Intervention durations ranged from 5 to 12 months. Three of the eight RCTs were conducted in HIC, including the UK. According to the SR authors, potential bias from funding sources of the 8 RCTs was either unclear or low risk. No information was provided on the type of analysis (ITT or PP) carried out by the studies.

- 4.87 Athe et al (2014) (AMSTAR 2 confidence rating: low) reported a greater increase in mean haemoglobin concentration in children who received iron-fortified foods compared with the control group after a mean intervention duration of 6.5 months (Weighted mean difference [WMD] 5.09g/l; 95% CI 3.23 to 6.95g/l;  $p < 0.0001$ ;  $I^2 = 90\%$ ; random-effects model; 18 RCTs, 5142 participants). Participants had a mean age of 4.7 years and the trials were conducted mainly in LMICs. Foods that were fortified included milk, orange juice, cereal-based staple foods, water. No information was provided on the type of analysis (ITT or PP) carried out by the studies.

### **Serum ferritin – fortification trials (iron with other micronutrients) in children without anaemia**

- 4.88 One SR with MA (Matsuyama et al, 2017) examined the effect of fortification with iron (and other micronutrients, mainly zinc, and vitamins A and C) on serum ferritin concentrations in children without anaemia.
- 4.89 Matsuyama et al (2017) (AMSTAR 2 confidence rating: moderate) included 2 RCTs in children aged 1 to 5 years from HIC. Findings were not pooled into a MA due to limitations in the data. One RCT (in 125 participants, mean age 17 months) reported a greater increase in mean serum ferritin (adjusted for CRP concentration) in the group that received milk fortified with iron (and zinc and B vitamins) after 5 months of the intervention compared with the control group in ITT analyses (quantitative findings NR). The other smaller RCT (in 36 participants, mean age 12 months) reported no difference in change from baseline of serum ferritin after 6 months of the intervention between the iron-fortified milk and non-fortified milk groups in PP analyses (quantitative findings NR). All children had normal CRP concentrations at baseline and at the end of the intervention. However, the study may not have been adequately powered for serum ferritin concentration as an outcome as the power calculation was performed for other measures of iron status.

### **Serum ferritin – fortification trials (iron with other micronutrients) in children with anaemia or high anaemia prevalence**

- 4.90 Two SRs with MA (Matsuyama et al, 2017; Pratt, 2015) examined the effect of fortification with iron (and other micronutrients, mainly zinc and vitamin A) on serum ferritin concentrations in children with anaemia or a high prevalence of anaemia.

- 4.91 Matsuyama et al (2017) (AMSTAR 2 confidence rating: moderate) included 2 RCTs in children aged 1 to 5 years from UMIC. Intervention groups received milk or formula fortified with iron (and vitamin A and zinc) while the control groups received non-fortified milk or milk fortified with vitamin A only. Findings were not pooled into a MA due to limitations in the data. One RCT (in 115 participants, mean age 20 months, 41% and 30% anaemia prevalence in intervention and control groups, respectively) reported no difference in change from baseline for serum ferritin (unadjusted for CRP) between the intervention and control groups after 6 months of the intervention in PP analysis. The other, larger RCT (in 570 participants with anaemia, mean age 22 months) reported a greater increase in serum ferritin concentration (unclear whether adjusted for CRP) in the intervention group after 12 months of the intervention compared with the control group in ITT analysis. Quantitative findings were NR for either study. According to the SR, both studies had either a low or unclear risk of bias from their funding sources.
- 4.92 Pratt (2015) (AMSTAR 2 confidence rating: critically low) included 1 additional trial in children aged 1 to 5 years in UMIC. The randomised trial (in 2666 participants, aged 36 months, 43 to 44% anaemia prevalence) reported no change from baseline in serum ferritin (adjusted for CRP) in children who received complementary foods fortified with iron (and zinc, vitamin A, vitamin C and folic acid) after 4 months of the intervention in PP analyses (quantitative findings NR). However, as all comparison groups in this trial received iron (at different doses), there was effectively no control group.

### **Iron deficiency – fortification trials (iron with other micronutrients) in children with a high prevalence of anaemia**

- 4.93 One SR without MA (Pratt, 2015) (AMSTAR 2 confidence rating: critically low) included 1 cluster-RCT that examined the effectiveness of a public health programme in Mexico that distributed milk fortified with iron (plus zinc and vitamin A) to children aged 12 to 30 months. The baseline anaemia prevalence in this group of children was 43%. The cluster-RCT (in 795 participants) reported that the fortified milk group had a reduction in the estimated prevalence of ID (serum ferritin less than 12µg/l) from 30% at baseline to 18% and 6% after 6 and 12 months, respectively. The reduction was greater than the reduction in the control group (from 36% at baseline to 42% and 17% after 6 and 12 months, respectively; treatment effect:  $p=0.006$ ). The study performed a PP analysis and adjusted for cluster effects.

### **Anaemia – fortification trials (iron with other micronutrients) in children with anaemia or with a high prevalence of anaemia**

- 4.94 One SR without MA (Pratt, 2015) examined the effect of fortification with iron (and other micronutrients, mainly zinc, vitamin A and folic acid) on the risk of anaemia in

children with anaemia at baseline or with a high prevalence of anaemia. Anaemia was defined as haemoglobin concentrations <110g/l.

- 4.95 Pratt (2015) (AMSTAR 2 confidence rating: critically low) included 3 trials (2 cluster-RCTs, 1 RCT) in children aged 1 to 5 years from UMIC and LMIC. Two trials used fortified milk (2 trials) and 1 trial used micronutrient powders (Sprinkles).
- 4.96 All 3 trials reported a reduction in the prevalence of anaemia after 2 to 12 months' intervention using PP analyses (none performed ITT analyses).
- 4.97 One cluster-RCT (in 795 participants, aged 12 to 30 months) reported a larger reduction in the estimated prevalence of anaemia from baseline to 6 and 12 months of the intervention in children who received milk fortified with iron (as well as zinc and vitamin A) compared with the control group (intervention group: 45% at baseline to 13% and 4% at 6 and 12 months, respectively; control group: 43% at baseline to 20% and 9% at 6 and 12 months, respectively; treatment effect  $p=0.02$ ). Analyses were adjusted for cluster effects.
- 4.98 The second cluster-RCT (in 2283 participants, aged 6 to 36 months) reported that a micronutrient powder intervention (which included 12.5mg iron, as well as zinc, vitamin A, vitamin C and folic acid) reduced anaemia prevalence from 72% to 52% after 2 months of the intervention, while anaemia prevalence increased in the control group from 72% to 75% ( $p<0.001$  for the difference at follow up). Analyses were adjusted for cluster effects.
- 4.99 The RCT (in 115 participants, mean age 20 months) reported that children who received milk fortified with iron (as well as zinc and folic acid) had a reduction in anaemia prevalence from 41% at baseline to 12% after 6 months of the intervention ( $p<0.001$ ); there was no change from baseline in anaemia prevalence in the control group (30% at baseline, 24% at 6 months;  $p=0.40$ ). Treatment with fortified milk was inversely associated with being anaemic after the 6 month intervention ( $p<0.03$ ), adjusted for age, sex and baseline anaemia. It was not clear what the exact outcome measure (for example, relative risk [RR] or odds ratio [OR]) for this association was.

### **Anaemia – fortification trials (iron with micronutrients) in children (baseline status NR)**

- 4.100 One SR with MA (Matsuyama et al, 2017) examined the effect of iron-fortified milk or formula (with or without other micronutrients, mainly zinc, vitamin C and vitamin D) on the risk of anaemia in children for which the baseline iron status was not reported.
- 4.101 Matsuyama et al (2017) (AMSTAR 2 confidence rating: moderate) reported in a subgroup analysis that iron fortification of milk or formula did not reduce the odds of anaemia in children aged 1 to 5 years compared with non-fortified milk (OR 0.46; 95% CI 0.19 to 1.12;  $I^2$  NR;  $p$ -value NR; 6 RCTs, participants NR). According

to the SR, the risk of publication bias for this outcome was minimal (from funnel plot symmetry). The SR did not provide any information on the type of analysis (ITT or PP) carried out by the studies.

## Summary: iron fortification and iron status

- 4.102 The evidence identified from SRs on the effect of iron fortification (with or without other micronutrients) on iron status is summarised in Table 4.11.

**Table 4.11 Summary of the evidence on the effect of iron fortification (with or without other micronutrients) on iron status**

Outcome (population subgroup)	Direction of effect <sup>1</sup>	Certainty of evidence
Hb concentration (in children without anaemia)	Not applicable	No SR evidence identified
Hb concentration (in children with anaemia or high anaemia prevalence)	Not applicable	Insufficient
Serum ferritin (in children without anaemia)	Not applicable	Insufficient
Serum ferritin (in children with anaemia or high anaemia prevalence)	Not applicable	Insufficient
Prevalence of ID (in children with anaemia or high anaemia prevalence)	Not applicable	Insufficient
Anaemia prevalence (in children without anaemia)	Not applicable	No SR evidence identified
Anaemia prevalence (in children with anaemia or high anaemia prevalence)	↓	Limited

Abbreviations: Hb, haemoglobin; ID, iron deficiency; SR, systematic review.

Definitions: ID (serum ferritin <12µg/l); anaemia (haemoglobin <110g/l).

<sup>1</sup> Direction of effect for reported outcomes: ↓decrease.

- 4.103 The available evidence from SRs on iron fortification (with or without other micronutrients) in children aged 1 to 5 years and iron status comes from 2 SRs (with MAs), 1 given a moderate confidence rating using the AMSTAR 2 tool, another given a low confidence rating, and 1 SR without MA given a critically low confidence rating.
- 4.104 Evidence from 3 trials included in the SR by Pratt (2015) suggests that fortification with iron and other micronutrients (including zinc, vitamin A and vitamin C) of milk, or micronutrient sprinkles reduces the prevalence of anaemia in children aged 6 to 36 months in LMIC and UMIC. The evidence was graded 'limited' because all trials performed per protocol analyses (which could overestimate effect sizes), the lack

of assessment by the SR of publication bias or potential bias from funding sources, the indirectness of the interventions (none of the trials examined iron fortification only), and unclear generalisability of findings to children living in the UK where the prevalence of iron deficiency anaemia is low (see Annex 10, Table A10.8 for details for the grading process).

- 4.105 There was 'insufficient' evidence from SRs to enable conclusions to be drawn on any effect of iron fortification on serum ferritin in children aged 1 to 5 years with anaemia or high prevalence of anaemia in UMIC from the 2 SRs by Matsuyama et al (2017) and Pratt (2015). The evidence from the 3 trials included in the 2 SRs was downgraded due to the lack of a control group in 1 trial, lack of information on study power, lack of assessment of publication bias, lack of or unclear adjustment of outcome measurements for inflammation, the indirectness of the interventions (none of the trials examined iron fortification only), and unclear generalisability of findings to children living in the UK where the prevalence of iron deficiency anaemia is low (see Annex 10, Table A10.7 for details for the grading process).
- 4.106 There was 'insufficient' evidence from SRs for all other outcomes (Table 4.11) as fewer than 3 primary studies included in the SRs examined these relationships.

## **Iron supplementation**

- 4.107 Much of the research examining interventions to prevent or reverse IDA in children aged under 5 years comes from supplementation trials conducted in LICs or LMICs where poverty, malnutrition (including multiple micronutrient deficiencies), infectious disease (such as malaria) and inflammation can complicate the interpretation of findings and limit generalisability to children based in the UK. For example, findings from the NDNS indicated that inflammation in children under 5 years is not at levels high enough to affect iron status measures (see [Prevalence of ID and IDA in the UK](#)).
- 4.108 In high income settings, including the UK, where mild iron deficiency is relatively common but IDA is rare, universal iron supplementation is not generally recommended because of the cost of such a programme, risk of accidental iron overdose (Szymlek-Gay et al, 2009), poor absorption and utilisation of other micronutrients (zinc and copper, for details see [SACN report 'Iron and Health'](#)), and the possible adverse side effects on growth in young children who do not have anaemia (see [Iron status and growth](#)).
- 4.109 Nonetheless, supplementation trials conducted in lower income countries have been useful in elucidating iron metabolism, deficiency and associated health outcomes.
- 4.110 Two SRs with MAs (De-Regil et al, 2011; Thompson et al, 2013) identified for this report showed that iron supplementation (daily or intermittent) is effective in improving haemoglobin and ferritin concentrations in children aged under 5 years

with baseline IDA, but has almost no effect in children who are iron replete (see Annex 8, Table A8.13 for details).

## **Iron and interactions with other micronutrients or food components**

- 4.111 Micronutrient intake is only one of the factors that impacts nutrient status. The absorption and excretion of nutrients is regulated by the body to match the availability of nutrients to the body's needs (SACN, 2010). To increase body content of a specific nutrient, it is therefore important to understand the factors that regulate its absorption and excretion, including interactions with other nutrients.
- 4.112 High iron intake may interfere with the metabolism of other similar metals, such as zinc and copper (SACN, 2010); iron supplementation of iron replete children may competitively inhibit intestinal absorption of these nutrients, potentially leading to deficiencies (Domellöf et al, 2013).
- 4.113 One SR with MA (Domellöf et al, 2013) identified for this report examined the effects of interactions between iron and other micronutrients or food components on iron status.
- 4.114 Domellöf et al (2013) examined whether tea consumption had any impact on iron status. This is because phenolic compounds found in tea (and coffee) bind iron and restrict its availability for absorption (SACN, 2010). Domellöf et al (2013) reported that in groups with high prevalence of ID (including infants and young children), tea consumption was inversely associated with serum ferritin and haemoglobin (quantitative data were not reported). However, the association disappeared after adjusting for confounding dietary factors. The SR concluded that tea consumption did not influence iron status in populations with adequate iron stores and that there was no need to advise any restrictions on tea drinking in healthy individuals with no risk of ID. However, in groups at risk of ID, the SR advised that drinking tea should be done between meals (at least 1 hour after eating).

## Iron and health

- 4.115 The main public health concerns associated with ID and IDA in childhood are the risk of delayed or abnormal neurological development, growth failure and impaired immune response (Domellöf et al, 2014). These health outcomes are considered below.

### Iron status and neurodevelopment

- 4.116 Evidence from observational studies indicates that ID and IDA are associated with many psychosocial, economic and biomedical disadvantages, which can independently affect development (SACN, 2010). Although deficits in neurological development are not solely attributable to ID and IDA, there may be a reduced risk at haemoglobin concentrations above 100 to 110g/l, the WHO (2001b) cut-off for IDA (SACN, 2010).

### ID without anaemia and neurodevelopment

- 4.117 The brain becomes iron deficient before the onset of anaemia, due to prioritisation of available iron to red blood cells over the brain and other organs (Cusick et al, 2018; Georgieff, 2017). Therefore, it is not appropriate to rely on identifying and preventing anaemia as a strategy to protect the developing brain (Georgieff, 2017) as there is growing evidence that ID without anaemia may be responsible for developmental deficits (Cusick et al, 2018; Eussen et al, 2015; Georgieff, 2017; Pasricha et al, 2013; Thompson et al, 2013).
- 4.118 However, the currently available haematological indices are not sensitive biomarkers of brain iron deficiency and dysfunction (Cusick et al, 2018). Current efforts are focussed on developing screening tools that are specific to iron-dependent brain health as opposed to red blood cell indicators (Georgieff, 2017).
- 4.119 Double-blinded RCTs of iron supplementation designed to prevent ID would offer the best opportunity to determine the role of iron in neurological development. However, there are few adequately powered, double-blinded RCTs examining this causal relationship (Pasricha et al, 2013), and a lack of dose response studies linking indicators of iron status as continuous risk factors with later cognitive outcomes (Domellöf et al, 2014).

### Evidence from supplementation trials

- 4.120 For this report, 1 SR with MA of supplementation trials was identified that examined the effect of iron on neurodevelopment and cognitive outcomes in children aged under 5 years with ID from mostly MIC (Pasricha et al, 2013). Following the methodological approach outlined in paragraphs 4.22 and 4.23, the evidence is described below but was not graded.

4.121 Pasricha et al (2013) (AMSTAR 2 confidence rating: high) reported that children aged 4 to 23 months with ID supplemented with iron (for 3 to 6 months) had improved cognitive development (measured by Bayley's mental development index) compared with the control group (Mean difference [MD] in score 5.90; 95% CI 1.81 to 10.00;  $p=0.005$ ;  $I^2=34\%$ ; random-effects model; 3 RCTs, 281 participants). However, Pasricha et al (2013) noted that the finding was driven by 1 RCT that was at high risk of bias while the other RCTs included in the MA may have been underpowered to find an effect. In addition, the RCTs included in this MA used the Bayley Mental Development Index and the Psychomotor Development Index to measure outcomes that may not be sensitive to small changes in cognitive development. Whether any benefit of iron supplementation in the shorter term is sustained is unclear.

### **IDA and neurological development**

4.122 There is an extensive body of research that considers the relationship between IDA and cognitive, motor and behavioural development in children. While most researchers conclude that IDA causes poor cognition in school-aged children, the effect on younger children remains controversial (SACN, 2010). RCTs to treat IDA are less likely to provide evidence of an effect of iron on neurological outcomes, which, depending on the age-group, co-morbidities (including infections) and duration of the IDA, may contribute to irreversible neurological deficits during early development.

### **Evidence from supplementation trials**

- 4.123 The SACN report on 'Iron and Health' (SACN, 2010) concluded that there was no clear evidence that iron treatment in the short term (less than 2 weeks) benefited psychomotor and mental development in children aged 3 years or under with anaemia. SACN stated that findings from longer-term trials (3 to 12 months) were difficult to interpret given that not all were randomised. However, there was some evidence of benefit of longer-term iron supplementation to motor development in children aged 3 years or under (SACN, 2010).
- 4.124 For this report, 1 SR with MA (Pasricha et al, 2013) was identified that examined the effect of iron supplementation on cognitive outcomes in children aged under 5 years with anaemia (not defined) from mostly MIC.
- 4.125 Pasricha et al (2013) (AMSTAR 2 confidence rating: high) reported no difference in effect of iron supplementation (for 3 to 6 months) on cognitive development (MD in score 4.46; 95% CI  $-9.32$  to  $18.24$ ;  $p=0.53$ ;  $I^2=80\%$ ; random-effects model; 3 RCTs, 113 participants) or psychomotor development (MD in score 4.20; 95% CI  $-9.88$  to  $18.29$ ;  $p=0.56$ ;  $I^2=78\%$ ; random-effects model; 3 RCTs, 113 participants) in anaemic children aged 4 to 23 months compared with the control group. However, it was unclear what the causes of anaemia in these children were (ID or

other causes), and the wide confidence intervals around treatment effects indicate that the MA may have lacked statistical power to detect small treatment effects.

## **Iron status and growth**

- 4.126 While iron is crucial to adequate growth during infancy (SACN, 2018), evidence from RCTs suggests that iron supplementation may have detrimental effects on the growth of infants and children who do not have ID or IDA (haemoglobin >110g/l and serum ferritin >12µg/l in most studies) (SACN, 2010).

### **Evidence from supplementation trials**

- 4.127 For this report, 1 SR with MA (Thompson et al, 2013) was identified that examined the effect of iron supplementation on linear growth and weight gain in children aged 2 to 5 years from mostly LMIC. Findings from this SR were not stratified by baseline iron status. Following the methodological approach outlined in paragraphs 4.22 and 4.23, the evidence was not graded.
- 4.128 Thompson et al (2013) (AMSTAR 2 confidence rating: moderate) reported no difference in effect on either linear growth or weight gain between children who received iron supplementation for up to 12 months and the control group (see Annex 8, Table A8.14 for details).

## **Iron status and immune function**

- 4.129 Iron has many important functions in the immune system. It has been suggested that iron deficiency could impair secretion of cytokines and reduce bactericidal macrophage activity and T-cell proliferation (Domellöf et al, 2014), and therefore increase susceptibility to infectious pathogens.
- 4.130 However, while iron is required for an individual's immune response, it is also required by pathogens for growth and replication. Supplemental iron may therefore favour infectious pathogens by providing them with a supply of iron which is required for their growth and replication (SACN, 2010).

### **Evidence from supplementation trials**

- 4.131 Two SRs with MAs (Pasricha et al, 2013)(Pasricha et al, 2013; Thompson et al, 2013) were identified that examined the effect of iron supplementation on infection. Most of the evidence included in these SRs were from trials conducted in LMICs where the co-existence of multiple nutrient deficiencies may affect resistance to infection (SACN, 2010). Malaria, which may be prevalent in some of these countries, also reduces haemoglobin concentrations independently of iron and other nutritional deficiencies (SACN, 2010). Following the methodological approach outlined in paragraphs 4.22 and 4.23, the evidence is described below but was not graded.

- 4.132 Pasricha et al (2013) (AMSTAR 2 confidence rating: high) reported that children aged 4 to 23 months who were supplemented with iron had an increased risk of vomiting (RR 1.38; 95% CI 1.10 to 1.73;  $I^2=1\%$ ;  $p=0.006$ ; 3 RCTs, 1020 participants). However, as the finding was not stratified by baseline iron status, it is unclear whether the magnitude of the risk differs in children with adequate versus low iron status.
- 4.133 Evidence on the effect of iron supplementation on fever was equivocal. Pasricha et al (2013) reported that iron supplementation increased the prevalence of fever (RR 1.16; 95% CI 1.02 to 1.31;  $p=0.02$ ;  $I^2=0$ ; random-effects model; 4 RCTs, 1318 participants); while 1 out of 3 trials included in Thompson et al (2013) that examined this outcome (but not included in a MA) reported that iron supplementation may increase the frequency of fever episodes (quantitative findings NR).
- 4.134 Both SRs reported that iron supplementation has no effect on diarrhoeal episodes or prevalence, or incidence or prevalence of lower respiratory tract infections in children aged up to 5 years (see Annex 8, Table A8.16 for details).

# Zinc

- 4.135 Zinc is present within every cell in the body and has a wide range of physiological functions, including a structural or catalytic role in all 6 classes of enzyme, regulation of gene expression and intracellular signalling.

## Current recommendations for zinc intake in the UK

- 4.136 The current UK DRVs for zinc (Table 4.12) were set by COMA in 1991 (DH, 1991).

**Table 4.12 Dietary reference values for zinc for children aged 1 to 6 years<sup>1</sup>**

Age	LRNI mg per day ( $\mu$ mol per day)	EAR mg per day ( $\mu$ mol per day)	RNI mg per day ( $\mu$ mol per day)
1 to 3 years	3.0 (45)	3.8 (60)	5.0 (75)
4 to 6 years	4.0 (60)	5.0 (75)	6.5 (100)

Abbreviations: EAR, Estimated Average Requirement; LRNI, Lower Reference Nutrient Intake; RNI, Reference Nutrient Intake

<sup>1</sup> Source: (DH, 1991).

## Zinc intake in the UK

- 4.137 Intake data in children in the UK aged 12 to 60 months from DNSIYC and NDNS years 2016 to 2019 are presented in Table 4.13.

**Table 4.13 Zinc intake in children aged 12 to 60 months in the UK (DNSIYC and NDNS years 2016 to 2019)**

Age	Intake from diet and supplements Mean intake as % of RNI	Intake from diet only Mean intake as % of RNI	Intake from diet and supplements % participants below LRNI	Intake from diet only % participants below LRNI	Number of participants
12 to 18 months <sup>1</sup>	200	200	4	4	1275
18 to 47 months <sup>2</sup>	101	96	8	8	306
48 to 60 months <sup>2</sup>	84	83	20	21	102

Abbreviations: LRNI, Lower Reference Nutrient Intake; RNI, Reference Nutrient Intake.

<sup>1</sup> Data from DNSIYC 2011 (DH, 2013).

<sup>2</sup> Data from NDNS years 2016 to 2019.

- 4.138 Older children were at higher risk of having zinc intakes below the LRNI. While 4 and 8% of children aged 12 to 18 months, and 18 to 47 months respectively, had zinc intakes from food sources below the LRNI, this increased to 21% of children aged 48 to 60 months. This trend may be due to the increase in the RNI (and LRNI) at 4 years and the decrease in milk consumption from 18 months and upwards (see chapter 3, Table 3.5). However, some caution should be taken when interpreting the data given concerns about the level of underreporting of intakes in the group of children with intakes below the LRNI (see paragraph 4.18).
- 4.139 Secondary analysis of the data from NDNS (years 2008 to 2019) was conducted to determine the characteristics of children (in 2 age groups: 18 to 47 months, and 48 to 60 months) with intakes below the LRNI for zinc and those with intakes at or above the LRNI (see Annex 11, Tables A11.17 to A11.20). Characteristics that were considered were age, sex, ethnicity and household socioeconomic status.
- 4.140 For both age groups, girls made up a higher proportion of the children with intakes below the LRNI (56% and 67%, respectively) compared with their proportion of the sample of children in this age group (49% and 53%, respectively). For children aged 18 to 47 months, Black or Black British children made up 8% of the children with intakes below the LRNI, but only 4% of the whole sample. For both age groups, children from households where the HRP had never worked (outside the home) or were in semi-routine occupations made up 15% of the children with intakes below the LRNI, but only 6% of the whole sample. However, some caution should be taken when interpreting the findings because the numbers of children with intakes below the LRNI for each age group was small (fewer than 90).
- 4.141 Time trend analysis of NDNS data (years 2008 to 2017) in children aged 18 to 36 months indicated a significant average annual reduction in daily zinc intake (from food sources only) of  $-0.05$  mg (95% CI  $-0.10$  to  $-0.01$ mg) for the 9-year period. For the same 9-year period, there was no significant change in the percentage of children with intakes (from food sources only) below the LRNI (0.4 percentage point change per year; 95% CI  $-0.3$  to 1.1 percentage points) (Bates et al, 2019). No time trend data was available for the other age groups.

## **Zinc intake and deprivation**

- 4.142 Zinc intake by IMD (see Glossary) in children aged 18 to 60 months is presented in Table 4.14. Mean zinc intake were lowest in quintile 4 and 5 (most deprived) (5.0mg per day) and highest in quintile 3 (5.4mg per day).

**Table 4.14 Zinc intake (from diet only) by IMD quintile in children aged 18 to 60 months in England (NDNS years 2008 to 2019)<sup>1</sup>**

Zinc intake mg/day	IMD quintile 1 (least deprived)	IMD quintile 2	IMD quintile 3	IMD quintile 4	IMD quintile 5 (most deprived)
Mean (95%CI)	5.2 (5.0 to 5.4)	5.2 (5.0 to 5.3)	5.3 (5.1 to 5.5)	5.0 (4.9 to 5.2)	5.0 (4.9 to 5.2)
Number of participants	210	211	182	234	277

Abbreviations: IMD, index of multiple deprivation

<sup>1</sup> Data from NDNS years 2008 to 2019.

- 4.143 There appears to be no clear relationship linking zinc intake with IMD, a broad indicator of deprivation, for children aged 18 to 60 months. However, another analysis of NDNS data (years 2012 to 2017) in children aged 18 to 36 months that used a narrower measure of household socioeconomic status (equivalised household income, see Glossary) suggested that every £10,000 increase in equivalised household income was associated with an average increase in zinc intake (from food sources only) of 0.09 mg per day (95% CI 0.01 to 0.18 mg per day) (Bates et al, 2019). The difference in findings between the IMD analysis and the analysis based on household income suggests that diet quality (at least with respect to iron intake) may be more closely linked with affordability of foods than other aspects of an individual's living environment.

## Dietary sources of zinc

- 4.144 Meat, legumes, eggs, fish, and grains and grain-based products are rich dietary zinc sources (EFSA, 2014). Due to the presence of dietary inhibitors of zinc absorption (for example, fibre and phytates) in some plant foods, zinc requirements for dietary intake may need to be adjusted upwards for populations in which animal products, the best sources of zinc, are limited or for those consuming plant-based diets (Ezzati et al, 2004). However, data on the effect of phytates on zinc absorption in children are limited (Krebs et al, 2014).
- 4.145 The main dietary contributors (including from supplements) to zinc intake in children in the UK with intakes below the LRNI for zinc were compared with those in children with intakes above the LRNI. Detailed results of this analysis of NDNS data (years 2008 to 2019) are presented in Annex 11, Tables A11.23 to A11.26 in children aged 18 to 47 months, and ages 48 to 60 months. The contribution of these food groups to TDEI is also shown.

- 4.146 The main dietary contributors to zinc intake were broadly similar across the age groups (milk, meat, bread and pizza being the principal sources). However, it is notable that children aged 18 to 47 months with intakes below the LRNI obtained a higher percentage of their zinc intake from meat and meat products (28.5%) than children with intakes at or above the LRNI (19.5%) (Annex 11, Table A11.23). On the other hand, children in this age group with intakes below the LRNI obtained a lower percentage of their zinc intake from milk and cream (16.2%) compared with children with intakes at or above the LRNI (22.2%).
- 4.147 As with iron and vitamin A, young children who avoid meat, milk or other dairy products due to restrictive diets or intolerance may be at increased risk of inadequate zinc intake.

## Assessment of zinc status

- 4.148 Zinc deficiency is largely related to inadequate intake or absorption of zinc from the diet although excess losses of zinc during diarrhoea may also contribute (Ezzati et al, 2004).
- 4.149 Identification of mild-to-moderate zinc deficiency remains a challenge due to the lack of sensitive and specific biomarkers. At a population level, the WHO has proposed 3 indicators to identify increased risk of deficiency: prevalence of inadequate dietary zinc intake, stunting, low serum or plasma zinc concentrations (Krebs et al, 2014).
- 4.150 Blood (serum or plasma) zinc concentration is affected by both inadequate and excess intake. Blood zinc concentration responds to an increase in intake over short periods. However, homeostatic mechanisms that act to maintain plasma zinc concentration within the physiological range may prevent high plasma concentrations from being sustained over a prolonged period (EFSA, 2014). Evidence from a large SR with MA suggests that zinc supplementation for more than 6 months in children aged under 5 years was less effective at increasing plasma or serum zinc concentrations than supplementing for less than 6 months (Mayo-Wilson et al, 2014).
- 4.151 Blood zinc concentrations are reduced in severe zinc deficiency (acquired or inherited) but as a biomarker of severe zinc deficiency, lacks sensitivity. At the same time, blood zinc concentrations lack specificity in moderate zinc deficiency (EFSA, 2014). Nevertheless, blood zinc concentration has been recommended as a biomarker of zinc status and of the population's risk of zinc deficiency by the WHO and UNICEF, among other health bodies (EFSA, 2014).
- 4.152 In the UK, blood zinc concentrations are not available from NDNS because the blood volumes collected in young children could not accommodate analysis of all biomarkers (it was also not measured in DNSIYC). However, intake data from NDNS indicated that mean zinc intake as a percentage of RNI decreased with

increase age (Table 4.13). This likely reflects the increase in the RNI for children aged 4 to 6 years (Table 4.12).

## **Systematic review evidence identified on zinc and health outcomes**

### **Interventions to improve zinc status**

#### **Zinc supplementation**

- 4.153 Trials conducted in LICs and LMICs have demonstrated the efficacy of zinc supplementation in improving the zinc status of young children. One large SR with MA of trials (Mayo-Wilson et al, 2014) reported that zinc supplementation increased serum or plasma zinc concentrations and lowered the risk of zinc deficiency in children aged under 5 years compared with no zinc supplementation (RR 0.41; 95% CI 0.37 to 0.47; p-value NR;  $I^2=90.6%$ ; 10 RCTs, 3761 participants).

#### **Zinc and interactions with other micronutrients**

- 4.154 Just as high iron intake may interfere with the metabolism of other similar metals (see Iron and interactions with other micronutrients or food components), adverse effects of zinc supplementation on iron status have also been observed (Sandström, 2001). However, findings from a large SR with MA (Mayo-Wilson et al, 2014) indicate that zinc supplementation does not have an important effect on iron status measures, including haemoglobin and serum or plasma ferritin, or the prevalence of ID or anaemia.
- 4.155 When considering the interaction of similar metals on zinc status, Mayo-Wilson et al (2014) reported that supplementing with zinc together with iron may be less effective at improving serum or plasma zinc concentrations and reducing the risk of zinc deficiency than supplementing with zinc alone in children under 5 years. Co-supplementing with iron may also reduce the effectiveness of zinc on linear growth compared with supplementing with zinc alone (see Annex 8, Table A8.17 for detailed results).

## Zinc and health

- 4.157 There is a lack of specific health effects of zinc deficiency due to its critical role in many core biochemical processes (EFSA, 2014). In its severest form, zinc deficiency can affect numerous organ systems, including gastrointestinal, skeletal, reproductive and central nervous systems (Mayo-Wilson et al, 2014); while mild-to-moderate zinc deficiency is characterised by growth impairment and altered immune function (Krebs et al, 2014).

### Low zinc status and growth

- 4.158 Young children are especially vulnerable to zinc deficiency given that periods of rapid growth increase zinc requirements that may be unmet (Mayo-Wilson et al, 2014).
- 4.159 As with iron, most of the evidence on the impact of low zinc status on growth in young children is informed by supplementation and fortification trials conducted in developing countries. However, recurrent infections such as diarrhoea, chronic inflammation and other micronutrient deficiencies which are associated with poverty can also adversely affect linear growth (Krebs et al, 2014) and can therefore complicate interpretation of findings from studies examining the relationship between zinc status and growth.
- 4.160 For this report, 1 SR with MA (Mayo-Wilson et al, 2014) was identified that examined the effect of zinc supplementation on linear growth and body weight in children aged under 5 years from mostly LMIC. Following the methodological approach outlined in paragraphs 4.22 and 4.23, the evidence is described below but the certainty of the evidence was not graded.

### Linear growth

- 4.161 Mayo-Wilson et al (2014) (AMSTAR 2 confidence rating: moderate) reported that children aged 1 to 5 years who were supplemented with zinc experienced greater linear growth than the control group (SMD  $-0.09$ ; 95% CI  $-0.14$  to  $-0.04$ ;  $I^2=42\%$ ; fixed-effects model; 27 estimates from 24 RCTs, 6155 participants; note that for this MA, a negative SMD favours zinc supplementation). However, as findings were not stratified by the baseline zinc status of participants, it is unclear whether baseline nutritional adequacy or deficiency modifies the effect of zinc supplementation on linear growth.

### Body weight

- 4.162 Mayo-Wilson et al (2014) reported that children aged 1 to 5 years who were supplemented with zinc gained more weight than the control group (SMD  $-0.06$ ; 95% CI  $-0.11$  to  $-0.01$ ;  $I^2=43\%$ ; fixed-effects model; 23 estimates from 20 RCTs, 5565 participants; note that for this MA, a negative SMD favours the intervention)

but an asymmetrical funnel plot suggested potential bias from small study effects or reporting bias. Meanwhile, zinc supplementation had no effect on the weight-to-height ratio in children aged 1 to 5 years (SMD  $-0.02$ ; 95% CI  $-0.08$  to  $0.05$ ;  $I^2=6.8\%$ ; fixed-effects model; 14 estimates from 12 RCTs, 4302 participants).

## Salt (sodium)

- 4.163 The main evidence for the association between high salt intakes and blood pressure (in adults) relates to sodium. The main dietary source of sodium is salt. This section therefore focuses on salt intake in the UK.

### Current recommendations for salt intake in the UK

- 4.164 The current DRVs for salt intake in the UK were set by COMA in 1991 (DH, 1991). In its report 'Salt and Health', SACN accepted the RNI values for sodium for infants and children that were set by COMA and used these as a basis to estimate target average salt intakes (Table 4.15). The target average salt intake does not represent an optimal or ideal consumption level for children but an achievable population goal. SACN concluded that attainment of these levels would require a substantial reduction in current levels of intake (SACN, 2003).

**Table 4.15 Reference nutrient intakes (RNI) for sodium and target average salt intakes for young children (SACN, 2003).**

Age	Sodium RNI mmol per day (mg per day)	Salt (grams per day)	Target average salt intake (grams per day)
1 to 3 years	22 (500)	1.2	2
4 to 6 years	30 (700)	1.8	3

### Salt intake in the UK

- 4.165 Salt intake in children aged 12 to 60 months in the UK from DNSIYC and NDNS (years 2016 to 2019) is presented in Table 4.16. Mean salt intake was above the target average salt intake in children aged 18 to 47 months (2.7 grams per day), where 76% of children in this age group had salt intakes above the target average salt intake. In the oldest age group, 47% of children had salt intakes above the target average salt intake.

**Table 4.16 Salt intake in children aged 12 to 60 months in the UK (DNSIYC and NDNS 2016 to 2019)<sup>1</sup>**

Age	Grams per day <sup>2</sup>	% participants above target salt intake	Number of participants
12 to 18 months	2.3 (0.9)	Data not available	1275
18 to 47 months	2.7 (0.9)	76	306
48 to 60 months	3.2 (1.0)	47	102

<sup>1</sup> Data in children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013), otherwise data from NDNS 2016 to 2019 (Bates et al, 2020).

<sup>2</sup> Mean (SD). Salt intake from food sources. Excludes discretionary salt.

## Main dietary sources of salt

- 4.166 Sodium is present in plant and animal derived foods as well as drinking water. As salt, it is added to foods during processing, cooking and at the table (SACN, 2003).
- 4.167 The main dietary sources of salt (excluding discretionary salt) in children aged 12 to 60 months in the UK are presented in Table 4.17. Meat, meat products and dishes, followed by bread, were the largest contributors to salt intake in all age groups. Milk also made a substantial contribution (>10%) in the two younger age groups, while in the oldest age group, biscuits, buns, cakes, pastries, fruit pies and puddings were key contributors.

**Table 4.17 Contribution of food groups to average daily salt intake<sup>1</sup> in children aged 12 to 60 months (DNSIYC and NDNS years 2016 to 2019)<sup>2</sup>. Non-consumers are included in the average.**

Contribution of food groups <sup>3,4,5</sup> to total salt intake	12 to 18 months %	12 to 18 months g per day	18 to 47 months %	18 to 47 months g per day	48 to 60 months %	48 to 60 months g per day
Meat, meat products and dishes	14.7	0.37	19.2	0.55	24.3	0.81
Bread	14.1	0.33	14.6	0.41	14.7	0.48
Milk <sup>6</sup>	13.6	0.30	11.2	0.28	7.7	0.24
Vegetables, vegetable products and dishes	6.5	0.15	4.5	0.11	4.6	0.15
Pizza, pasta, rice, products and dishes	5.9	0.15	6.9	0.20	7.2	0.24
Biscuits, buns, cakes, pastries, fruit pies, puddings	5.8	0.13	7.8	0.20	8.4	0.25
Formula milks <sup>7</sup>	4.9	0.08	0.7	0.01	0	0.00
Breakfast cereals	3.6	0.08	3.6	0.09	3.1	0.10
Commercially manufactured foods and drinks specifically marketed for infants and young children	3.2	0.05	0.3	0.01	0.3	0.00
Yoghurt fromage frais and dairy desserts <sup>6</sup>	3.0	0.06	2.2	0.05	1.9	0.05
Savoury sauces pickles gravies and condiments	2.7	0.06	3.0	0.08	3.5	0.12
Soup	2.3	0.06	1.6	0.05	1.6	0.06
Crisps and savoury snacks	2.0	0.05	4.2	0.12	3.7	0.11
Low calorie soft drinks <sup>8</sup>	1.0	0.03	1.7	0.05	2.5	0.08
Fruit	0.6	0.01	0.6	0.01	0.4	0.01
Sugar preserves and confectionery	0.4	0.01	0.7	0.02	1.3	0.04
Ice cream <sup>6</sup>	0.2	0.00	0.4	0.01	0.6	0.02
Sugar-sweetened beverages <sup>9</sup>	0.1	0.00	0.1	0.00	0.1	0.00
Fruit juice and smoothies	0.1	0.00	0.1	0.00	0.1	0.00
Beverages dry weight	0.0	0.00	0.2	0.01	0.2	0.01
Number of participants	1275	1275	306	306	102	102

<sup>1</sup> Salt intake from food sources. Excludes discretionary salt.

<sup>2</sup> Data in children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013) otherwise data from NDNS years 2016 to 2019 (Bates et al, 2020).

<sup>3</sup> Food groups are ordered by largest to smallest % contribution in the youngest age group.

<sup>4</sup> Food groups that contribute less than 0.5% of intake in all age groups are not presented.

<sup>5</sup> Average % contribution for each food group has been calculated from the % contribution for each individual. Non-consumers are included in the average.

<sup>6</sup> Includes non-dairy alternatives

<sup>7</sup> Formula milks include milks marketed to children over the age of 1 year, also known as 'toddler milks' and 'growing-up milks' (see Glossary).

<sup>8</sup> Includes low calorie, diet, no added sugar, sugar-free drinks. Excludes mineral water.

<sup>9</sup> Includes carbonated drinks, concentrates and ready to drink products with added sugars.

## Salt and health

- 4.168 Blood pressure in childhood is strongly predictive of blood pressure in later life (Bao et al, 1995). Hypertension is one of the most important modifiable risk factors for cardiovascular, cerebrovascular and renal disease (WHO, 2017). The global prevalence of hypertension in children (aged 19 years and under) is estimated to be around 4%, with a higher prevalence in children with obesity (between 7% and 25%) and overweight (between 2% and 9%) compared with children with healthy weight (Song et al, 2019).
- 4.169 In its 2003 report 'Salt and Health', SACN found evidence that exposure to increased dietary sodium in early life may programme the development of higher blood pressure later in life (SACN, 2003). The Committee however concluded that there was insufficient evidence to be precise about upper limits for salt intake in relation to cardiovascular risk in children. At the time of the report's publication, the evidence of a contribution from salt intake to raised blood pressure in children was limited and it was not clear whether sodium intake in isolation was a factor in the development of hypertension in the young which then tracked into adulthood. Nevertheless, the evidence suggested that long-term consumption of salt by children at levels currently habitual for adults was potentially harmful in later life. SACN therefore advised that it would be inadvisable for children in the UK to become accustomed to adult levels of salt consumption.
- 4.170 For this report, no new SR evidence on the health effects of salt or sodium intake in children aged 1 to 5 years was identified.
- 4.171 SACN therefore continues to endorse its 2003 recommendation that health benefits would be gained from a reduction in average salt intake in order to achieve the recommended target average salt intake levels for this age group.

# Vitamin A

## Physiological requirements

- 4.172 Vitamin A is a fat-soluble vitamin and is required for vision, embryonic growth and development, immune function, and for normal development and differentiation of tissues (SACN, 2005). Vitamin A is obtained from the diet either as preformed vitamin A (mainly retinol and retinyl esters) in foods of animal origin or as provitamin A carotenoids, dietary precursors of retinol, in plant-derived foods (EFSA, 2015b).
- 4.173 Children have a requirement for vitamin A for growth, in addition to the requirement (as in adults) to compensate for the loss of body stores (DH, 1991).

## Current recommendations for vitamin A intake in the UK

- 4.174 The UK government recommends that children aged from 6 months up to 5 years are given vitamin supplements containing vitamin A (as well as vitamins C and D) every day. This is a precautionary measure to ensure that requirements are met at a time when it is difficult to be certain that the diet provides a reliable source of vitamin A (PHE, 2016a). Vitamin A is also included in vitamin drops provided under the Healthy Start scheme in England, Wales and Northern Ireland (see Annex 1, Table A1.2 for details on the scheme). The latest available data (January 2023) indicated that uptake of Healthy Start vitamins by local authority ranged from 46% to 80% (median 62%) in England; 58% to 73% (median 66%) in Wales; and 49% to 56% (median 54%) in Northern Ireland (NHS, 2023a).
- 4.175 The current UK DRVs for vitamin A (Table 4.18) were set by COMA in 1991 (DH, 1991) and remained unchanged after SACN reviewed dietary advice on foods and supplements containing retinol (SACN, 2005). To account for the contribution from provitamin A carotenoids to total vitamin A intake, the total vitamin A content of the diet is usually expressed as micrograms ( $\mu\text{g}$ ) of retinol equivalents (RE):  $1\mu\text{g RE} = 1\mu\text{g retinol} = 6\mu\text{g beta-carotene} = 12\mu\text{g other carotenoids with provitamin A activity}$  (WHO and FAO, 1967).

**Table 4.18 DRVs for vitamin A for children aged 1 to 6 years<sup>1</sup>**

Age	LRNI	EAR	RNI	TUL for retinol <sup>2</sup>
	RE µg per day			
1 to 3 years	200	300	400	800
4 to 6 years	200	300	400	1100

Abbreviations: EAR, Estimated Average Requirement; LRNI, Lower Reference Nutrient Intake; RE, Retinol Equivalents; RNI, Reference Nutrient Intake; TUL, Tolerable Upper Level

<sup>1</sup> Source: (DH, 1991).

<sup>2</sup> The TUL for retinol was set by the European Scientific Committee on Food (SCF). Note that the Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT) does not set a TUL for children aged 1 to 6 years (see paragraph 4.178).

- 4.176 In 2005, SACN set a Guidance Level (GL) for retinol intake for adults, which represents an approximate indication of levels that would not be expected to cause adverse effects. The GL was derived from limited data and is less secure than the Safe Upper Limit (SUL), which represents an intake level that can be consumed daily over a lifetime without significant risk to health and is based on adequate available evidence (SACN, 2005). SACN did not set a SUL or GL for retinol intake for children because of insufficient data.
- 4.177 In 2002, the European Scientific Committee on Food (SCF) established a TUL for preformed vitamin A (retinol) for children as well as for adults (Table 4.18). The TUL represents the highest level of daily nutrient intake that is likely to pose no risk of adverse health effects for almost all individuals in the general population (SACN, 2005). In its [statement on the potential risks from high levels of vitamin A in the infant diet](#) (COT, 2013) the Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT) stated that while high intakes of preformed vitamin A can be acutely toxic, high intakes of beta-carotene and other provitamin A carotenoids from food alone have not been found to cause toxicity.
- 4.178 In an [addendum to its 2013 statement](#), COT considered the TUL values derived by the European SCF for children aged 1 to 6 years and concluded that these were not appropriate for this age group. COT concluded that TULs could not be established for children aged 1 to 6 years based on the currently available data. However, the COT found no scientific basis for changing current UK government advice (see chapter 10).

## Vitamin A intake in the UK

- 4.179 Intake data in children in the UK aged 12 to 60 months from the DNSIYC and NDNS years 2016 to 2019 are presented in Table 4.19.

**Table 4.19 Vitamin A intake (RE) in children aged 12 to 60 months in the UK (DNSIYC and NDNS years 2016 to 2019)**

Age	Intake from diet and supplements Mean intake as % RNI	Intake from diet only Mean intake as % RNI	Intake from diet and supplements % below LRNI	Intake from diet only % below LRNI	Intake from diet and supplements % above TUL for retinol <sup>3</sup>	Intake from diet only % above TUL for retinol <sup>3</sup>	Number of participants
12 to 18 months <sup>1</sup>	175	169	2	2	1.9	0.9	1275
18 to 47 months <sup>2</sup>	136	115	8	9	4.2	0.4	306
48 to 60 months <sup>2</sup>	153	132	7	10	1.7	0.0	102

Abbreviations: LRNI, Lower Reference Nutrient Intake; RE, retinol equivalents; RNI, Reference Nutrient Intake; TUL, Tolerable Upper Limit

<sup>1</sup> Data from DNSIYC 2011 (DH, 2013).

<sup>2</sup> Data from NDNS years 2016 to 2019.

<sup>3</sup> Set by the European Scientific Committee on Food (SCF). Note that COT does not set a TUL for children aged 1 to 6 years (see paragraph 4.178).

- 4.180 Mean vitamin A intake was above the RNI in all age groups. At the lower end, 9% of children aged 18 to 47 months and 10% of children aged 48 to 60 months had vitamin A intake (RE) from food sources below the LRNI. However, the data should be interpreted with some caution given concerns with the level of underreporting of intakes in the group of children with intakes below the LRNI (paragraph 4.18).
- 4.181 At the same time, 4.2% of children aged 18 to 47 months had retinol intakes above the TUL that was set by the European SCF, which appear to be driven by retinol-containing dietary supplements (Table 4.22). However, given COT's concerns regarding the TUL set by the European SCF (see paragraph 4.178), the data should be interpreted with caution.
- 4.182 Secondary analysis of the data from NDNS (years 2008 to 2019) was conducted to determine the characteristics of children (in 2 age groups: 18 to 47 months, and 48 to 60 months) with intakes below the LRNI for vitamin A and those with intakes at or above the LRNI (see Annex 11, Tables A11.17 to A11.20). Characteristics that were considered were age, sex, ethnicity and household socioeconomic status.
- 4.183 For children aged 18 to 47 months, Black or Black British children made up 9% of the children with intakes below the LRNI, but only 4% of the whole sample. Children from households where the HRP never worked (outside the home) made up 18% of the children with intakes below the LRNI, but only 6% of the whole

sample. However, some caution should be taken when interpreting the findings because the total number of children with intakes below the LRNI was small (n=95).

- 4.184 The number of children aged 48 to 60 months with intakes below the LRNI for vitamin A was too small to allow a similar breakdown of characteristics in this group.
- 4.185 Time trend analysis of NDNS data (years 2008 to 2019) in children aged 18 to 36 months indicated an average annual reduction in vitamin A intake (from food sources only) of  $-2.4\%$  (95% CI  $-3.5$  to  $-1.2\%$ ), equivalent to a reduction of 23% over an 11-year period (Bates et al, 2020). However, over the same 11-year period, there was no significant change in the percentage of children with intakes (from food sources only) below the LRNI (0.5 percentage point average change per year; 95% CI  $-0.2$  to  $1.1$  percentage points). No time trend data was available for the other age groups.
- 4.186 There are several challenges in assessing vitamin A intake due to its uneven distribution in foods, some of which are consumed irregularly. The recording of food intake in DNSIYC and NDNS is restricted to a short continuous time period (4 days) and therefore estimated intake values may not represent intakes over the longer term for vitamin A (and other micronutrients) which are not widely distributed in foods. That is, the habitual intake of rarely consumed foods may be over or underestimated at an individual level (although estimates of population mean intake should be reliable) (SACN, 2018). Possible overage, that is the practice of adding retinol to animal feed at levels higher than those stated on the label, adds further uncertainty to estimated intake values (SACN, 2005).
- 4.187 For the small number of children with intakes that exceeded the TUL, it is not possible to say definitively how much above the TUL an intake might be to be of concern as this would depend on how long the TUL was exceeded for and the size and age of the individual. In addition, dietary intakes, particularly consumption of foods that are rich in vitamin A (for example, liver products), vary from day to day so that many individuals reporting vitamin A intakes above the TUL are unlikely to have consistently high intakes over a prolonged duration. The TUL is intended to reflect risks relating to long term exposure and is not a threshold above which adverse effects will occur in the short-term; thus, an occasional exceedance above the TUL is not of concern. However, the higher or more sustained the exceedance, the greater the risk of adverse effects occurring.

## **Vitamin A intake and deprivation**

- 4.188 Vitamin A intake by IMD (see Glossary) in children aged 18 to 60 months is presented in Table 4.20. Mean vitamin A intake was highest in quintile 1 (least deprived) ( $562\mu\text{g RE}$  per day) and lowest in quintile 5 (most deprived) ( $421\mu\text{g RE}$  per day). Mean intake in quintile 1 was significantly higher than in quintiles 4 and

5, while the mean intake in quintile 2 was significantly higher than that in quintile 5 (as indicated by non-overlapping confidence intervals).

**Table 4.20 Vitamin A intake (from diet only) by IMD quintile in children aged 18 to 60 months in England (NDNS years 2008 to 2019)**

Vitamin A intake µg RE/day	IMD quintile 1 (least deprived)	IMD quintile 2	IMD quintile 3	IMD quintile 4	IMD quintile 5 (most deprived)
Mean (95%CI)	562 (523 to 601)	540 (500 to 579)	520 (481 to 560)	489 (455 to 522)	421 (396 to 445)
Number of participants	210	211	182	234	277

Abbreviations: IMD, index of multiple deprivation; RE, retinol equivalents  
Data from NDNS years 2008 to 2019.

- 4.189 Evidence that vitamin A intake follows a social gradient in the UK is supported by an analysis on NDNS data (years 2012 to 2017) in children aged 18 to 36 months that used a narrower measure of household socioeconomic status (equivalised household income, see Glossary). This analysis indicated that every £10,000 increase in equivalised household income was associated with an average increase in vitamin A intake (from food sources only) of 5.14µg per day (95% CI 2.23 to 8.14µg per day) (Bates et al, 2019).

## Dietary sources of vitamin A

- 4.190 Vitamin A is a fat-soluble vitamin obtained from the diet either as preformed vitamin A (mainly retinol and retinyl esters) in foods of animal origin or as provitamin A carotenoids, dietary precursors of retinol, in plant-derived foods (EFSA, 2015b).
- 4.191 Natural sources of retinol are foods of animal origin, dairy products, and fish. Liver and liver products are particularly rich sources of retinol. Fortified foods (especially margarine) and supplements (including fish liver oils) are also important sources of retinol (SACN, 2005). Foods rich in provitamin A carotenoids (alpha- and beta-carotenes, beta-cryptoxanthin) include vegetables, such as sweet potatoes, carrots and dark green leafy vegetables, and fruits (EFSA, 2015b).
- 4.192 The absorption efficiency of retinol is high, between 70 to 90% while the bioavailability of provitamin A carotenoids (that is, the amount available for utilisation) is lower, ranging from less than 5% to 50% (SACN, 2005).
- 4.193 To take account of the contribution from provitamin A carotenoids, the total vitamin A content of the diet is usually expressed as micrograms ( $\mu\text{g}$ ) of RE (see Glossary).
- 4.194 The contribution to vitamin A intake from animal sources (retinol) and plant-based sources (total carotene) in children aged 1 to 5 years in the UK is presented in Table 4.21.

**Table 4.21 Sources of vitamin A intake (retinol and total carotene) in children aged 12 to 60 months in the UK (DNSIYC and NDNS 2016 to 2019)**

Age group	Retinol ( $\mu\text{g}/\text{day}$ ) intake from diet and supplements Mean (SD)	Retinol ( $\mu\text{g}/\text{day}$ ) intake from diet only Mean (SD)	Total carotene ( $\mu\text{g}/\text{day}$ ) intake from diet and supplements Mean (SD)	Total carotene ( $\mu\text{g}/\text{day}$ ) intake from diet only Mean (SD)	Number of participants
12 to 18 months <sup>1</sup>	341 (183)	319 (147)	2144 (1871)	2141 (1870)	1275
18 to 47 months <sup>2</sup>	319 (221)	236 (121)	1347 (1214)	1345 (1215)	306
48 to 60 months <sup>2</sup>	306 (223)	225 (134)	1827 (2047)	1826 (2047)	102

<sup>1</sup> Data from DNSIYC 2011.

<sup>2</sup> Data from NDNS years 2016 to 2019.

- 4.195 Intake of retinol from food decreased with age, potentially reflecting the drop in milk or formula milks consumption in the older age groups (Table 4.21).

- 4.196 The main dietary contributors (including from supplements) to vitamin A intake in children with intakes below the LRNI for vitamin A were compared with those in children with intakes above the LRNI. Detailed results of this analysis of NDNS data (years 2008 to 2019) are presented in Annex 11, Tables A11.27 to A11.28 in children aged 18 to 47 months. The contribution of these food groups to TDEI is also shown. For children aged 48 to 60 months, the number of children with intakes below the LRNI for iron was too small to be presented.
- 4.197 For children aged 18 to 47 months, the difference in the relative and absolute contributions of food groups to vitamin A intakes between children with intakes at or above the LRNI compared with those with intakes below the LRNI was most pronounced for carrots and dietary supplements (Annex 11, Table A11.17). Carrots contributed 15% (106µg per day) to vitamin A intakes in the children who met or exceeded the LRNI compared with 4.1% (7µg per day) in the children with intakes below the LRNI. Dietary supplements contributed 7.2% (65µg per day) to vitamin A intake in the children with intakes at or above the LRNI but did not contribute to vitamin A intakes in the children below the LRNI.
- 4.198 While children with intakes below the LRNI obtained a higher proportion of their vitamin A intake from milk and cream, cheese and yoghurt, fromage frais and dairy desserts, their absolute intake of vitamin A (µg per day) from these foods was lower than that in children with intakes at or above the LRNI (Annex 11, Table A11.17). This may be accounted for by their lower TDEI, smaller body size, a greater tendency to underreport energy intakes (see paragraph 4.18) or a combination of these factors.
- 4.199 Only 46 children aged 18 to 47 months had high vitamin A intakes (Table 4.22). For the 48 to 60 month age group, the number of children with high vitamin intakes (n=8) was too small for data on the dietary contributors to be presented.

**Table 4.22 Contributors to retinol intake in children aged 18 to 47 months who exceeded the TUL<sup>2</sup> for vitamin A (including from supplements)<sup>1</sup>**

Food group	% contribution <sub>3,4</sub>	µg per day
Retinol containing dietary supplements	[58.5]	[648]
Meat, meat products and dishes	[16.8]	[259]
Milk <sup>5</sup>	[7.2]	[80]
Formula milks <sup>6</sup>	[3.1]	[42]
Butter and fat spreads	[2.8]	[31]
Cheese <sup>5</sup>	[2.5]	[25]
Biscuits, buns, cakes, pastries, fruit pies, puddings	[2.2]	[23]
Eggs, products and dishes	[1.7]	[16]
Yoghurt, fromage frais, dairy desserts <sup>5</sup>	[1.2]	[12]
Pizza, pasta, rice products and dishes	[0.9]	[10]
Commercially manufactured foods and drinks specifically marketed for infants and young children	[0.9]	[10]
Ice cream <sup>5</sup>	[0.8]	[9]
Number of participants	46	46

[ ] Data presented in square brackets denotes that the estimates are based on a cell size ≥30 and <50

<sup>1</sup> Data from NDNS years 2008 to 2019.

<sup>2</sup> Set by the European Scientific Committee on Food (SCF). Note that the COT does not set a TUL for children aged 1 to 6 years (see paragraph 4.178).

<sup>3</sup> Food groups that contributed less than 0.5% to retinol intake are not presented.

<sup>4</sup> Average % contribution for each food group has been calculated from the % contribution for each individual. Non-consumers are included in the average.

<sup>5</sup> Includes non-dairy alternatives.

<sup>6</sup> Formula milks include milks marketed to children over the age of 1 year, also known as 'toddler milks' and 'growing-up milks' (see Glossary).

- 4.200 Dietary supplements were the principal source of high intake of vitamin A (Table 4.22) although the risk of adverse effects from high intakes is unclear given COT's caution with the TUL set by the European SCF (see paragraph 4.178). It should be noted that COT concluded that the possibility of adverse effects cannot be excluded in high consumers, primarily those who regularly eat liver (see Table 10.1, chapter 10). However, if effects did occur it would be in a small proportion of consumers.
- 4.201 Although data are not available to determine what proportion of the children with high intakes of vitamin A would have been eligible to receive vitamin A supplements through the Healthy Start scheme (Annex 1, Table A1.2), an analysis undertaken by COT of data from DNSIYC and NDNS (years 2009 to 2012) indicated that among children eligible for the scheme, uptake of these vitamins was unlikely to result in intakes above the TUL (COT, 2017).

## Assessment of vitamin A status

- 4.202 Vitamin A absorbed in excess of immediate needs is stored in the liver. The size of liver reserves is therefore one objective measure of vitamin A status, but it cannot readily be determined in individuals (DH, 1991).
- 4.203 Plasma retinol concentration has been used as a biochemical measure of habitual dietary intake (retinol exposure). Plasma retinol concentrations are homeostatically controlled over a wide range of liver reserves and normal levels of consumption are usually unrelated to plasma concentrations. Mean plasma retinol values fall when liver stores are exhausted and increase at liver concentrations above 300µg/g. When the capacity for storage of retinol in liver is exceeded or the rate of intake is greater than the rate it can be removed by the liver, there is a marked increase in plasma concentrations. Therefore, plasma retinol concentrations are insensitive indicators of intake or body reserves unless they are very high or very low (SACN, 2005).
- 4.204 Plasma retinol concentrations are reduced during the inflammatory response accompanying conditions such as fever and infection (SACN, 2005). Infection can lower mean plasma or serum retinol concentration by as much as 25% independently of vitamin A intake (EFSA, 2015b).
- 4.205 Vitamin A status (plasma retinol concentrations) in children aged 12 to 60 months from the DNSIYC and NDNS (years 2008 to 2019) are presented in Table 4.23. Concentrations below 0.35 µmol/l are considered to reflect severe deficiency and concentrations between 0.35 µmol/l and 0.70 µmol/l to reflect mild deficiency. It should be noted that the evidence for these thresholds is confined mainly to non-elderly adults (Bates et al, 1997).

**Table 4.23 Vitamin A status (plasma retinol) in children aged 12 to 60 months in the UK (DNSIYC and NDNS years 2008 to 2019)**

Age	Plasma retinol (µmol/l) Mean (SD)	% below 0.35µmol/l <sup>1</sup>	% at 0.35 to 0.70µmol/l <sup>1</sup>	Number of Participants
12 to 18 months <sup>2</sup>	No data	No data	No data	No data
18 to 47 months <sup>3</sup>	1.03 (0.26)	0	7	103
48 to 60 months <sup>3</sup>	[1.12 (0.30)] <sup>4</sup>	[0] <sup>e</sup>	[10] <sup>e</sup>	41

<sup>1</sup> Thresholds confined mainly to non-elderly adults (Bates et al, 1997).

<sup>2</sup> Plasma retinol was not measured in this age group.

<sup>3</sup> Data from NDNS years 2008 to 2019.

<sup>4</sup> [ ] data presented in square brackets denotes that the estimates are based on a cell size between 30 and 49.

- 4.206 With the sample sizes being small, the data suggest that there is no evidence of severe deficiency in children aged 12 to 60 months but some evidence that 10% of children aged 18 to 47 months had a retinol concentration at a level associated with mild deficiency in an adult population.

## **Systematic review evidence identified on vitamin A and health outcomes**

### **Interventions to improve vitamin A status**

#### **Vitamin A supplementation**

- 4.207 For this report, 1 SR with MA (Imdad et al, 2017) was identified that examined the effect of vitamin A supplementation on serum retinol concentrations and vitamin A deficiency (VAD). Most interventions lasted up to 1 year and were performed in LICs, LMICs and UMICs. The SR did not report findings stratified by the baseline vitamin A status of participants. Therefore, the evidence is described below but was not graded (see paragraphs 4.22 and 4.23).
- 4.208 Imdad et al (2017) (AMSTAR confidence rating: high) reported that vitamin A supplementation increased serum retinol concentrations in children aged up to 5 years compared to the control group using a fixed-effects model but heterogeneity was high (SMD 0.26; 95% CI 0.22 to 0.30;  $p < 0.001$ ;  $I^2 = 95\%$ ; 14 trials, 11,788 participants). When the analysis was repeated using a random-effects model to test for small study bias, a larger effect size (SMD 0.50; 95% CI 0.30 to 0.70;  $p$ -value NR) together with an asymmetrical funnel plot suggested that small studies reported larger effects. The SR also reported that vitamin A supplementation reduced the risk of VAD in children up to 5 years old (RR 0.71; 95% CI 0.65 to 0.78;  $p < 0.001$ ;  $I^2 = 78\%$ ; fixed effects model; 4 trials, 2262 participants), but heterogeneity was high.

#### **Vitamin A fortification**

- 4.209 Two SRs with MA (Das et al, 2013; Eichler et al, 2012) were identified that examined the effect of fortification with vitamin A (alone or with other micronutrients) on serum retinol concentrations and VAD. Common food vehicles were milk, staple cereals, biscuits, monosodium glutamate, sugar, flour and seasoning. Interventions lasted beyond 6 months and were performed in LMIC and UMIC. Neither SR reported findings stratified by the baseline vitamin A status of participants. Therefore, the evidence is described below but was not graded (see paragraphs 4.22 and 4.23).

- 4.210 Das et al (2013) (AMSTAR confidence rating: critically low) reported that vitamin A fortification increased serum retinol concentrations compared with the control group but with high heterogeneity (SMD 0.61; 95% CI: 0.39 to 0.83;  $p < 0.0001$ ;  $I^2 = 84\%$ ; random-effects model; 5 effect estimates from 3 RCTs, 2362 participants). Three of the 5 effect estimates included children aged 3 to 6 years old (55.5% weighting in the MA). Das et al (2013) also reported that vitamin A fortification had no effect on prevalent VAD compared with the control group but with high heterogeneity (RR 0.39; 95% CI 0.09 to 1.74;  $p = 0.22$ ;  $I^2 = 88\%$  random effects model; 4 effect estimates from 2 RCTs, 1465 participants). Three out of the 4 effect estimates included children aged 3 to 6 years old (70.9% weighting in the MA).
- 4.211 Eichler et al (2012) AMSTAR confidence rating: low) reported that vitamin A fortification (with other micronutrients) also increased serum retinol concentrations in children aged 6 months to 3 years (MD 3.7 $\mu$ g/dl; 95% CI 1.3 to 6.1 $\mu$ g/dl;  $p$ -value NR;  $I^2 = 37\%$ ; 4 RCTs, participants NR).

## **Vitamin A and interactions with other nutrients**

- 4.212 Among the macronutrients, it is well established that the absorption of vitamin A (retinol, retinyl esters and carotenoids) as a 'fat-soluble' vitamin is affected by dietary fat intake. Only 3 to 5g of dietary fat per meal is needed to ensure efficient absorption of beta-carotene in humans (Tanumihardjo et al, 2016). Adequate intake of high quality protein has also been shown to improve the bioconversion of provitamin A carotenoids to retinol in the small intestine (Tanumihardjo et al, 2016).
- 4.213 In terms of micronutrients, it has been suggested that poor zinc status may negatively affect vitamin A status biomarkers given that zinc and vitamin A work synergistically for many functions in the body (Tanumihardjo et al, 2016). For example, zinc deficiency has been shown to affect the transport of retinol from the liver into the systemic circulation in animal models. However, no consistent relationship between zinc and vitamin A status has been shown in humans (EFSA, 2015b).
- 4.214 Deficiency and excess of vitamin A can also lead to impaired vitamin D function by impacting vitamin D receptor activation and binding to the retinoid X receptor (RXR), affecting the ability of 1,25(OH)<sub>2</sub>D to exert genomic and non-genomic effects (Bouillon et al, 2019).
- 4.215 See [Vitamin A deficiency and anaemia](#) for a discussion on interactions between vitamin A and iron.

## Vitamin A and health

- 4.216 VAD can adversely affect several physiological functions, such as vision, immunity, and worsening of low iron status (EFSA, 2015b).

### Vitamin A deficiency and ophthalmological outcomes

- 4.217 Vitamin A is essential for maintaining the visual cycle in the retina (EFSA, 2015b). VAD of sufficient duration or severity can lead to several visual disorders such as xerophthalmia, the leading cause of preventable childhood blindness globally. It encompasses a spectrum of clinical ocular manifestations of VAD, from milder stages of night blindness and Bitot's spots, to potentially blinding stages of corneal xerosis, ulceration and necrosis (WHO, 2009).

### Evidence from supplementation trials

- 4.218 One SR with MA (Imdad et al, 2017) was identified that examined the effect of vitamin A supplementation on ophthalmological outcomes in children mostly aged up to 5 years. Most interventions lasted beyond 1 year and were conducted in LICs, LMICs or UMICs. The SR did not report findings stratified by the baseline vitamin A status of participants. Therefore, the evidence is described below but was not graded (see paragraphs 4.22 and 4.23).
- 4.219 Imdad et al (2017) (AMSTAR 2 confidence rating: high) reported that children supplemented with vitamin A had a decreased risk of incident night blindness (RR 0.53; 95% CI 0.28 to 0.99; p-value NR; fixed-effects model; 1 RCT, participants NR) and prevalent night blindness (RR 0.32; 95% CI 0.21 to 0.50; p-value NR;  $I^2=0\%$ ; fixed-effects model; 2 RCTs, 22,972 participants) compared with the control group.
- 4.220 Imdad et al (2017) also reported that vitamin A supplementation had no effect on Bitot's spots incidence (RR 0.93; 95% CI 0.76 to 1.14; p-value NR;  $I^2=N/A$ ; fixed-effects model; 5 RCTs, 1,063,278 participants) but decreased the risk of prevalent Bitot's spots (RR 0.42; 95% CI 0.33 to 0.53; p-value NR;  $I^2=49\%$ ; fixed effects model), incident xerophthalmia (RR 0.85; 95% CI 0.70 to 1.03; p-value NR;  $I^2=63\%$ ; fixed-effects model; 3 RCTs, participants NR) and prevalent xerophthalmia (RR 0.31; 95% CI 0.22 to 0.45;  $I^2=0\%$ ; fixed-effects model; 2 RCTs, 22,972 participants).

### Vitamin A deficiency and immune function

- 4.221 The importance of vitamin A in immune function is well established (Stephensen, 2001). VAD impairs innate immunity by impeding normal regeneration of mucosal barriers damaged by infection and diminishing the function of frontline immune cells such as neutrophils and macrophages. Vitamin A is also essential for

adaptive immunity, playing a role in the development of T-helper cells and modulates antibody-mediated responses to infection.

- 4.222 Pre-existing VAD may worsen infection in young children (WHO, 2009). In LMICs, VAD in infants and young children has been associated with increased mortality from infection, and increased infectious morbidity (Imdad et al, 2017).
- 4.223 Vitamin A intake and body stores can also be reduced during an inflammatory response to infection or injury by depressing appetite, reducing intestinal absorption, and increasing urinary excretion of vitamin A (Rubin et al, 2017). Inflammation can also cause the sequestration of vitamin A in the liver, leading to low serum retinol concentrations (hyporetinolaemia), a condition that has been reported in children in association with acute infections (for example, measles, malaria, diarrhoea, human immunodeficiency viruses) in developing countries (Rubin et al, 2017).

## **Vitamin A deficiency and growth**

### **Evidence from supplementation trials**

- 4.224 One SR with MA (Ramakrishnan et al, 2009) was identified that examined the effect of vitamin A supplementation on growth outcomes in children aged under 5 years from mostly LMICs. The SR did not report findings stratified by the baseline vitamin A status of participants. Therefore, the evidence is described below but was not graded (see paragraphs 4.22 and 4.23).
- 4.225 Ramakrishnan et al (2009) (AMSTAR 2 confidence rating: critically low) reported that vitamin A supplementation (with and without other micronutrients) in children aged 1 to 5 years had no effect on linear growth (Cohen's effect size 0.08; 95% CI -0.18 to 0.34; p-value NR; heterogeneity:  $p < 0.05$ ; random-effects model; 17 RCTs, 69,320 participants), weight gain (Cohen's effect size -0.03; 95% CI -0.23 to 0.18; p-value NR; heterogeneity:  $p < 0.01$ ; random-effects model; 17 RCTs, 69,320 participants) or change in weight-for-height z-score (Cohen's effect size 0.01; 95% CI -0.06 to 0.09; p-value NR; heterogeneity; NR; random-effects model; 5 RCTs, participants NR) compared with the control group. However, there was significant heterogeneity in the summary estimates and it is unclear whether the null findings would generalise to children with VAD.

### **Vitamin A deficiency and anaemia**

- 4.226 Anaemia can result from VAD due to multiple roles of vitamin A in supporting iron mobilisation and transport, and production of red blood cells (WHO, 2009). Administering vitamin A has been shown to enhance haemoglobin response to iron supplementation during adolescence and pregnancy (Tanumihardjo et al, 2016).

- 4.227 One SR with MA (Das et al, 2013) was identified that examined the effect of vitamin A fortification on iron status in children from LMICs. The SR did not report findings stratified by the baseline vitamin A status of participants. Therefore, the evidence is described below but was not graded (see paragraphs 4.22 and 4.23).
- 4.228 Das et al (2013) (AMSTAR 2 confidence rating: critically low) reported that vitamin A fortification (of monosodium glutamate, sugar or flour) increased haemoglobin concentration in children aged 48 to 72 months compared with the control group (SMD 0.48; 95% CI: 0.07 to 0.89; p=0.02; I<sup>2</sup>=93%; random-effects model; 2 RCTs, 1538 participants).

# Vitamin D

## Physiological requirements

- 4.229 Vitamin D, together with calcium and phosphorus, is required during infancy and early childhood to meet the demands of rapid growth for healthy skeletal development. Prolonged deficiency of vitamin D during periods of bone growth in children leads to a failure or delay of endochondral calcification at the growth plates of the long bones which results in rickets and an accumulation of excess unmineralised osteoid (bone matrix) in all bones; the low mineral to bone matrix ratio in bone results in osteomalacia (Pettifor, 2012). The main signs of rickets are skeletal deformity with bone pain or tenderness; and muscle weakness. Deficiencies of calcium and phosphorus can also cause rickets (SACN, 2016).

## Sources of vitamin D

- 4.230 The 2 major forms of vitamin D are vitamin D2 (ergocalciferol) and vitamin D3 (cholecalciferol). The main sources of vitamin D are sunlight exposure (skin synthesis) and foods or dietary supplements (containing either vitamin D2 or D3). Between the months of April and September in the UK, skin synthesis is the main source of vitamin D for most people. Vitamin D3 is the only form produced cutaneously. Vitamin D2 is formed in fungi and yeast by UVB exposure of ergosterol (SACN, 2016).
- 4.231 Dietary sources are essential when the amount of sunlight containing UVB light is limited (for example, in winter) or exposure to sunlight containing UVB light is restricted (for example, lack of time spent outdoors or little skin exposure) (SACN, 2016).
- 4.232 Dietary sources of vitamin D include natural food sources, fortified foods and supplements. There are few naturally rich food sources of vitamin D. Those that contain significant amounts are mostly of animal origin and contain vitamin D3 (for example, oily fish, red meat, egg yolk). Animal products (for example, meat, fat, liver, kidney) also contain 25-hydroxyvitamin D (25(OH)D), which is the major circulating metabolite of vitamin D (and is widely used as a biomarker of vitamin D status) (SACN, 2016). Wild mushrooms are a rich source of vitamin D2. Fortified foods (for example, breakfast cereals, fat spreads) and dietary supplements contain either vitamin D2 or D3.

## Current recommendations for vitamin D intake in the UK

- 4.233 The current UK recommendation for vitamin D is to give children aged 1 to 5 years a daily supplement containing 10µg (400 IU) of vitamin D (SACN, 2016). Vitamin D is also included in vitamin drops provided under the Healthy Start scheme in England, Wales and Northern Ireland; while in Scotland, vitamin D drops are provided for free to all children from birth to age 3 years (see Annex 1, Table A1.2 for details). The latest available data (January 2023) indicated that uptake of Healthy Start vitamins by local authority ranged from 46% to 80% (median 62%) in England; 58% to 73% (median 66%) in Wales; and 49% to 56% (median 54%) in Northern Ireland (NHS, 2023a).

## Vitamin D intake in the UK

- 4.234 Mean vitamin D intake (% RNI) for vitamin D in children not breastfed and children breastfed (excluding breast milk) from DNSIYC and NDNS years 2016 to 2019 are presented in Table 4.24.
- 4.235 For children not breastfed, mean intake (including from dietary supplements) was 55% of the RNI in children aged 12 to 18 months, 30% in children aged 18 to 47 months, and 28% in children aged 48 to 60 months. For breastfed children aged 12 to 18 months, mean intake (excluding the contribution from breast milk) was 26% of the RNI from food and 37% from food and supplements.

**Table 4.24 Vitamin D intake in children aged 12 to 60 months in the UK (DNSIYC and NDNS years 2016 to 2019)**

	Mean intake as % of RNI Non-breastfed <sup>1</sup> Intake from diet and supplements	Mean intake as % of RNI Non-breastfed <sup>1</sup> Intake from diet only	Mean intake as % of RNI Breastfed excluding breast milk <sup>2</sup> Intake from diet and supplements	Mean intake as % of RNI Breastfed excluding breast milk <sup>2</sup> Intake from diet only	Number of participants
12 to 18 months <sup>3</sup>	55	50	37	26	1275
18 to 47 months <sup>4</sup>	40	24	Not applicable	Not applicable	306
48 to 60 months <sup>4</sup>	39	25	Not applicable	Not applicable	102

Abbreviations: LRNI, Lower Reference Nutrient Intake; RNI, Reference Nutrient Intake

<sup>1</sup> Vitamin D intake does not include values for breastfed children as the vitamin D content of breast milk is not known. Note breastfeeding status is defined by whether it was recorded in the 4-day diary (Lennox et al, 2013).

<sup>2</sup> Vitamin D intake includes values for breastfed children excluding the contribution from breast milk (therefore excluding any exclusively breastfed children (n=2) as the vitamin D content of breast milk is not known. Note breastfeeding status is defined by whether it was recorded in the 4-day diary (Lennox et al, 2013).

<sup>3</sup> Data from DNSIYC 2011 (DH, 2013).

<sup>4</sup> Data from NDNS years 2016 to 2019.

4.236 NDNS data (years 2016 to 2019) indicated that only 25% of children aged 18 to 36 months were given a vitamin D supplement.

4.237 Time trend analysis of NDNS data (years 2008 to 2017) in children aged 18 to 36 months showed a non-significant average annual change in vitamin D intake of 1.2% (95% CI -1.1 to 3.5%) for the 9-year period (Bates et al, 2019). No time trend data was available for the other age groups.

## Vitamin D intake and deprivation

4.238 Vitamin D intake by IMD (see Glossary) in children aged 18 to 60 months is presented in Table 4.25

**Table 4.25 Vitamin D intake (from diet only) by IMD quintile in children aged 18 to 60 months in England (NDNS years 2008 to 2019)**

Vitamin D intake µg/day	IMD quintile 1 (least deprived)	IMD quintile 2	IMD quintile 3	IMD quintile 4	IMD quintile 5 (most deprived)
Mean (95%CI)	1.83 (1.64 to 2.02)	2.10 (1.92 to 2.28)	2.16 (1.89 to 2.43)	2.09 (1.86 to 2.31)	2.16 (1.91 to 2.40)
Number of participants	210	211	182	234	277

Abbreviations: IMD, index of multiple deprivation.

Data from NDNS years 2008 to 2019.

4.239 The analysis indicated no apparent relationship between vitamin D intake and IMD (as indicated by overlapping confidence intervals). However, IMD is a broad measure of deprivation. Another analysis of NDNS data (years 2012 to 2017) in children aged 18 to 36 months that used a narrower measure of household socioeconomic status (equivalised household income, see Glossary) indicated that every £10,000 increase in equivalised household income was associated with an average increase in vitamin D intake (from food sources only) of 4.66µg per day (95% CI 0.85 to 8.62µg per day) (Bates et al, 2019). The difference in findings between the IMD analysis and the analysis based on household income suggests that diet quality (at least with respect to vitamin D intake) may be more closely linked with affordability of foods than other aspects of an individual's living environment.

## Vitamin D intake by ethnic group in the UK

4.240 Data on vitamin D intake by ethnic group from DNSIYC and the NDNS (years 2016 to 2019) are presented in Table 4.26. Sample numbers were insufficient to analyse data from specific ethnic groups.

**Table 4.26 Vitamin D intake ( $\mu\text{g}$  per day) by ethnic group for young children in the UK (DNSIYC and NDNS years 2016 to 2019)**

Age	White <sup>3</sup> Intake from diet and supplements Mean (SD)	White <sup>3</sup> Intake from diet only Mean (SD)	Other ethnic groups <sup>4</sup> Intake from diet and supplements Mean (SD)	Other ethnic groups <sup>4</sup> Intake from diet only Mean (SD)
12 to 18 months <sup>1</sup>	3.6 (3.6)	3.3 (3.2)	4.7 (4.5)	3.8 (4.0)
18 to 60 months <sup>2</sup>	2.9 (2.8)	2.3 (2.1)	3.1 (3.1)	2.9 (3.4)

<sup>1</sup> Data from DNSIYC 2011 (DH, 2013).

<sup>2</sup> Data from NDNS 2016 to 2019.

<sup>3</sup> 1085 participants in the 12 to 18 months age group. 343 participants in the 18 to 60 months age group.

<sup>4</sup> 190 participants in the 12 to 18 months age group (81 South Asian; 109 other ethnicity); 63 participants in the 18 to 60 months age group. Sample sizes were insufficient to analyse data from specific ethnic groups.

## Assessment of vitamin D status

4.241 In the UK, a serum 25(OH)D concentration of less than 25nmol/L has been the threshold adopted to define increased risk of rickets and osteomalacia (DH, 1998; SACN, 2016).

4.242 Vitamin D status in children aged 12 to 60 months from the DNSIYC and the NDNS (years 2008 to 2019) is presented in Table 4.27.

**Table 4.27 Vitamin D status (serum 25(OH)D) in children aged 12 to 60 months in the UK (DNSIYC and NDNS 2008 to 2019)**

Age	25(OH)D nmol/l Mean (SD)	% below 25nmol/l	Number of participants
12 to 18 months <sup>1</sup>	64.3 (24.3)	2	300
18 to 47 months <sup>2</sup>	58.3 (23.2)	9	116
48 to 60 months <sup>2</sup>	[47.7 (21.3)] <sup>3</sup>	[28] <sup>3</sup>	49

Abbreviations: 25(OH)D, 25-hydroxy vitamin D; SD, standard deviation.

<sup>1</sup> Data from DNSIYC 2011 (DH, 2013).

<sup>2</sup> Data from NDNS years 2008 to 2019.

<sup>3</sup> [ ] data presented in square brackets denotes that the estimates are based on a cell size between 30 and 49.

- 4.243 Time trend analysis of NDNS data (years 2008 to 2019) in children aged 18 to 36 months showed no significant change in serum 25(OH)D (0.1 nmol/l; 95% CI -1.4 to 1.5 nmol/l) for the 11-year period, and no significant change in the percentage of children below the 25 (OH) vitamin D threshold of 25 nmol/l (-0.3 percentage point average change per year; 95% CI -1.5 to 0.9 percentage points) (Bates et al, 2020). No time trend data was available for the other age groups.

## **Vitamin D status and deprivation**

- 4.244 An analysis of NDNS data (years 2012 to 2017) suggested that every £10,000 increase in equivalised household income (see Glossary) was associated with an average increase in serum 25(OH)D concentrations of 3.71 nmol/l (95% CI 0.83 to 6.59 nmol/l) in children aged 18 to 36 months (Bates et al, 2019).

## **Vitamin D status and ethnicity**

- 4.245 Lower plasma or serum 25(OH)D concentrations have been observed in people with dark skin pigmentation compared with those with lighter skin colour (SACN, 2016). It is not clear if this is due to skin pigmentation or to physiological or lifestyle differences since dark skin is only one of many cultural and biological factors that could influence the plasma or serum 25(OH)D concentration of individuals from ethnic groups with darker skin pigmentation (SACN, 2016).
- 4.246 Table 4.28 compares the vitamin D status of children aged 12 to 18 months by ethnic group (Lennox et al, 2013). Although the sample size was too small to draw firm conclusions, the data indicated that, compared with white children, children from other ethnic groups were at higher risk of vitamin D deficiency. More recent data in the UK also suggested that children from a Black or Asian background were at higher risk of clinical manifestations of vitamin D deficiency (see [Nutritional rickets and osteomalacia](#)). Data from the NDNS were insufficient to perform a similar analysis in children aged 18 to 60 months.

**Table 4.28 Vitamin D status (25(OH)D) by ethnic group in children aged 12 to 18 months in the UK (DNSIYC)<sup>1</sup>**

	25(OH)D nmol/l <sup>2</sup> Mean (SD) Other ethnic groups <sup>3</sup>	25(OH)D nmol/l <sup>2</sup> Mean (SD) White <sup>4</sup>	% below 25µmol/l Other ethnic groups <sup>3</sup>	% below 25µmol/l White <sup>4</sup>
12 to 18 months	[61.0 (25.7)] <sup>2</sup>	66.1 (24.4)	[4] <sup>2</sup>	1

Abbreviations: 25(OH)D, 25-hydroxy vitamin D; SD, standard deviation.

<sup>1</sup> Data from DNSIYC 2011 (DH, 2013). Note that blood samples were not collected over a full calendar year

<sup>2</sup> [ ] data presented in square brackets denotes that the estimates are based on a cell size between 30 and 49.

<sup>3</sup> 40 participants in the 12 to 18 months age group. Sample sizes were insufficient to analyse data from specific ethnic groups.

<sup>4</sup> 191 participants in the 12 to 18 months age group.

## Systematic review evidence identified on vitamin D and health outcomes

### Interventions to improve vitamin D status

- 4.247 No new evidence was identified from SRs on the effect of vitamin D supplementation on vitamin D status in young children since the SACN report on [‘Vitamin D and health’](#) (SACN, 2016) and the cut-off date for consideration of evidence for this report (November 2022).
- 4.248 In relation to vitamin D fortification, 1 SR without MA was identified that included studies that examined the effect of vitamin D fortification of milk or formula milks on vitamin D status in children aged 1 to 5 years living in HIC, including the UK (Hojsak et al, 2018). However, the focus of the SR was to evaluate the composition of ‘Young child formula’ (that is, formula milks targeted at children aged 1 to 3 years) and their nutritive role in European children. Vitamin D intake was neither a primary exposure nor included in the search terms of this review. Therefore, the literature search conducted by Hojsak et al (2018) was unlikely to be comprehensive for identifying studies on vitamin D fortification.
- 4.249 Details of the SRs can be found in Annex 5, Table A5.2. Quality assessment of the SRs using the AMSTAR 2 tool can be found in Annex 8 (Table A8.3). Additional data extracted from the primary studies can be found in Annex 9 (Table A9.22). The criteria used to grade the evidence are provided in chapter 2 (Table 2.4, paragraphs 2.54 to 2.58). Summary tables of the evidence grading process for this section are provided in Annex 10 (Table A10.9). Following the methodological

approach described in paragraphs 4.22 and 4.23, the certainty of the evidence was graded.

- 4.250 Hojsak et al (2018) (AMSTAR 2 confidence rating: critically low) included 3 RCTs (in a total of 635 participants, aged 1 to 6 years, from HIC) that reported that vitamin D-fortified milk or formula milk (for 20 weeks in 2 studies and 6 months in 1 study) increased serum vitamin D or decreased risk of vitamin D deficiency (defined as serum 25(OH)D <50 nmol/l in the studies) compared with the control group. One of the RCTs reported that vitamin D fortification prevented an expected decrease in vitamin D status during the winter months in Northern Europe. Average (mean or median) baseline vitamin D status of the children in the intervention groups in the 3 RCTs ranged from 54 to 70 nmol/l. Two of the 3 studies were funded by manufacturers of formula milk.

### Summary: vitamin D fortification and vitamin D status

- 4.251 The evidence identified from SRs on vitamin D fortification (of milk or formula milk) and vitamin D status is summarised in Table 4.29.

**Table 4.29 Summary of the evidence on the relationship between vitamin D fortification and vitamin D status**

Exposure	Outcome	Direction of effect	Certainty of evidence
Vitamin D fortification (of milk or formula milk)	Vitamin D status	Increase in effect	Limited

- 4.252 The available evidence from SRs on vitamin D fortification in children aged 1 to 5 years and vitamin D status is from 1 SR without MA, given a critically low confidence rating using the AMSTAR 2 tool.
- 4.253 Evidence from 3 RCTs included in the SR by Hojsak et al (2018) suggests that vitamin D fortification of milk or formula milk improves vitamin D status or decreases the risk of vitamin D deficiency in children aged 1 to 5 years. One RCT also reported that vitamin D fortification of milk prevents an otherwise frequently observed decrease in serum vitamin D concentration in the winter months in Northern Europe. The evidence was graded 'limited' given the small number studies identified, lack of quantitative data to judge effect sizes and confidence intervals, a literature search that was not comprehensive for vitamin D as an exposure, and a lack of accounting for possible bias from industry funding of the RCTs (see Annex 10, Table A10.9 for details for the grading process).

# Vitamin D and health

## Nutritional rickets and osteomalacia

- 4.254 The re-emergence of nutritional rickets in children in many countries, including the UK, has become a public health concern. A UK-wide surveillance study reported that rickets mostly affects children aged under 5 years (60 months), with an estimated annual incidence of 1.39 (95% CI 1.05 to 1.81) per 100,000 children, and reaching 3.49 (95% CI 2.3 to 5.08) per 100,000 in children aged 12 to 23 months (Julies et al, 2020). Most cases were from Black or South Asian ethnic groups, and at diagnosis, 78% of cases were not reportedly receiving any vitamin supplements (Julies et al, 2020).
- 4.255 It has long been recognised that rickets can also impact tooth development (for details see [Vitamin D status and oral health](#) in chapter 9, and [SACN's 2016 report 'Vitamin D and Health'](#)).
- 4.256 For this report, no new evidence from SRs was identified on the relationship between vitamin D status or vitamin D supplementation and risk of nutritional rickets in children since the SACN report ['Vitamin D and Health'](#) (SACN, 2016) and the cut-off date for consideration of evidence for this report (November 2022).
- 4.257 The SACN report ['Vitamin D and Health'](#) included a total of 44 studies which included measurements of serum 25(OH)D concentration in children with rickets. Evidence was mainly from cross-sectional observational studies and case reports and may therefore have been influenced by confounding. Since most studies did not measure calcium intake it was not clear whether the cause of rickets was vitamin D deficiency and/or calcium deficiency. A distinct threshold serum 25(OH)D concentration above which there is no risk of rickets could not be identified but the data suggested overall that the risk increased at serum 25(OH)D concentration <25nmol/l; this concentration is, however, not a clinical threshold diagnostic of the disease.
- 4.258 No evidence was identified on the relationship between vitamin D status or vitamin D supplementation and osteomalacia in children aged 1 to 5 years in the SACN report ['Vitamin D and Health'](#) (SACN, 2016); and no new evidence from SRs was identified for this report.

## Bone health indices (bone mineral content, bone mineral density, biochemical markers of bone turnover)

- 4.259 For this report, no new evidence from SRs was identified on the relationship between vitamin D status or vitamin D supplementation and bone health indices in children since the SACN report ['Vitamin D and Health'](#) (SACN, 2016) and the cut-off date for consideration of evidence for this report (November 2022).

4.260 In the SACN report [‘Vitamin D and Health’](#), effects of vitamin D supplementation on bone health indices in children aged 1 to 3 years came from 1 cross-sectional study which reported an association between serum 25(OH)D concentration >75nmol/l and higher bone mineral content or bone mineral density at the forearm and whole body but not at the lumber spine. The evidence base for children aged 1 to 3 years was too small to draw any conclusions (SACN, 2016).

# Vitamin C

## Physiological requirements

- 4.262 Vitamin C (ascorbic acid) is a water soluble vitamin. It functions as an anti-oxidant but may also exhibit pro-oxidant properties. Vitamin C is a co-factor and modulator of metabolic reactions and is essential for wound healing and the prevention of scurvy (DH, 1991).
- 4.263 In short-term studies vitamin C has been observed to increase iron uptake from food, but this effect is attenuated in longer term studies and current evidence suggests that vitamin C does not substantially affect iron status(SACN, 2010).

## Current recommendations for vitamin C intake

- 4.264 The current UK recommendation is that children aged from 6 months up to 5 years should be given vitamin supplements containing vitamin C (and vitamins A and D). This is a precautionary measure to ensure that requirements are met at a time when it is difficult to be certain that the diet provides a reliable source of vitamin C (PHE, 2016a). Vitamin C is also included in the vitamin drops provided under the Healthy Start scheme in England, Wales and Northern Ireland (see Annex 1, Table A1.2 for details on the scheme). The latest available data (January 2023) indicated that uptake of Healthy Start vitamins by local authority ranged from 46% to 80% (median 62%) in England; 58% to 73% (median 66%) in Wales; and 49% to 56% (median 54%) in Northern Ireland (NHS, 2023a).
- 4.265 The current UK DRVs for vitamin C were set by COMA in 1991 (DH, 1991). The DRVs for vitamin C in children aged 1 to 6 years are the following:
- LRNI: 8mg per day
  - EAR: 20mg per day
  - RNI: 30mg per day

## Vitamin C intake in the UK

- 4.266 Intake data in children in the UK aged 12 to 60 months from DNSIYC and NDNS (years 2016 to 2019) are presented in Table 4.30.

**Table 4.30 Vitamin C intake in children aged 12 to 60 months in the UK (DNSIYC and NDNS years 2016 to 2019)**

Age	Intake from diet and supplements Mean intake as % RNI	Intake from diet only Mean intake as % RNI	Intake from diet and supplements % participants below LRNI	Intake from diet only % participants below LRNI	Number of participants
12 to 18 months <sup>1</sup>	208	202	0	0	1275
18 to 47 months <sup>2</sup>	248	214	0	0	306
48 to 60 months <sup>2</sup>	270	230	0	0	102

Abbreviations: LRNI, Lower Reference Nutrient Intake; RNI, Reference Nutrient Intake.

<sup>1</sup> Data from DNSIYC 2011 (DH, 2013).

<sup>2</sup> Data from NDNS 2016 to 2019.

4.267 No time trend data from NDNS is available for vitamin C.

## Vitamin C intake and deprivation

4.268 Vitamin C intake (from diet only) by Index of Multiple Deprivation (IMD) in children aged 18 to 60 months is presented in Table 4.31. There was no evidence of any relationship between vitamin C intake (from diet only) and IMD (as indicated by overlapping confidence intervals).

**Table 4.31 Vitamin C intake (from diet only) by IMD quintile in children aged 18 to 60 months in England (NDNS years 2008 to 2019)**

Vitamin C intake mg/day	IMD quintile 1 (least deprived)	IMD quintile 2	IMD quintile 3	IMD quintile 4	IMD quintile 5 (most deprived)
Mean (95%CI)	72.6 (67.7 to 77.6)	67.1 (63.0 to 71.3)	73.9 (68.4 to 79.4)	69.4 (65.2 to 73.7)	66.7 (62.6 to 70.8)
Number of participants	210	211	182	234	277

Abbreviations: IMD, index of multiple deprivation.

Data from NDNS years 2008 to 2019.

## Dietary sources of vitamin C

4.269 The main dietary contributors to vitamin C intake in children aged 18 to 47 months, and 48 to 60 months from NDNS (years 2016 to 2019) are presented in Table 4.32 and Table 4.33.

**Table 4.32 Contributors to vitamin C intake in children aged 18 to 47 months<sup>1</sup>**

Food Group	% contribution <sup>2,3</sup>	mg per day
Fruit	24.1	18.0
Soft drinks <sup>4</sup>	16.8	12.3
Fruit juice and smoothies	12.9	12.2
Milk <sup>5</sup>	10.1	5.3
Vegetables, vegetable products and dishes	8.2	5.4
Potatoes, potato products and dishes	6.6	3.6
Dietary supplements	6.5	7.8
Formula milks <sup>6</sup>	3.0	3.2
Breakfast cereals	1.5	0.9
Commercially manufactured foods and drinks specifically marketed for infants and young children	1.2	1.0
Sugar and chocolate confectionery	1.0	0.9
Number of participants	306	306

<sup>1</sup> Data from NDNS years 2016 to 2019.

<sup>2</sup> Food groups that contribute less than 1% of intake are not presented.

<sup>3</sup> Average % contribution for each food group has been calculated from the % contribution for each individual. Non-consumers are included in the average.

<sup>4</sup> Fizzy drinks, squashes and ready-to-drink still drinks, both those with added sugar and diet types

<sup>5</sup> Includes non-dairy alternatives.

<sup>6</sup> Formula milks include milks marketed to children over the age of 1 year, also known as 'toddler milks' and 'growing-up milks' (see Glossary).

**Table 4.33 Contributors to vitamin C intake<sup>1</sup> in children aged 48 to 60 months**

Food Group	% contribution <sup>2,3</sup>	mg/day
Fruit	22.6	19.7
Soft drinks <sup>4</sup>	18.0	14.5
Fruit juice and smoothies	14.1	15.5
Vegetables, vegetable products and dishes	9.5	7.4
Dietary supplements	9.0	12.2
Milk <sup>5</sup>	7.2	4.5
Potatoes, potato products and dishes	6.8	4.5
Meat, meat products and dishes	3.4	2.0
Breakfast cereals	2.1	1.6
Sugar and chocolate confectionery	1.4	1.2
Number of participants	102	102

<sup>1</sup> Data from NDNS years 2016 to 2019.

<sup>2</sup> Food groups that contribute less than 1% of intake are not presented.

<sup>3</sup> Average % contribution for each food group has been calculated from the % contribution for each individual. Non-consumers are included in the average.

<sup>4</sup> Fizzy drinks, squashes and ready-to-drink still drinks, both those with added sugar and diet types.

<sup>5</sup> Includes non-dairy alternatives.

- 4.270 In both age groups, fruit and fruit juice contributed around 40% to vitamin C intake. Soft drinks contributed another 17 to 18% to vitamin C intake, which is of potential concern given the association between free sugars' intake and the development of dental caries, as well as excess weight gain (SACN, 2015).
- 4.271 Dietary supplements contributed a further 6.5% and 9% to vitamin C intake in the 18 to 47 months age group and 48 to 60 months age group, respectively.

# 5 Foods, dietary patterns, and dietary components

## Background

- 5.1 Dietary or nutritional exposures have traditionally been examined by investigating intakes of single nutrients or consumption of individual foods or food groups. Since the early 2000s, dietary pattern analysis, which considers the whole diet, has gained popularity as a promising alternative in nutrition research because it can take into account relationships between individual foods, food groups and nutrients which cannot be captured by studying single dietary components (Gherasim et al, 2020; Jacobs & Tapsell, 2007; Jannasch et al, 2021; Ocke, 2013).
- 5.2 This chapter is divided into 3 parts. Consideration is first given to the major food groups consumed by children aged 1 to 5 years in the UK followed by systematic review (SR) evidence identified on dietary patterns and dietary components (that is, low or no calorie sweeteners and probiotics) in this age group.

## Foods

- 5.3 The UK government dietary advice for the whole population (currently aged 5 years and older) is encapsulated in the national food model, [the Eatwell Guide](#). This shows the proportions in which different types of foods should be consumed and is underpinned by current UK dietary recommendations.
- 5.4 This section of the report is divided into the main food groups of the Eatwell Guide:
- vegetables and fruit
  - dairy products (excluding milk and dairy alternative or plant-based drinks)
  - foods rich in starchy carbohydrates (for example, bread, rice, pasta)
  - non-dairy sources of protein (for example, beans, pulses, fish, eggs and meat).
- 5.5 This section also includes a subsection on foods (and drinks) that are energy dense and high in (saturated) fat, salt or (free) sugars. These products are not needed in the diet. It is recommended that if these foods and drinks are consumed, it should be done infrequently and in small amounts (PHE, 2018).
- 5.6 This section also covers the contribution of commercially manufactured foods and drinks (excluding [formula milks](#)) that are marketed specifically for infants and young children to young children's diets, and summarises the key conclusions and

recommendations from the joint 2017 SACN-COT statement [‘Assessing the health benefits and risks of the introduction of peanut and hen’s egg into the infant diet before six months of age in the UK’](#). Details can be found in SACN’s report ‘Feeding in the first year of life’ (SACN, 2018).

## **Limitations of the systematic review evidence on foods**

- 5.7 While some evidence from SRs was identified on the health impact of vegetables and fruit, dairy products (milk and formula milk is covered separately in chapter 6) and foods that are energy dense and high in saturated fats, salt or free sugars, no SR evidence was identified on the health impact of any other foods and food groups for children aged 1 to 5 years (for example, foods rich in starchy carbohydrates or non-dairy protein sources).
- 5.8 Many of the primary studies included in the SRs did not adjust for key potential confounding factors or mediators or effect modifiers that are important to consider when studying the health impact of individual foods or food groups. These include age, sex and socioeconomic status (SES).
- 5.9 Primary studies that examined outcomes relating to or resulting from effects on energy balance (for example, body mass index [BMI]) did not always adequately account for children’s body size at baseline. A child who is larger at baseline may consume more food and drink (and more energy overall) than a smaller child. Therefore, the possibility of reverse causation, where body size drives food and drink consumption rather than the other way around, cannot be ruled out.
- 5.10 Primary studies that examined specific foods also did not always adjust for total dietary energy intake (TDEI) (see chapter 3, paragraphs 3.48 and 3.49) or intake of other foods (Jacobs & Tapsell, 2007).
- 5.11 Primary studies that examined cognition-related outcomes did not always adjust for child baseline cognition and parental cognition.

## **Vegetables and fruit**

- 5.12 The current UK recommendation is that between the ages of 2 and 5 years, children should gradually move towards eating the same foods as the rest of the family, including aiming to eat at least 5 portions of vegetables and fruit every day (NHS, 2019a). However, there is a lack of agreed portion sizes for vegetables and fruit for this age group in the UK.

## Vegetables (excluding potatoes) and fruit (excluding juice) consumption in the UK

- 5.13 Data from the Diet and Nutrition Survey of Infants and Young Children (DNSIYC) and the National Diet and Nutrition Survey (NDNS rolling programme years 2016 to 2019) on the consumption and contribution of vegetables (excluding potatoes) and fruit (excluding juice) to the diets of children aged 12 to 60 months in the UK are presented in Table 5.1 to 5.3.
- 5.14 Nearly all children in all age groups consumed vegetables or fruit or both over the 4 day survey period. Therefore, average values in consumers were similar to the average values at a population level (which includes non-consumers).
- 5.15 Children aged 12 to 18 months consumed, on average, 170 grams per day of vegetables (excluding potatoes) and fruit (excluding fruit juice). For children aged 18 to 47 months, and aged 48 to 60 months, consumption was, on average, 178 grams per day and 217 grams per day, respectively.
- 5.16 In all age groups, fruit consumption was higher than vegetable consumption.
- 5.17 It was not possible to establish whether the vegetables and fruit consumed were 'processed', where 'processed' is understood as vegetables and fruit that have been blended, pulped, pureed, extruded or powdered (Swan et al, 2018).

**Table 5.1 Vegetables (excluding potatoes) and fruit (excluding juice) consumption (grams per day and % consumers) in children aged 12 to 18 months in the UK (DNSIYC)<sup>1</sup>**

Food Group	Mean consumption (SD) in grams per day Includes non-consumers <sup>5</sup>	% (number) of consumers over 4 days	Mean consumption (SD) in grams per day Consumers only
Total vegetables <sup>2</sup> (excluding potatoes and juice)	74 (46)	99 (1269)	74 (46)
Total fruit <sup>3</sup> (excluding juice)	96 (69)	99 (1261)	97 (68)
Total vegetables (excluding potatoes) and fruit <sup>4</sup> (excluding juice)	170 (92)	100 (1275)	170 (92)

Abbreviations: SD, standard deviation

<sup>1</sup> Data from DNSIYC 2011 (Lennox et al, 2013).

<sup>2</sup> Includes vegetables in composite dishes and manufactured products.

<sup>3</sup> Includes fruit in composite dishes and manufactured products.

<sup>4</sup> Includes vegetables and fruit in composite dishes and manufactured products.

<sup>5</sup> Number of participants: 1275

**Table 5.2 Vegetables and fruit (excluding juice) consumption (grams per day) in children aged 18 to 47 months in the UK (NDNS years 2016 to 2019)<sup>1</sup>**

Food Group	Mean consumption (SD) in grams per day Includes non-consumers <sup>5</sup>	% (number) of consumers over 4 days	Mean consumption (SD) in grams per day consumers only
Total vegetables <sup>2</sup>	68 (43)	97 (297)	70 (42)
Total fruit (excluding juice) <sup>3</sup>	110 (74)	99 (301)	112 (74)
Total vegetables and fruit (excluding fruit juice) <sup>4</sup>	178 (98)	100 (306)	178 (98)

Abbreviations: SD, standard deviation

<sup>1</sup> Data from NDNS 2016 to 2019.

<sup>2</sup> Includes vegetables in composite dishes and manufactured products.

<sup>3</sup> Includes fruit in composite dishes and manufactured products.

<sup>4</sup> Includes vegetables and fruit in composite dishes and manufactured products.

<sup>5</sup> Number of participants: 306

**Table 5.3 Vegetables and fruit (excluding juice) consumption (grams per day and contribution to TDEI) in children aged 48 to 60 months in the UK (NDNS years 2016 to 2019)<sup>1</sup>**

Food Group	Grams per day Mean (SD) including non-consumers <sup>5</sup>	% (number) of consumers over 4 days	Grams per day Mean (SD) consumers only
Total vegetables <sup>2</sup>	87 (56)	99 (101)	88 (55)
Total fruit (excluding juice) <sup>3</sup>	131 (93)	100 (101)	131 (93)
Total vegetables and fruit (excluding fruit juice) <sup>4</sup>	217 (117)	100 (102)	217 (117)

Abbreviations: SD, standard deviation

<sup>1</sup> Data from NDNS 2016 to 2019.

<sup>2</sup> Includes vegetables in composite dishes and manufactured products.

<sup>3</sup> Includes fruit in composite dishes and manufactured products.

<sup>4</sup> Includes vegetables and fruit in composite dishes and manufactured products.

<sup>5</sup> Number of participants: 102

- 5.18 Time trend analysis of NDNS data (years 2008 to 2017) in children aged 18 to 36 months indicated a significant decrease in mean consumption of vegetables (average change per year  $-1.7\text{g}$ ; 95% CI  $-2.8\text{g}$  to  $-0.6\text{g}$ ) for the 9-year period but no significant change in mean consumption of fruit (excluding fruit juice) (average change per year  $-0.7\text{g}$ ; 95% CI  $-2.7\text{g}$  to  $1.3\text{g}$ ) or total vegetables or fruit (average

change per year  $-2.4\text{g}$ ; 95% CI  $-5.1$  to  $0.3\text{g}$ ) (Bates et al, 2019). No time trend data was available for the other age groups.

## Vegetables and fruit consumption by deprivation

5.19 Consumption of vegetables (excluding potatoes) and fruit by Index of Multiple Deprivation (IMD) (see Glossary) in children aged 18 to 60 months (including non-consumers) is presented in Table 5.4.

**Table 5.4 Vegetables and fruit consumption by IMD quintile in children aged 18 to 60 months in England (NDNS years 2008 to 2019)<sup>1</sup>.**

Food group	IMD quintile 1 (least deprived)	IMD quintile 2	IMD quintile 3	IMD quintile 4	IMD quintile 5 (most deprived)
Vegetables (excluding juice) <sup>2</sup> mean (95%CI) (grams per day)	82 (75 to 88)	80 (73 to 86)	73 (67 to 80)	72 (66 to 79)	64 (59 to 69)
Fruit (excluding juice) <sup>3</sup> mean (95%CI) (grams per day)	133 (122 to 144)	125 (114 to 136)	118 (107 to 130)	111 (101 to 120)	93 (85 to 102)
Number of participants	210	212	182	234	277

Abbreviations: IMD, index of multiple deprivation.

<sup>1</sup> Data from NDNS years 2008 to 2019. Includes non-consumers.

<sup>2</sup> includes vegetables in composite dishes and manufactured products.

<sup>3</sup> Includes fruit in composite dishes and manufactured products.

5.20 Vegetable consumption was lowest (64 grams per day) in quintile 5 (most deprived) and highest (82 grams per day) in quintile 1 (least deprived). Vegetable consumption was significantly higher in quintiles 1 and 2 than in quintile 5 (as indicated by non-overlapping confidence intervals).

5.21 Fruit consumption was lowest (93 grams per day) in quintile 5 (most deprived) and highest (133 grams per day) in quintile 1 (least deprived). Fruit consumption was significantly higher in quintile 1 than in quintiles 4 and 5 (as indicated by non-overlapping confidence intervals).

## **Systematic review evidence identified on vegetable and fruit (excluding juice) consumption and health**

- 5.22 One SR without meta-analysis (MA) (Ledoux et al, 2011) was identified that included studies that examined the health impact of vegetables and fruit (excluding juice) consumption. SR evidence on [fruit juice](#) is covered in chapter 6 ('Drinks').
- 5.23 Key outcomes examined by the SR were measures of body composition or weight status (BMI, body weight, body fat).
- 5.24 Vegetables and fruit may have different health effects depending on how they are presented or prepared (for example, puréed, mashed, chopped). SACN advised that the sugars naturally present in fruit and vegetables that have been blended, pulped, puréed, extruded or powdered should be treated as free sugars on the basis that the cellular structure has been broken down (Swan et al, 2018). However, Ledoux et al (2011) did not specify how vegetables and fruit were prepared or presented to study participants.
- 5.25 Details of the SR included in this section can be found in Annex 5 (Tables A5.3). Quality assessment of the SR using the AMSTAR 2 tool can be found in Annex 8 (Tables A8.4). Additional data extracted on the primary studies can be found in Annex 9 (Table A9.23).
- 5.26 The criteria used to grade the evidence are provided in chapter 2 (Table 2.4, paragraphs 2.54 to 2.59).
- 5.27 All primary studies included in the SR were conducted in high-income countries (HIC) (defined according to the [World Bank classification system](#)).

## **Vegetable and fruit (excluding juice) consumption and body composition**

### **Vegetable and fruit (excluding juice) consumption and BMI or body weight**

- 5.28 One SR without MA (Ledoux et al, 2011) (AMSTAR 2 confidence rating: critically low) was identified that examined the relationship between vegetable and fruit (excluding juice) consumption and obesity outcomes. The SR included 2 PCS that examined this relationship in children aged 1 to 5 years.
- 5.29 One PCS (in 1379 participants) reported that each additional serving of vegetables in children between ages 2 and 5 years was associated with a 0.09kg (95% CI 0.05 to 0.13; p=0.02) greater weight gain per year (follow up 6 to 12 months) after adjusting for age, sex, SES and ethnicity. However, the relationship no longer held

when the analysis was additionally adjusted for consumption of all other food groups (quantitative findings NR). This PCS also did not report the vegetable and fruit classification used in its analysis.

- 5.30 The second PCS (in 972 participants) reported no association between vegetable and fruit consumption and BMI z-score in children aged 1 to 5 years from low-income families after 2 years' follow up, adjusted for SES and ethnicity (quantitative findings NR). The exposure did not include juice, carrots, potatoes and salads.

### **Vegetable and fruit (excluding juice) consumption and body fat**

- 5.31 No evidence was identified from SRs on the relationship between vegetables and fruit (excluding juice) consumption and body fat in children aged 1 to 5 years.

### **Summary: vegetable and fruit (excluding juice) consumption and body composition**

- 5.32 The evidence identified from SRs on whole vegetable and fruit consumption in children aged 1 to 5 years and body composition is summarised in Table 5.5.

**Table 5.5 Summary of the evidence on vegetables and fruit consumption and obesity outcomes**

Exposure	Outcome	Direction of association	Certainty of evidence
Vegetables and fruit (excluding juice)	Body Mass Index or body weight	Not applicable	Insufficient
Vegetables and fruit (excluding juice)	Body fat	Not applicable	No systematic review evidence identified

- 5.33 The available evidence from SRs on the relationship between vegetable and fruit (excluding juice) consumption and body composition in children aged 1 to 5 years is from 1 SR without MA given a critically low confidence rating using the AMSTAR 2 tool.
- 5.34 There was insufficient evidence from SRs to enable conclusions to be drawn on any relationship between vegetable and fruit (excluding juice) consumption and BMI or body weight in children aged 1 to 5 years as fewer than 3 primary studies included in the SR examined this relationship.

## Dairy products

- 5.35 The terms 'cheese', 'yoghurt' and 'cream' are protected terms reserved exclusively for dairy products (which includes milk or any milk-based product) (Dougkas et al, 2019).
- 5.36 This section covers dairy products (cheese, yoghurt, fromage frais) excluding formula milks, cows' milk and other dairy milks (for these, see chapter 6). However, in the subsection [Total dairy consumption in the UK](#), dietary survey data on the consumption of cows' milk and other dairy milks in the UK have been included.
- 5.37 The current UK recommendation is that children can be given pasteurised full-fat cheeses and dairy products from age 6 months. Full-fat dairy products are recommended up to the age of 2 years, after which it is recommended to introduce lower fat dairy products (NHS, 2023c).

### Total dairy consumption (excluding formula milks) in the UK

- 5.38 Data from DNSIYC and NDNS (years 2016 to 2019) on the consumption and contribution of dairy products (including cows' milk and other dairy milks but excluding formula milks) to the diets of children aged 12 to 60 months in the UK are presented in Table 5.6 to 5.8. Values that include non-consumers provide an estimate of the overall contribution of dairy products to the diets of young children, while values for consumers provide an estimate of the quantities consumed.
- 5.39 Nearly all children consumed dairy products over the 4 day survey period. Therefore, average values in consumers were similar to the average values at a population level (which includes non-consumers).
- 5.40 Dairy products (excluding formula milks) contributed approximately 27% TDEI, 22% TDEI and 15% TDEI in children aged 12 to 18 months, 18 to 47 months, and 48 to 60 months, respectively. Of the main dairy products examined, cows' milk and other dairy milks were the largest contributors to TDEI in all age groups.

**Table 5.6 Dairy consumption (grams per day and contribution to TDEI) in children aged 12 to 18 months in the UK (DNSIYC)<sup>1</sup>**

Food Group	Mean consumption (SD) in grams per day Includes non-consumers	% Contribution to TDEI Includes non-consumers	Percentage (number) consumers over 4 days	Mean consumption (SD) in grams per day Consumers only	% Contribution to TDEI Consumers only
Total milk <sup>2</sup>	289 (236)	19.0	90 (1149)	322 (228)	21.2
Yoghurt and fromage frais <sup>5</sup>	48 (40)	4.8	85 (1072)	57 (37)	5.6
Cheese <sup>4</sup>	8 (10)	2.7	71 (900)	11 (10)	3.8
Cream and other milk products <sup>3</sup>	0.4 (2.2)	0.1	10 (122)	4 (6)	1.2
Total dairy <sup>6</sup>	345 (240)	26.6	98 (1247)	353 (237)	27.2

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake.

<sup>1</sup> Data from DNSIYC 2011 (Lennox et al, 2013). Number of participants: 1275

<sup>2</sup> Total milk includes: all types of cows' milk and other dairy milk.

<sup>3</sup> Cream and other milk products includes 1% and skimmed milk. All types of dairy cream, dairy toppings, crème fraîche

<sup>4</sup> Cheese excludes cheese in manufactured products and homemade recipe dishes

<sup>5</sup> Yoghurt and fromage frais includes unsweetened and sugar-sweetened products.

<sup>6</sup> Total dairy: total of milk, cream, cheese, yoghurt and fromage frais.

**Table 5.7 Dairy consumption (grams per day and contribution to TDEI) in children aged 18 to 47 months in the UK (NDNS years 2016 to 2019)<sup>1</sup>**

Food Group	Mean consumption (SD) in grams per day Includes non-consumers	% Contribution to TDEI Includes non-consumers	% (number) of consumers over 4 days	Mean consumption (SD) in grams per day Consumers only	% Contribution to TDEI Consumers only
Total milk <sup>2</sup>	261 (204)	14.8	92 (283)	283 (197)	16.1
Cheese <sup>4</sup>	10 (12)	3.3	76 (228)	14 (13)	4.4
Yoghurt and fromage frais <sup>5</sup>	39 (45)	3.2	71 (218)	54 (45)	4.5
Cream and other milk products <sup>3</sup>	1 (2)	0.2	17 (58)	4 (4)	0.9
Total dairy <sup>6</sup>	311 (213)	21.5	96 (295)	322 (208)	22.3

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake.

<sup>1</sup> Data from NDNS 2016 to 2019. Number of participants: 306

<sup>2</sup> Total milk includes: all types of cows' milk and other dairy milk.

<sup>3</sup> Cream and other milk products: All types of dairy cream, dairy toppings, crème fraîche.

<sup>4</sup> Cheese excludes cheese in manufactured products and homemade recipe dishes

<sup>5</sup> Yoghurt and fromage frais – includes unsweetened and sugar-sweetened products.

<sup>6</sup>Total dairy: total of milk, cream and other milk products, cheese, yoghurt and fromage frais.

**Table 5.8 Dairy consumption (grams per day and contribution to TDEI) in children aged 48 to 60 months in the UK (DNSIYC and NDNS years 2016 to 2019)<sup>1</sup>**

Food Group	Mean consumption (SD) in grams per day Includes non-consumers	% Contribution to TDEI Includes non-consumers	% (number) of consumers over 4 days	Mean consumption (SD) in grams per day Consumers only	% Contribution to TDEI Consumers only
Total milk <sup>2</sup>	229 (214)	9.6	93 (96)	247 (212)	10.4
Cheese <sup>4</sup>	9 (11)	2.4	69 (72)	13 (11)	3.5
Yoghurt and fromage frais <sup>5</sup>	33 (38)	2.3	64 (71)	51 (36)	3.6
Cream and other milk products <sup>3</sup>	1 (4)	0.3	16 (18)	No data <30 consumers	No data <30 consumers
Total dairy <sup>6</sup>	271 (216)	14.7	97 (99)	281 (214)	15.2

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake.

<sup>1</sup> Data from NDNS 2016 to 2019. Number of participants: 102

<sup>2</sup> Total milk: All types of cows' milk and other dairy milk.

<sup>3</sup> Cream and other milk products: All types of dairy cream, dairy toppings, crème fraîche.

<sup>4</sup> Cheese excludes cheese in manufactured products and homemade recipe dishes

<sup>5</sup> Yoghurt and fromage frais – includes unsweetened and sugar-sweetened products.

<sup>6</sup> Total dairy: total of milk, cream, cheese, yoghurt and fromage frais.

## Total dairy consumption and deprivation

- 5.41 Total dairy consumption by IMD (see Glossary) in children aged 18 to 60 months (including non-consumers) is presented in Table 5.9.
- 5.42 Total dairy consumption was highest (342 grams per day) in quintile 1 (least deprived) and lowest in quintiles 4 and 5 (most deprived). Total dairy consumption was significantly higher in quintile 1 than quintiles 4 and 5 (as indicated by non-overlapping confidence intervals).

**Table 5.9 Total dairy consumption by IMD quintile in children aged 18 to 60 months in England (NDNS years 2008 to 2019)<sup>1</sup>**

Consumption Mean (95%CI)	IMD quintile 1 (least deprived)	IMD quintile 2	IMD quintile 3	IMD quintile 4	IMD quintile 5 (most deprived)
Total dairy <sup>1</sup> (grams per day)	342 (310 to 373)	323 (296 to 351)	322 (294 to 350)	281 (255 to 307)	282 (258 to 306)
Number of participants	210	212	182	234	277

Abbreviations: IMD, index of multiple deprivation.

<sup>1</sup> Data from NDNS years 2008 to 2019. Includes non-consumers.

<sup>2</sup> Total of milk, cream, cheese, yoghurt and fromage frais

## Systematic review evidence identified on dairy products and health

- 5.43 One SR with MA (de Beer, 2012) and 3 SRs without MA (Dougkas et al, 2019; Dror & Allen, 2014; Tandon et al, 2016) were identified that included studies that examined the health impact of consuming dairy products (including milk).
- 5.44 Exposures were total dairy consumption (including milk) and consumption of individual dairy products (yoghurt, cheese, cream or crème fraiche). No distinction was made between dairy products that were sweetened (with free sugars or low or no calorie sweeteners), unsweetened or were plain in flavour.
- 5.45 Key outcomes examined were measures of body composition (BMI, body weight, body fat), linear growth (height), bone health, blood pressure and cognitive development.
- 5.46 All primary studies included in the SRs were conducted in HIC.
- 5.47 Details of the 4 SRs included in this section can be found in Annex 5 (Table A5.3). Quality assessment of the SRs using the AMSTAR 2 tool can be found in Annex 8 (Tables A8.4 and A8.5). Additional data extracted on the primary studies can be found in Annex 9 (Table A9.24).
- 5.48 The criteria used to grade the evidence are provided in chapter 2 (Table 2.4, paragraphs 2.54 to 2.59). Summary tables of the evidence grading process for this section are provided in Annex 10 (Tables A10.10 and A10.36).

## **Total dairy consumption and body composition or linear growth**

### **BMI and body fat**

- 5.49 One SR without MA (Dougkas et al, 2019) (AMSTAR 2 confidence rating: low) examined the relationship between total dairy consumption and BMI or body fat and included 4 PCS (of which 2 were in the same cohort of children) that examined this relationship in children aged 1 to 5 years. Of the 4 PCS, 3 reported an inverse association between total dairy consumption and BMI or body fat and 1 PCS reported no association.
- 5.50 One PCS (in 53 participants) reported that higher average consumption of dairy products (servings per day) at age 2 years was associated with a lower % body fat (beta coefficient  $-3.54\%$ ; SE 1.04;  $p=0.001$ ) and body fat (g) (beta coefficient  $-907.06\text{g}$ ; SE 284.06;  $p=0.003$ ) after 6 years' follow up compared with children with a lower average consumption of dairy products at age 2 years. The analyses were adjusted for sex, BMI, and intakes of calcium, protein, carbohydrates and fat.
- 5.51 One PCS (in 92 participants) reported that 'low' dairy product consumption ( $<1.75$  servings/day) in children aged 3 to 6 years compared with 'high' dairy product consumption ( $>1.75$  servings per day) was associated with greater subcutaneous fat (25mm; 95% CI NR;  $p=0.005$ ) and higher BMI (2 units; 95% CI NR;  $p=0.046$ ) in early adolescence (8 years of follow-up). The analyses were adjusted for age, physical activity, maternal education, baseline anthropometric measures, saturated fat intake and TDEI.
- 5.52 One PCS (in 362 participants) reported that greater consumption of dairy products as % TDEI at age 18 months was associated with a decrease in BMI (beta coefficient  $-0.21 \text{ kg/m}^2$ ; 95% CI  $-0.41$  to  $0.01 \text{ kg/m}^2$ ;  $p=0.04$ ) at age 8 years, compared with protein, meat and fruit consumption. The analysis was adjusted for sex, birth weight, parental obesity status, ethnicity, smoking in pregnancy, paternal education.
- 5.53 Another PCS (in 335 participants) in the same cohort of children as the study described in paragraph 5.52 reported no association between energy-adjusted dairy product consumption at age 18 months and BMI assessed at age 8 years (estimate of association NR; 95% CI NR;  $p=0.09$ ). However, the analysis was unadjusted.

### **Linear growth**

- 5.54 One SR without MA (Dror & Allen, 2014) (AMSTAR 2 confidence rating: critically low) examined the relationship between total dairy consumption and linear growth and included 1 PCS that examined this relationship in children aged 1 to 5 years. The PCS (in 335 participants) reported no difference in height at age 8 years

between quintiles of energy-adjusted dairy consumption at age 1.5 years (quantitative findings NR). The analysis was adjusted for child's age, sex, measures of SES, baseline weight status and TDEI.

## Summary: total dairy consumption and body composition or linear growth

- 5.55 The evidence identified from SRs on total dairy consumption and body composition (BMI or body fat) or linear growth in children aged 1 to 5 years is summarised in Table 5.10.

**Table 5.10 Summary of the evidence on total dairy consumption and body composition or linear growth**

Exposure	Outcome	Direction of association	Certainty of evidence
Total dairy consumption	Body fat (% or grams)	Not applicable	Insufficient
Total dairy consumption	Body Mass Index	Not applicable	Insufficient
Total dairy consumption	Linear growth	Not applicable	Insufficient

- 5.56 The available evidence from SRs on the relationship between total dairy consumption and body composition or linear growth in children aged 1 to 5 years is from 1 SR given a critically low confidence rating using the AMSTAR 2 tool.
- 5.57 Although there were 3 PCS that examined the association between total dairy consumption and BMI, 2 of the 3 studies used a dataset from the same longitudinal cohort study. Because there were only 2 independent PCS, the evidence was graded insufficient.
- 5.58 There was insufficient evidence from SRs to enable conclusions to be drawn on any relationship between total dairy consumption and body fat or linear growth in children 1 to 5 years as fewer than 3 primary studies included in the SR examined these relationships.

## Total dairy consumption and other health outcomes

### Bone health

- 5.59 One SR without MA (Dror & Allen, 2014) (AMSTAR 2 confidence rating: critically low) was identified that examined the relationship between total dairy consumption and bone health and included 1 PCS that examined this relationship in children aged 1 to 5 years. The PCS (in 106 participants) reported that consumption of 2 or

more servings of dairy products per day from ages 3 to 5 years was associated with a higher total body bone mineral content (grams) (estimate of association NR; 95% CI NR; p=0.009) and bone area (cm<sup>2</sup>) (estimate of association NR; 95% CI NR; p=0.02) at ages 15 to 17 years compared with consumption of less than 2 servings of dairy per day. Analyses were adjusted for sex, physical activity, age, height, BMI, and % body fat (from dual-energy x-ray absorptiometry) at the time of the bone scan.

### Summary: total dairy consumption and bone health

5.60 The evidence identified from SRs on total dairy consumption and bone health in children aged 1 to 5 years is summarised in Table 5.11.

**Table 5.11 Summary of the evidence on dairy consumption and bone health**

Exposure	Outcome	Direction of association	Certainty of evidence
Total dairy consumption	Bone mineral content (grams)	Not applicable	Insufficient
Total dairy consumption	Bone area (cm)	Not applicable	Insufficient

5.61 The available evidence from SRs on the relationship between total dairy consumption and bone health in children aged 1 to 5 years is from 1 SR given a critically low confidence rating using the AMSTAR 2 tool.

5.62 There was 'insufficient' evidence from SRs to enable conclusions to be drawn on any relationship between total dairy consumption and bone health in children aged 1 to 5 years as fewer than 3 primary studies included in the SR examined this outcome.

### Blood pressure

5.63 One SR without MA (Dror & Allen, 2014) (AMSTAR 2 confidence rating: critically low) was identified that examined the relationship between total dairy consumption and blood pressure and included 2 PCS in children aged 1 to 5 years. Both PCS (in a total of 430 participants) reported that higher dairy consumption in children aged 1 to 5 years was associated with lower blood pressure in childhood and adolescence.

5.64 One of the PCS (in 335 participants) reported that children in the highest quintile of energy-adjusted dairy consumption at age 1.5 years had a lower systolic blood pressure (SBP) and diastolic blood pressure (DBP) at age 8 years compared with the lowest quintile (estimate of association NR; both p<0.05), adjusted for age,

sex, SES, baseline weight status, maternal smoking status during pregnancy, TDEI.

5.65 The second PCS (in 95 participants) reported that children who consumed >2 servings per day of dairy at ages 3 to 6 years experienced smaller annual gains in SBP from ages 3 to 13 years compared with children who consumed <2 servings per day of dairy (beta coefficient 2.90 (SE 0.18) compared with beta coefficient 2.21 (SE 0.24) mmHg per year; 95% CI NR; p-value NR). However, the PCS reported no difference in gains in DBP between the groups (quantitative findings NR). Analyses were adjusted for baseline blood pressure, physical activity, intakes of magnesium and sodium per day at age 3 to 6 years and change in BMI from ages 3 to 12 years.

### Summary: total dairy consumption and blood pressure

5.66 The evidence identified from SRs on total dairy consumption and blood pressure in children aged 1 to 5 years is summarised in Table 5.12.

**Table 5.12 Summary of the evidence on total dairy consumption and blood pressure**

Exposure	Outcome	Direction of association	Certainty of evidence
Total dairy consumption	Systolic blood pressure	Not applicable	Insufficient
Total dairy consumption	Diastolic blood pressure	Not applicable	Insufficient

5.67 The available evidence from SRs on the relationship between total dairy consumption and blood pressure in children aged 1 to 5 years is from 1 SR given a critically low confidence rating using the AMSTAR 2 tool.

5.68 There was 'insufficient' evidence from SRs to enable conclusions to be drawn on any relationship between dairy consumption and blood pressure in children aged 1 to 5 years as fewer than 3 primary studies included in SRs examined this relationship.

### Cognitive outcomes

5.69 One SR without MA (Tandon et al, 2016) (AMSTAR 2 confidence rating: critically low) was identified that examined the relationship between total dairy consumption and cognitive ability and included 1 PCS in children aged 1 to 5 years. The PCS (in 1346 participants) reported that greater dairy consumption at ages 2 and 3 years was associated with better verbal cognitive outcomes at age 10 years (quantitative findings NR). The analysis was adjusted for sex, maternal age, maternal education, family income, a father living with family, reading to the child,

maternal Bradburn Negative Affect score (maternal mental health distress) and breastfeeding duration.

### **Summary: total dairy consumption and cognitive outcomes**

- 5.70 The evidence identified from SRs on total dairy consumption and cognitive ability in children aged 1 to 5 years is summarised in Table 5.13.

**Table 5.13 Summary of the evidence on total dairy consumption and cognitive outcomes**

Exposure	Outcome	Direction of association	Certainty of evidence
Total dairy consumption	Verbal cognitive outcomes	Not applicable	Insufficient

- 5.71 The available evidence from SRs on the relationship between dairy consumption and cognitive outcomes in children aged 1 to 5 years is from 1 SR given a critically low confidence rating using the AMSTAR 2 tool.
- 5.72 There was ‘insufficient’ evidence from SRs to enable conclusions to be drawn on any relationship between dairy consumption and cognitive ability in children aged 1 to 5 years as fewer than 3 primary studies included in SRs examined this relationship.

## **Individual dairy products and weight status or linear growth**

### **Weight status (overweight or obesity)**

- 5.73 One SR without MA (Dougkas et al, 2019) (AMSTAR 2 confidence rating: low) examined the relationship between consumption of individual dairy products and obesity outcomes and included 1 PCS in children aged 1 to 5 years. The PCS (in 14,224 participants) reported that higher consumption of cheese but lower consumption of cream or crème fraiche at age 2.5 years was associated with overweight or obesity at age 5 years (quantitative findings NR). The analyses were adjusted for parental education and parental BMI.

### **Linear growth**

- 5.74 One SR with MA (de Beer, 2012) (AMSTAR 2 confidence rating: critically low) examined the relationship between consumption of yoghurt and linear growth and included 1 RCT in children aged 1 to 5 years. As the MA conducted by de Beer (2012) pooled estimates from studies from other age groups, findings from the RCT are reported separately below.

- 5.75 The RCT (in 402 participants) reported that children (mean age 3.3 years at baseline) who received 125g of yoghurt for 5 days a week for 9 months experienced greater linear growth than children in the control group (no intervention) (MD 0.19cm; 95% CI 0.0481 to 0.3319cm; p<0.05).

## Summary: consumption of individual dairy products and weight status or linear growth

- 5.76 The evidence identified from SRs on consumption of individual dairy products and weight status or linear growth in children aged 1 to 5 years is summarised in Table 5.14.

**Table 5.14 Summary of the evidence on consumption of individual dairy products and weight status or linear growth**

Exposure	Outcome	Direction of effect or association	Certainty of evidence
Cheese consumption	Overweight or obesity	Not applicable	Insufficient
Cream or crème fraiche consumption	Overweight or obesity	Not applicable	Insufficient
Yoghurt consumption	Linear growth	Not applicable	Insufficient

- 5.77 The available evidence from SRs on the relationship between consumption of individual dairy products and weight status or linear growth in children aged 1 to 5 years is from 2 SRs, 1 given a low confidence rating using the AMSTAR 2 tool, the other given a critically low confidence rating.
- 5.78 There was 'insufficient' evidence from SRs to enable conclusions to be drawn on any relationship between consumption of individual dairy products and weight status or linear growth in children aged 1 to 5 years as fewer than 3 primary studies included in the SRs examined these relationships.

## Foods rich in starchy carbohydrates

- 5.79 Starchy foods, such as potatoes, bread, rice, pasta and breakfast cereals, are the main sources of carbohydrates in the UK diet for young children (see Carbohydrates in chapter 3).
- 5.80 Due to the lack of evidence on the health impact of starchy carbohydrates in its report '[Carbohydrates and health](#)', SACN made no quantitative recommendations regarding the amounts of starchy carbohydrates that should be consumed by children aged under 2 years. However, SACN did recommend that, from about age

6 months, gradual diversification of the diet is encouraged, including increasing amounts of wholegrains (SACN, 2015). It is also recommended that children under age 2 years should not consume exclusively wholegrain varieties of starchy carbohydrates as satiety could be achieved before adequate energy and nutrients are consumed (NHS, 2023c). For UK recommendations on carbohydrates (total carbohydrates, free sugars and dietary fibre), see [chapter 3](#).

## **Consumption of foods rich in starchy carbohydrates consumption in the UK**

- 5.81 Data on the consumption and contribution of foods rich in starchy carbohydrates to the diets of children aged 12 to 60 months from DNSIYC and NDNS (years 2016 to 2019) are presented in Table 5.15 to Table 5.17. Values that include non-consumers provide an estimate of the overall contribution of starchy carbohydrates to the diets of young children, while values for consumers provide an estimate of the quantities consumed.
- 5.82 Almost all children aged 12 to 60 months consumed foods rich in starchy carbohydrates over the 4 day survey period. Therefore, average values in consumers were similar to the average values at a population level (which includes non-consumers).
- 5.83 Foods rich in starchy carbohydrates contributed approximately 17% TDEI in children aged 12 to 18 months, and 21% TDEI in children aged 18 to 60 months. Of the main sources of starchy carbohydrates examined, bread made the largest contribution to TDEI followed by breakfast cereals (with a total sugars content less than 22.5 grams per 100g) in all age groups.

**Table 5.15 Consumption of foods rich in starchy carbohydrates (grams per day and contribution to TDEI) in children aged 12 to 18 months in the UK (DNSIYC)<sup>1</sup>**

Food Group	Mean consumption (SD) in grams per day Includes non-consumers	% Contribution to TDEI Includes non-consumers	% (number) of consumers over 4 days	Mean consumption (SD) in grams per day Consumers only	% Contribution to TDEI Consumers only
Bread <sup>2</sup>	24 (20)	6.0	87 (1110)	27 (19)	6.9
Breakfast cereals sugar <22.5g per 100g <sup>6</sup>	17 (27)	4.6	77 (989)	22 (29)	6.0
Pasta <sup>3</sup>	22 (30)	2.8	69 (884)	32 (31)	4.0
Potatoes <sup>5</sup>	23 (29)	1.9	74 (952)	31 (29)	2.6
Rice <sup>4</sup>	10 (22)	1.4	39 (473)	25 (30)	3.6
Total foods rich in starchy carbohydrates <sup>7</sup>	95 (57)	16.7	99 (1269)	95 (57)	16.8

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake

<sup>1</sup> Data from DNSIYC, 2011 (Lennox et al, 2013). Number of participants: 1275

<sup>2</sup> All types of wheat and non-wheat bread and rolls.

<sup>3</sup> Plain and filled pasta. Homemade pasta dishes and pasta based products and ready meals. Excludes meat based dishes including pasta.

<sup>4</sup> Plain rice, homemade rice based dishes and rice based products.

<sup>5</sup> Boiled, mashed, baked potatoes; homemade potato based dishes, instant, canned potatoes. Excludes chips, fried potatoes and fried potato products.

<sup>6</sup> All types of breakfast cereals with total sugars <22.5g per 100g (green or amber).

<sup>7</sup> Starchy carbohydrates: sum of bread, pasta, rice, potatoes, breakfast cereals green or amber for sugar

**Table 5.16 Consumption of foods rich in starchy carbohydrates (grams per day and contribution to TDEI) in children aged 18 to 47 months in the UK (NDNS years 2016 to 2019)<sup>1</sup>**

Food Group	Mean consumption (SD) in grams per day Includes non-consumers	% Contribution to TDEI Includes non-consumers	% (number) of consumers over 4 days	Mean consumption (SD) in grams per day Consumers only	% Contribution to TDEI Consumers only
Bread <sup>2</sup>	41 (29)	10.0	95 (290)	43 (28)	10.5
Breakfast cereals sugar <22.5g per 100g <sup>6</sup>	14 (22)	4.1	69 (224)	20 (25)	5.9
Pasta <sup>3</sup>	25 (33)	3.0	66 (207)	38 (34)	4.5
Potatoes <sup>5</sup>	25 (27)	1.9	74 (232)	33 (27)	2.6
Rice <sup>4</sup>	12 (26)	1.7	44 (141)	28 (32)	3.8
All foods rich in starchy carbohydrates <sup>7</sup>	117 (57)	20.7	100 (306)	117 (57)	20.7

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake

<sup>1</sup> Data from NDNS years 2016 to 2019. Number of participants: 306

<sup>2</sup> All types of wheat and non-wheat bread and rolls.

<sup>3</sup> Plain and filled pasta. Homemade pasta dishes and pasta based products and ready meals. Excludes meat based dishes including pasta.

<sup>4</sup> Plain rice, homemade rice based dishes and rice based products.

<sup>5</sup> Boiled, mashed, baked potatoes; homemade potato based dishes, instant, canned potatoes. Excludes chips, fried potatoes and fried potato products.

<sup>6</sup> All types of breakfast cereals with total sugars <22.5g per 100g (green or amber).

<sup>7</sup> Starchy carbohydrates: sum of bread, pasta, rice, potatoes, breakfast cereals green or amber for sugar

**Table 5.17 Consumption of foods rich in starchy carbohydrates (grams per day and contribution to TDEI) in children aged 48 to 60 months in the UK (NDNS years 2016 to 2019)<sup>1</sup>**

Food Group	Grams per day Mean (SD) including non-consumers	% Contribution to TDEI including non-consumers	% (number) of consumers over 4 days	Grams per day Mean (SD) in consumers only	% Contribution to TDEI for consumers only
Bread <sup>2</sup>	47 (30)	10.0	95 (97)	49 (28)	10.5
Breakfast cereals sugar <22.5g per 100g <sup>6</sup>	14 (15)	4.0	73 (74)	19 (14)	5.5
Pasta <sup>3</sup>	23 (28)	2.6	68 (71)	34 (27)	3.8
Rice <sup>4</sup>	16 (28)	2.0	48 (40)	[34 (32)]	[4.1]
Potatoes <sup>5</sup>	25 (31)	1.7	65 (69)	39 (30)	2.6
All foods rich in starchy carbohydrates <sup>7</sup>	126 (65)	20.3	100 (102)	126 (65)	20.3

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake

<sup>1</sup> Data from NDNS years 2016 to 2019. Number of participants; 102

<sup>2</sup> All types of wheat and non-wheat bread and rolls.

<sup>3</sup> Plain and filled pasta. Homemade pasta dishes and pasta based products and ready meals. Excludes meat based dishes including pasta.

<sup>4</sup> Plain rice, homemade rice based dishes and rice based products.

<sup>5</sup> Boiled, mashed, baked potatoes; homemade potato based dishes, instant, canned potatoes. Excludes chips, fried potatoes and fried potato products.

<sup>6</sup> All types of breakfast cereals with total sugars <22.5g per 100g (green or amber).

<sup>7</sup> Starchy carbohydrates: sum of bread, pasta, rice, potatoes, breakfast cereals green or amber for sugar

## Consumption of foods rich in starchy carbohydrates and deprivation

5.84 Consumption of foods rich in starchy carbohydrates by IMD (see Glossary) in children aged 18 to 60 months (including non-consumers) is presented in Table 5.18. The analysis indicated that there was no evidence of any relationship between consumption of starchy carbohydrates and IMD quintile (as indicated by overlapping confidence intervals).

**Table 5.18 Consumption of foods rich in starchy carbohydrates by IMD quintile in children aged 18 to 60 months in England (NDNS years 2008 to 2019)<sup>1</sup>**

Consumption Mean (95%CI)	IMD quintile 1 (least deprived)	IMD quintile 2	IMD quintile 3	IMD quintile 4	IMD quintile 5 (most deprived)
Foods rich in starchy carbohydrates <sup>2</sup> (grams per day)	118 (112 to 125)	120 (112 to 127)	121 (113 to 129)	121 (114 to 128)	117 (111 to 124)
Number of participants	210	212	182	234	277

Abbreviations: IMD, index of multiple deprivation.

<sup>1</sup> Data from NDNS years 2008 to 2019. Includes non-consumers.

<sup>2</sup> Starchy carbohydrates: sum of bread, pasta, rice, potatoes, breakfast cereals green or amber for sugar.

## Non-dairy sources of protein

- 5.85 Non-dairy sources of protein include meat, beans, pulses, fish, eggs and nuts.
- 5.86 The current UK recommendation for non-dairy sources of protein is that young children should consume 1 or 2 portions of foods rich in (non-dairy) protein each day. For more details, see [Current UK recommendations for protein for young children](#).

### Consumption of non-dairy sources of protein in the UK

- 5.87 Data from NDNS (years 2016 to 2019) and DNSIYC on the consumption and contribution of foods that are non-dairy sources of protein to the diets of children aged 12 to 60 months in the UK are presented in Table 5.19 to 5.21. Values that include non-consumers provide an estimate of the overall contribution of non-dairy sources of protein to the diets of young children, while values in consumers provide an estimate of the quantities consumed.
- 5.88 Nearly all children consumed non-dairy sources of protein over the 4 day survey period. Therefore, average values for consumers were similar to the average values at a population level (which includes non-consumers).
- 5.89 Non-dairy sources of protein contributed around 11% TDEI, 15% TDEI and 16% TDEI in children aged 12 to 18 months, 18 to 47 months and 48 to 60 months, respectively. Of the main non-dairy sources of protein examined, processed and unprocessed meat were the largest contributors in all age groups and the contribution of processed meat was higher in the older age groups. Consumers

aged 12 to 18, 18 to 47 and 48 to 60 months consumed 24, 29 and 39 grams per day, respectively, of unprocessed meat; and 21, 31 and 39 grams per day of processed meat, respectively.

**Table 5.19 Consumption of non-dairy sources of protein (grams per day and contribution to TDEI) in children aged 12 to 18 months in the UK (DNSIYC 2011)<sup>1</sup>**

Food Group	Mean consumption (SD) in grams per day Includes non-consumers	% Contribution to TDEI Includes non-consumers	% (number) of consumers over 4 days	Mean consumption (SD) in grams per day Consumers only	% Contribution to TDEI Consumers only
Unprocessed meat <sup>2</sup>	20 (23)	3.4	84 (1070)	24 (23)	4.1
Processed meat <sup>3</sup>	14 (19)	3.3	64 (844)	21 (19)	5.2
Fish <sup>4</sup>	10 (14)	2.0	54 (696)	18 (15)	3.2
Eggs <sup>5</sup>	7 (11)	1.1	44 (544)	16 (12)	2.6
Beans and pulses <sup>6</sup>	12 (19)	1.1	50 (630)	24 (20)	2.3
Plain nuts <sup>7</sup>	0.1 (1.3)	0.0	3 (35)	[3 (6)]	[1.3]
Total non-dairy protein sources <sup>8</sup>	63 (40)	10.8	98 (1246)	64 (39)	11.1

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake; [ ]: data based on cell size below 50

<sup>1</sup> Data from DNSIYC 2011. Number of participants: 1275.

<sup>2</sup> Processed meat: Bacon, ham, sausages, burgers, meat pies

<sup>3</sup> Unprocessed meat: Beef, pork, lamb, chicken, turkey, offal. Includes products, ready meals and homemade dishes

<sup>4</sup> Fish: All types of fish and shellfish including in manufactured products, ready meals and homemade dishes.

<sup>5</sup> Eggs: Boiled, fried, poached, scrambled eggs; omelettes.

<sup>6</sup> All types of beans and pulses including baked beans. Includes bean and pulse based products, dishes and ready meals. Excludes beans and pulses as a component of other products and dishes

<sup>7</sup> All types of unsalted uncoated nuts. Excludes nuts in manufactured products and dishes

<sup>8</sup> Non-dairy protein sources: sum of processed and unprocessed meat, fish, eggs, beans and pulses, plain nuts

**Table 5.20 Consumption of non-dairy sources of protein (grams per day and contribution to TDEI) in children aged 18 to 47 months in the UK (NDNS years 2016 to 2019)<sup>1</sup>**

Food Group	Mean consumption (SD) in grams per day Includes non-consumers	% Contribution to TDEI Includes non-consumers	% (number) of consumers over 4 days	Mean consumption (SD) in grams per day Consumers only	% Contribution to TDEI Consumers only
Processed meat <sup>3</sup>	24 (27)	5.7	80 (252)	31 (27)	7.2
Unprocessed meat <sup>2</sup>	24 (23)	3.8	84 (255)	29 (22)	4.6
Fish <sup>4</sup>	12 (15)	2.1	54 (178)	21 (14)	3.9
Eggs <sup>5</sup>	10 (14)	1.4	51 (166)	19 (15)	2.7
Beans and pulses <sup>6</sup>	13 (20)	1.3	50 (157)	27 (22)	2.7
Plain nuts <sup>7</sup>	1 (3)	0.3	11 (32)	[5 (7)]	[2.6]
Total non-dairy protein sources <sup>8</sup>	84 (39)	14.7	99 (304)	85 (38)	14.8

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake.

<sup>1</sup> Data from NDNS years 2016 to 2019. Number of participants: 306

<sup>2</sup> Unprocessed meat: Beef, pork, lamb, chicken, turkey, offal. Includes products, ready meals and homemade dishes

<sup>3</sup> Processed meat: Bacon, ham, sausages, burgers, meat pies

<sup>4</sup> Fish: All types of fish and shellfish including in manufactured products, ready meals and homemade dishes.

<sup>4</sup> Eggs: Boiled, fried, poached, scrambled eggs; omelettes.

<sup>5</sup> All types of beans and pulses including baked beans. Includes bean and pulse-based products, dishes and ready meals. Excludes beans and pulses as a component of other products and dishes

<sup>6</sup> All types of unsalted uncoated nuts. Excludes nuts in manufactured products and dishes

<sup>7</sup> Non-dairy protein sources: sum of processed and unprocessed meat, fish, eggs, beans and pulses, plain nuts

**Table 5.21 Consumption of non-dairy sources of protein (grams per day and contribution to TDEI) in children aged 48 to 60 months in the UK (NDNS years 2016 to 2019)<sup>1</sup>**

Food Group	Grams per day Mean (SD) including non-consumers	% Contribution to TDEI including non-consumers	% (number) of consumers over 4 days	Grams per day Mean (SD) in consumers only	% Contribution to TDEI for consumers only
Processed meat <sup>3</sup>	34 (31)	7.0	88 (88)	39 (30)	7.9
Unprocessed meat <sup>2</sup>	34 (33)	4.5	86 (84)	39 (32)	5.2
Fish <sup>4</sup>	12 (14)	1.7	54 (55)	22 (13)	3.1
Eggs <sup>5</sup>	10 (15)	1.3	52 (49)	[20 (15)]	[2.5]
Beans and pulses <sup>6</sup>	17 (24)	1.2	60 (60)	28 (25)	2.0
Plain nuts <sup>7</sup>	0.5 (3)	0.1	9 (11)	No data <30 consumers	No data <30 consumers
All non-dairy protein sources <sup>8</sup>	107 (58)	15.7	100 (102)	107 (58)	15.7

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake;

<sup>1</sup> Data from NDNS years 2016 to 2019. Number of participants: 102

<sup>2</sup> Unprocessed meat: Beef, pork, lamb, chicken, turkey, offal. Includes products, ready meals and homemade dishes

<sup>3</sup> Processed meat: Bacon, ham, sausages, burgers, meat pies

<sup>4</sup> Fish: All types of fish and shellfish including in manufactured products, ready meals and homemade dishes.

<sup>5</sup> Eggs: Boiled, fried, poached, scrambled eggs; omelettes.

<sup>6</sup> All types of beans and pulses including baked beans. Includes bean and pulse based products, dishes and ready meals. Excludes beans and pulses as a component of other products and dishes

<sup>7</sup> All types of unsalted uncoated nuts. Excludes nuts in manufactured products and dishes

<sup>8</sup> Non-dairy protein sources: sum of processed and unprocessed meat, fish, eggs, beans and pulses, plain nuts

## Consumption of non-dairy sources of protein and deprivation

- 5.90 Consumption of non-dairy sources of protein by IMD (see Glossary) in children aged 18 to 60 months (including non-consumers) is presented in Table 5.22.
- 5.91 The analysis indicated that although there were small differences in the consumption of non-dairy protein sources between IMD quintile 1 (least deprived) and quintiles 2 and 5 (most deprived), there was no obvious relationship between consumption of non-dairy protein sources and IMD quintile (as indicated by overlapping confidence intervals).

**Table 5.22 Consumption of non-dairy sources of protein by IMD quintile in children aged 18 to 60 months in England (NDNS years 2008 to 2019)<sup>1</sup>**

Consumption Mean (95%CI)	IMD quintile 1 (least deprived)	IMD quintile 2	IMD quintile 3	IMD quintile 4	IMD quintile 5 (most deprived)
Non-dairy protein sources <sup>2</sup> (grams per day)	60 (55 to 65)	65 (60 to 71)	62 (57 to 68)	62 (57 to 68)	66 (60 to 71)
Number of participants	210	212	182	234	277

Abbreviations: IMD, index of multiple deprivation.

<sup>1</sup> Data from NDNS years 2008 to 2019. Includes non-consumers.

<sup>2</sup> Non-dairy protein sources: sum of unprocessed meat, fish, eggs, beans and pulses, plain nuts. Excludes processed meat.

## Foods high in saturated fats, salt or free sugars

- 5.92 Foods that are high in saturated fats, salt or free sugars are placed outside of the main [Eatwell Guide](#) because they are not needed in the diet. If these foods and drinks are consumed, it should be done infrequently and in small amounts (PHE, 2018).
- 5.93 This category, often referred to as ‘HFSS’ foods (and drinks), typically includes sugar-sweetened beverages (SSBs), cakes, confectionery, biscuits, crisps, and savoury snacks (Scarborough et al, 2016). However, there is currently neither a single definition nor method for categorising foods and drinks as ‘HFSS’.
- 5.94 For example, in the UK, a nutrient profiling model (NPM) was developed by the Food Standards Agency to differentiate foods based on their nutrient composition in the context of television advertising of foods to children (DH, 2011). The NPM is based on a simple scoring system where points are allocated on the basis of the nutrient content of 100g of a food or drink. Foods that score 4 or more points and drinks that score 1 or more points are classified as ‘less healthy’ (or ‘HFSS’ which, in the context of the NPM, stands for foods that are high in saturated fats, sodium or total sugars). Such products are subject to regulatory controls on advertising to children, as well as restrictions on their promotion in retail settings (DHSC, 2022).
- 5.95 At the same time, voluntary nutrition labelling on the front of pre-packaged foods and drinks, known as ‘front of pack’ (FoP) nutrition labelling, provides consumers with a colour coded visual display of the nutrient composition of a food or drink to enable them to make healthier dietary choices. The colour coding scheme of red, amber and green is used to represent whether a product is ‘high’ (red), ‘medium’

(amber) or 'low' (green) in total fat, saturated fats, total sugars or salt, alongside how much energy it provides (which is presented on a neutral background). The criteria for 'high' (red) are based on a percentage reference intake (RI) for each nutrient provided per 100g/ml or per portion (DH, 2016). RIs for energy, total fat, saturated fats, total sugars and salt are specified in [EU Regulation No. 1169/2011 on the provision of food information to consumers \(EU FIC\)](#) and are the maximum absolute amounts that should be consumed in a day, based on an average sized woman doing an average amount of physical activity (DH, 2016). A food is 'high' in a nutrient (for example, saturated fats) if it provides >25% of the RI of that nutrient per 100g or >30% of the RI per portion. A drink is 'high' in a nutrient if it provides >12.5% of the RI per 100ml or >15% of the RI per portion (DH, 2016). In addition, portion size criteria apply to food portions or serving sizes greater than 100g and for drinks greater than 150ml. These additional criteria ensure that products which contribute more than 30% (for food) and 15% (for drinks) of an adult's recommended daily maximum intake for a particular nutrient are labelled as red ('high') for the respective nutrient, regardless of their content per 100g or ml.

- 5.96 It is important to note that neither the colour coding nor descriptors 'high', 'medium' or 'low' represent nutrition claims.
- 5.97 Another term that has gained currency in recent years is 'ultra-processed' foods (UPFs). UPF is a term coined by the researchers who developed the NOVA classification system and includes foods that are clearly less healthy (such as sugar-sweetened drinks, confectionery, salty snacks) but also those that would be encouraged as part of a healthier diet in line with the Eatwell Guide, such as some wholemeal sliced breads, baked beans, lower-fat yoghurts, wholegrain breakfast cereals and vegetable pasta sauces. For more details on existing food processing classifications, including NOVA, and the suitability and methods to apply food processing definition(s) as a dietary exposure, see [SACN's position statement 'Processed foods and health'](#).
- 5.98 In recent years, the 'out of home' sector has become an important determinant of food intake and diet quality in the UK (PHE, 2017b). Although the sector is not well defined, it broadly covers businesses that provide food and meals bought and eaten out of the home, taken away or delivered to the home (also known as the eating out, takeaway and delivery sector) (PHE, 2020a). In the UK, one fifth of children eat food from out of home food outlets at least once a week (PHE, 2017b). Meals and snacks from such outlets are typically higher in energy, saturated fats and salt than home-cooked meals (Huang et al, 2021; Robinson et al, 2018).

## **Consumption of foods that are energy dense and high in saturated fats, salt or free sugars in the UK**

- 5.99 As there is currently no single method for defining or categorising foods (and drinks) that are energy dense and high in saturated fats, salt or free sugars (see paragraphs 5.93 to 5.95), SACN took a pragmatic approach when selecting food groups from dietary surveys (DNSIYC and NDNS) to highlight in this section. The selected food groups (not exhaustive) were considered most likely to contain foods or drinks that were either energy dense and high in saturated fats, salt or free sugars.
- 5.100 Data from DNSIYC and NDNS (years 2016 to 2019) on the consumption and contribution of foods (and drinks) that are energy dense and high in saturated fats, salt or free sugars to the diets of children aged 12 to 60 months in the UK are presented in Table 5.23 to 5.25. Values that include non-consumers provide an estimate of the overall contribution of foods to the diets of young children, while values in consumers provide an estimate of the quantities consumed.
- 5.101 Nearly all children consumed foods and drinks from the list of selected foods and drinks. Therefore, average values in consumers were similar to the average values at a population level (which includes non-consumers).
- 5.102 The selected foods and drinks contributed approximately 16% TDEI, 24% TDEI and 30% TDEI in children aged 12 to 18 months, 18 to 47 months and 48 to 60 months, respectively. Of the foods and drinks that were examined, biscuits, buns, cakes and pastries was the largest food group contributor to TDEI.

**Table 5.23 Consumption of foods and drinks that are energy dense and are high in saturated fats, salt or free sugars (grams per day and contribution to TDEI) in children aged 12 to 18 months in the UK (DNSIYC 2011)<sup>1</sup>**

Food Group	Mean consumption (SD) in grams per day Includes non-consumers	% Contribution to TDEI Includes non-consumers	% (number) of consumers over 4 days	Mean consumption (SD) in grams per day Consumers only	% Contribution to TDEI Consumers only
Biscuits, buns, cakes, pastries	11 (12)	4.7	78 (998)	15 (12)	6.1
Dairy desserts <sup>2</sup>	28 (33)	3.1	63 (802)	45 (31)	4.9
Chips and fried potato products	9 (14)	1.8	44 (582)	19 (16)	4.0
Confectionery	3 (6)	1.5	41 (558)	8 (7)	3.6
Puddings	10 (23)	1.1	30 (399)	33 (32)	3.6
Crisps and savoury snacks	2 (4)	1.1	43 (538)	5 (4)	2.7
Sugar, preserves, sweet spreads	2 (4)	0.6	45 (555)	4 (4)	1.3
Pizza	2 (7)	0.6	12 (156)	18 (12)	4.5
Sugar-sweetened beverages <sup>3</sup>	42 (119)	0.4	27 (333)	156 (186)	1.6
Ice cream	2 (6)	0.4	15 (196)	13 (10)	2.4
Breakfast cereals high sugar <sup>4</sup>	1 (4)	0.3	11 (139)	7 (9)	3.0
Flavoured milks <sup>5</sup>	1 (8)	0.1	2 (24)	No data <30 consumers	No data <30 consumers
Salted nuts	0 (0)	0.0	0.2 (2)	No data <30 consumers	No data <30 consumers
Total selected foods and drinks	113 (135)	15.6	98 (1253)	115 (135)	15.9

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake.

<sup>1</sup> Data from DNSIYC 2011. Number of participants: 1275

<sup>2</sup> Excludes yoghurt and fromage frais

<sup>3</sup> Includes carbonated drinks, concentrates and ready to drink products with added sugars.

<sup>4</sup> Products with sugar content >22.5g per 100g.

<sup>5</sup> Includes milkshakes, flavoured milk based drinks, hot chocolate, evaporated and condensed

**Table 5.24 Consumption of foods and drinks that are energy dense and are high in saturated fats, salt or free sugars (grams per day and contribution to TDEI) in children aged 18 to 47 months in the UK (NDNS years 2016 to 2019)<sup>1</sup>**

Food Group	Mean consumption (SD) in grams per day Includes non-consumers	% Contribution to TDEI Includes non-consumers	% (number) of consumers over 4 days	Mean consumption (SD) in grams per day Consumers only	% Contribution to TDEI Consumers only
Biscuits, buns, cakes, pastries	22 (20)	8.4	91 (278)	24 (20)	9.3
Crisps and savoury snacks	6 (7)	2.9	72 (214)	8 (7)	4.0
Confectionery	8 (11)	2.8	66 (208)	12 (12)	4.3
Chips and fried potato products	13 (17)	2.4	62 (189)	20 (17)	3.8
Dairy desserts <sup>2</sup>	18 (31)	1.5	43 (143)	41 (35)	3.5
Pizza	6 (12)	1.3	25 (70)	22 (15)	5.2
Sugar, preserves, sweet spreads	3 (5)	1.2	67 (210)	5 (6)	1.8
Breakfast cereals high sugar <sup>3</sup>	3 (6)	1.1	26 (69)	11 (8)	4.1
Ice cream	6 (11)	1.0	33 (101)	18 (13)	3.0
Puddings	8 (18)	0.7	25 (80)	30 (24)	2.8
Sugar-sweetened beverages <sup>4</sup>	19 (60)	0.4	21 (70)	88 (105)	1.7
Salted nuts	0.2 (1.3)	0.1	4 (12)	No data <30 consumers	No data <30 consumers
Flavoured milks <sup>5</sup>	1 (10)	0.1	4 (15)	No data <30 consumers	No data <30 consumers
Total selected foods and drinks	112 (87)	23.9	99 (304)	112 (87)	24.1

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake.

<sup>1</sup> Data from NDNS years 2016 to 2019. Number of participants 306

<sup>2</sup> Excludes yoghurt and fromage frais.

<sup>3</sup> Products with sugar content >22.5g per 100g.

<sup>4</sup> Includes carbonated drinks, concentrates and ready to drink products with added sugars.

<sup>5</sup> Includes milkshakes, flavoured milk based drinks, hot chocolate, evaporated and condensed

**Table 5.25 Consumption of foods and drinks that are energy dense and are high in saturated fats, salt or free sugars (grams per day and contribution to TDEI) in children aged 48 to 60 months in the UK (NDNS years 2016 to 2019)<sup>1</sup>**

Food Group	Mean consumption (SD) in grams per day Includes non-consumers	% Contribution to TDEI Includes non-consumers	% (number) of consumers over 4 days	Mean consumption (SD) in grams per day Consumers only	% Contribution to TDEI Consumers only
Biscuits, buns, cakes, pastries	29 (21)	10.1	94 (94)	31 (21)	10.7
Confectionery	13 (13)	3.8	72 (75)	18 (13)	5.3
Chips and fried potato products	18 (19)	3.1	75 (79)	25 (18)	4.1
Crisps and savoury snacks	6 (8)	2.6	59 (65)	11 (7)	4.4
Sugar, preserves, sweet spreads	7 (8)	2.0	80 (82)	9 (8)	2.5
Ice cream	12 (18)	1.9	51 (50)	24 (20)	3.7
Pizza	7 (13)	1.5	29 (35)	[24 (13)]	[5.1]
Breakfast cereals high sugar <sup>2</sup>	4 (8)	1.3	28 (33)	[14 (10)]	[4.8]
Dairy desserts <sup>3</sup>	14 (23)	1.2	44 (47)	[33 (24)]	[2.7]
Puddings	10 (23)	0.8	23 (29)	No data <30 consumers	No data <30 consumers
Flavoured milks <sup>4</sup>	10 (32)	0.7	18 (16)	No data <30 consumers	No data <30 consumers
Sugar-sweetened beverages <sup>5</sup>	32 (89)	0.5	22 (22)	No data <30 consumers	No data <30 consumers
Salted nuts	1 (4)	0.2	5 (4)	No data <30 consumers	No data <30 consumers
Total selected foods and drinks	165 (115)	29.7	100 (102)	165 (115)	29.7

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake.

<sup>1</sup> Data from NDNS years 2016 to 2019. Number of participants = 102

<sup>2</sup> Products with sugar content >22.5g per 100g.

<sup>3</sup> Excludes yoghurt and fromage frais.

<sup>4</sup> Includes milkshakes, flavoured milk based drinks, hot chocolate, evaporated and condensed

<sup>5</sup> Includes carbonated drinks, concentrates and ready to drink products with added sugars.

## Consumption of foods that are energy dense and high in saturated fats, salt or free sugars and deprivation

- 5.103 Consumption of foods that are energy dense and high in saturated fats, salt or free sugars by IMD (see Glossary) in children aged 18 to 60 months is presented in Table 5.26.
- 5.104 The analysis indicated that although there were differences in the consumption of the selected foods and drinks between the least deprived IMD quintiles (quintile 1 and 2) and quintiles 3 to 5 (most deprived), there was no significant relationship (as indicated by overlapping confidence intervals).

**Table 5.26 Consumption of selected foods that are energy dense and high in saturated fats, salt or free sugars by IMD quintile in children aged 18 to 60 months in England (NDNS years 2008 to 2019)<sup>1</sup>**

Consumption Mean (95%CI)	IMD quintile 1 (least deprived)	IMD quintile 2	IMD quintile 3	IMD quintile 4	IMD quintile 5 (most deprived)
Selected foods <sup>2</sup> mean (grams per day)	156 (137 to 175)	145 (130 to 161)	169 (147 to 191)	158 (142 to 174)	163 (147 to 180)
Number of participants	210	212	182	234	277

Abbreviations: HFSS, high (saturated) fat salt sugar foods; IMD, index of multiple deprivation.

<sup>1</sup> Data from NDNS years 2008 to 2019. Includes non-consumers

<sup>2</sup> Sugar, preserves and sweet spreads; confectionery; sugar-sweetened beverages; high sugar breakfast cereals; biscuits, buns, cakes, pastries; puddings, crisps and savoury snacks; salted nuts; ice cream; chips and fried potato products; flavoured milks, dairy desserts, pizza

## Systematic review evidence identified on foods that are energy dense and high in saturated fats, salt or free sugars

- 5.105 For this report, SR evidence was identified on the health impact of sugar-sweetened beverages (see [sugar-sweetened beverages in chapter 6](#)). SR evidence was also identified on the health impact of dietary patterns characterised by the consumption of 'ultra-processed' foods as defined by the NOVA food classification system (see paragraph 5.93). This evidence is described in under [Diet quality in this chapter](#).

## Commercially manufactured foods and drinks marketed specifically for infants and young children (excluding formula)

- 5.106 While home-prepared foods are generally recommended to help introduce infants and young children to a range of appropriate flavours and textures, commercially manufactured foods and drinks (excluding formula and water) that are marketed specifically for children aged 4 months to 36 months are widely available in the UK (PHE, 2019a). Products that are sold in the UK include (PHE, 2019a):
- baby meals (composite main meals, 100% fruit and vegetable purées, dry cereals, desserts, breakfasts, soups)
  - finger foods (savoury or sweet, fruit or vegetable based finger foods)
  - drinks that exclude formula milk and plain water.
- 5.107 Commercially manufactured foods and drinks marketed specifically for infants and young children must comply with [EU Commission Directive 2006/125/EC](#), which was retained as UK law after the UK left the EU. The Directive sets out rules on composition and labelling, and specific rules on the presence of pesticide residues for 'processed cereal-based foods' and 'baby foods' other than processed cereal-based foods. In addition to these requirements, products must also comply with other specific provisions in relation to hygiene, the use of food additives, the presence of contaminants and the use of materials intended to come into contact with the foods (PHE, 2019a).
- 5.108 However, an evidence review published by Public Health England (PHE) in 2019 found that the messaging, labelling and marketing of commercially manufactured foods and drinks marketed specifically for infants and young children was not always in line with young child feeding recommendations (PHE, 2019a).
- 5.109 Moreover, the nutrient composition of many products available on the market in the UK was inconsistent with UK dietary recommendations for this age group (PHE, 2019a). Some infant foods had added sugar or salt or contained ingredients that are high in sugar or salt. This was particularly common with 'finger foods', whereby sweet finger foods made up nearly two-thirds of the infant finger food market in 2017 to 2018 (PHE, 2019a).
- 5.110 The review also found that sweet finger foods provided a greater proportion of energy intake that is suggested for snack occasions throughout the day for children aged 1 year (12.8% of the Estimated Average Requirement (EAR) versus the recommended 10% of the EAR, which is based on the nutrient framework used for [the example early years menus](#)). Finger foods such as sweet and savoury biscuits, crisps and puffs, and processed dried fruit products, are not consistent

with the types of foods given as examples of healthy snacks for young children (that is, fruit, vegetable sticks, toast, bread or plain yoghurt) (PHE, 2019a).

- 5.111 Commercially manufactured finger foods have been the growth driver in the infant food market in recent years. Data from Kantar Nutrition showed that spend on these products increased from £61 million in 2014 to £101 million in 2018, and volume sales grew by 10.8% from 2017 to 2018. This mirrored the growth in the wider snack food market (PHE, 2019a).
- 5.112 PHE's report stated the concern that the way many products were labelled and marketed may have encouraged snacking as well as perceptions that these products formed an expected and appropriate part of a child's diet. In addition, the use of nutrition and implied health claims, as well as 'health halo' statements, could suggest to parents that these products were healthier than their nutrient composition indicated (PHE, 2019a).
- 5.113 In addition, PHE found that around one-third of commercially manufactured infant foods were packaged in pouches, many of which have nozzles. Sucking from these pouches may be harmful for developing teeth. Current UK guidance on preventing dental caries states that from the age of 6 months children should be encouraged to drink from a free-flow cup rather than one with a valve which requires a child to suck (DHSC, 2021c). However, advice on how to feed baby foods packaged in pouches (with a spoon) has not been consistent across the market (PHE, 2019a). Moreover, no systematic review evidence on different modes of presenting food and drinks in children aged 1 year and over was identified for this report.

## **Consumption of commercially manufactured foods and drinks marketed specifically for infants and young children in the UK**

- 5.114 Data from DNSIYC and NDNS (years 2016 to 2019) on the consumption and contribution of commercially manufactured foods and drinks marketed specifically for infants and young children to the diets of children aged 12 to 60 months in the UK are presented in Table 5.27 and Table 5.28. Values that include non-consumers provide an estimate of the overall contribution of these products to the diets of young children, while values for consumers only provide an estimate of the quantities consumed.
- 5.115 Data are presented for children aged 12 to 18 and 18 to 47 months only. There were too few consumers aged 48 to 60 months for data in this age group to be presented.
- 5.116 For the remainder of this section, commercially manufactured foods and drinks marketed specifically for infants and young children will be referred to as

‘commercially manufactured infant foods’ and ‘commercially manufactured infant drinks’ for brevity.

- 5.117 Sixty-five percent of children aged 12 to 18 months consumed commercially manufactured infant foods and drinks over the 4 day survey period, while 20% of children aged 18 to 47 months consumed these products.
- 5.118 Commercially manufactured infant foods and drinks made a greater contribution to TDEI in children aged 12 to 18 months (6.2% TDEI) compared with older age groups (1.0% TDEI) at a population level (which includes both consumers and non-consumers of these products) (Table 5.27). Consumers of these products aged 12 to 18 months obtained 9.6% TDEI from these products (mainly from foods) while consumers aged 18 to 47 months obtained approximately 5% TDEI (only from foods).
- 5.119 As DNSIYC data is from 2011, it is likely that, given the upward trend in the purchasing of commercially manufactured foods and drinks (see paragraph 5.111), children aged 12 to 18 months are now obtaining a higher proportion of their TDEI from these products.

**Table 5.27 Consumption of commercially manufactured foods and drinks marketed specifically for infants and young children (grams per day) and contribution to TDEI in children aged 12 to 18 months in the UK (DNSIYC)<sup>1</sup>**

Food Group	Mean consumption (SD) in grams per day Includes non-consumers	% Contribution to TDEI  Includes non-consumers	% (number) of consumers over 4 days	Mean consumption (SD) in grams per day Consumers only <sup>2</sup>	% Contribution to TDEI  Consumers only <sup>2</sup>
Foods <sup>3</sup>	58 (93)	6.0	64 (807)	91(103)	9.4
Drinks <sup>4</sup>	10 (50)	0.2	7 (94)	140 (126)	3.3
Foods and drinks combined	68 (113)	6.2	65 (819)	105 (126)	9.6

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake.

<sup>1</sup> Data in children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013).

<sup>2</sup> There were 807 consumers for commercially manufactured infant foods, 94 consumers for commercial infant drinks and 819 consumers for commercially manufactured infant foods and drinks combined.

<sup>3</sup> Commercially manufactured infant foods include instant and ready to eat foods specifically manufactured for young children

<sup>4</sup> Commercially manufactured infant drinks include powdered, concentrated and ready-to-drink beverages specifically manufactured for young children

**Table 5.28 Consumption of commercially manufactured foods and drinks marketed specifically for infants and young children (grams per day) and contribution to TDEI in children aged 18 to 47 months in the UK (NDNS years 2016 to 2019)<sup>1</sup>**

Food Group	Mean consumption (SD) in grams per day Includes non-consumers	% Contribution to TDEI Includes non-consumers	% (number) of consumers over 4 days	Mean consumption (SD) in grams per day Consumers only <sup>2</sup>	% Contribution to TDEI Consumers only <sup>2</sup>
Foods <sup>3</sup>	9 (28)	1.0	20 (77)	47 (47)	5.0
Drinks	0.2 (4)	0.0	0.5 (1)	No data <30 consumers	No data <30 consumers
Foods and drinks <sup>4</sup> combined	10 (28)	1.0	20 (78)	47 (46)	4.9

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake.

<sup>1</sup> Data from NDNS years 2016 to 2019.

<sup>2</sup> There were 77 consumers for commercially manufactured infant foods and 78 consumers for commercially manufactured infant foods and drinks combined. There were too few consumers of commercial infant drinks in this age group for data to be presented.

<sup>3</sup> Commercially manufactured infant foods include instant and ready to eat foods specifically manufactured for young children

<sup>4</sup> Commercially manufactured infant drinks include powdered, concentrated and ready-to-drink beverages specifically manufactured for young children

## **Contribution of commercially manufactured foods and drinks marketed specifically for infants and young children in the UK to intakes of free sugars, saturated fats and salt**

- 5.120 Table 5.29 and Table 5.30 present data from both dietary surveys on the contribution of commercially manufactured infant foods and drinks to intakes of free sugars, saturated fats and salt in children aged 12 to 47 months. There were too few consumers in the 48 to 60 month age group for data to be presented.
- 5.121 Among consumers aged 12 to 18 months (65% of this age group), commercially manufactured infant foods and drinks provided 20% of free sugars intake (13.5% at a population level). Specifically, commercially manufactured infant foods provided 17.8% of free sugars intake (11.8% at a population level) while commercially manufactured infant drinks provided 22.1% (1.7% at a population level). Among consumers aged 18 to 47 months (20% of this age group), commercially manufactured infant foods contributed 11.5% to free sugars intake (2.3% at a population level). Commercially manufactured infant drinks did not make an appreciable contribution to free sugars intake in this age group.

5.122 Among consumers aged 12 to 18 months, commercially manufactured infant foods contributed 4.5% to saturated fat intake (2.9% at a population level) and 5.0% to salt intake (3.2% at a population level). This reduced to 2.7% and 1.6%, respectively, in consumers aged 18 to 47 months (and 0.5% and 0.3%, respectively, at a population level).

**Table 5.29 Contribution of commercially manufactured foods and drinks marketed specifically for infants and young children to free sugars, saturated fat and salt intakes in children aged 12 to 18 months in the UK (DNSIYC)<sup>1</sup>**

Food Group	% Contribution to free sugars intake including non-consumers	% Contribution to free sugars intake in consumers only	% Contribution to saturated fat intake including non-consumers	% Contribution to saturated fat intake in consumers only	% Contribution to salt intake including non-consumers	% Contribution to salt intake in consumers only
Foods <sup>2</sup>	11.8	17.8	2.9	4.5	3.2	5.0
Drinks <sup>3</sup>	1.7	22.1	0.0	0.0	0.0	0.7
Foods and drinks combined	13.5	20.0	2.9	4.5	3.2	5.0

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake.

<sup>1</sup> Data in children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013). There were 807 consumers for commercially manufactured infant foods, 94 consumers for commercially manufactured infant drinks and 819 consumers for commercially manufactured infant foods and drinks combined.

<sup>2</sup> Commercially manufactured infant foods include instant and ready to eat foods specifically manufactured for young children

<sup>3</sup> Commercially manufactured infant drinks include powdered, concentrated and ready-to-drink beverages specifically manufactured for young children

**Table 5.30 Contribution of commercially manufactured foods and drinks marketed specifically for infants and young children to free sugars, saturated fat and salt intakes in children aged 18 to 47 months in the UK (NDNS years 2016 to 2019)<sup>1</sup>**

Food Group	% Contribution to free sugars intake including non-consumers	% Contribution to free sugars intake in consumers only	% Contribution to saturated fat intake including non-consumers	% Contribution to saturated fat intake in consumers only	% Contribution to salt intake including non-consumers	% Contribution to salt intake in consumers only
Foods <sup>2</sup>	2.3	11.5	0.5	2.7	0.3	1.6
Foods and drinks <sup>3</sup> combined	2.4	11.5	0.5	2.7	0.3	1.6

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake.

<sup>1</sup> Data from NDNS years 2016 to 2019. There were 77 consumers for commercially manufactured infant foods and 78 consumers for commercially manufactured infant foods and drinks combined. There were too few consumers of commercially manufactured infant drinks in this age group for data to be presented.

<sup>2</sup> Commercially manufactured infant foods include instant and ready to eat foods specifically manufactured for young children

<sup>3</sup> Commercially manufactured infant drinks include powdered, concentrated and ready-to-drink beverages specifically manufactured for young children

## Allergenic foods

- 5.124 To inform SACN's review of feeding in the first year of life, a joint working group comprising SACN and COT members with an independent chair was convened to undertake a benefit:risk assessment on the timing of introduction of peanut and hen's egg into the infant diet and risk of developing allergy to these foods.
- 5.125 The conclusions and recommendations from the resulting joint SACN-COT statement [‘Assessing the health benefits and risks of the introduction of peanut and hen's egg into the infant diet before six months of age in the UK’](#) (SACN/COT, 2018) were endorsed and reiterated in SACN's [‘Feeding in the first year of life’](#) report (SACN, 2018). They included the following conclusion and associated recommendation:
- There was reasonable evidence to demonstrate that the deliberate exclusion or delayed introduction of peanut or hen's egg beyond 6 to 12 months of age may increase the risk of allergy to the same foods. Importantly, once introduced, these foods should continue to be consumed as part of the infant's usual diet in order to minimise the risk of allergy to peanut or hen's egg developing after initial exposure. Families of infants with a history of early-onset eczema or suspected food allergy may wish to seek medical advice before introducing these foods.
  - The deliberate exclusion of peanut and hen's egg beyond 6 to 12 months of age may increase the risk of allergy to the same foods. Once introduced, and where tolerated, these foods should be part of the infant's usual diet, to suit both the individual child and family. If initial exposure is not continued as part of the infant's usual diet, then this may increase the risk of sensitisation and subsequent food allergy.
- 5.126 For this report, no SR evidence was identified on the impact of diet or nutrition on the development or prevention of allergies in children aged 1 to 5 years.

# Dietary patterns

## Background

- 5.127 Dietary pattern analysis is used to examine dietary behaviours of populations and represents the combinations of foods and nutrients that are consumed in real life (Schulz et al, 2021). Many dietary patterns provide an indication of adherence to population dietary guidelines or the overall 'healthiness' of a diet, commonly described as 'diet quality' (Gherasim et al, 2020). Dietary pattern analysis can also identify other types of dietary patterns depending on the aim and method (Ocke, 2013).
- 5.128 There are at least 3 different approaches to dietary pattern analysis: hypothesis-driven, exploratory, and hybrid approaches (Jannasch et al, 2021; Ocke, 2013; Schulz et al, 2021).
- 5.129 Hypothesis-driven approaches (also known as 'a priori' approaches) compare the dietary intake of a population group against a predefined or established dietary pattern (for example, the Mediterranean diet) or existing dietary guidelines. Adherence to the predefined dietary pattern or dietary guidelines is measured using a scoring system or index (for example, the MED score for the Mediterranean diet and various diet quality indices [DQIs] in the case of adherence to specific dietary guidelines) (Gherasim et al, 2020; Ocke, 2013; Schulz et al, 2021). The main advantage of hypothesis-driven approaches is that, in principle, they can be applied to different populations. However, the establishment of such scoring systems involves a level of subjectivity and therefore their use is not entirely objective (Gherasim et al, 2020).
- 5.130 Hypothesis-driven dietary patterns can give an indication of the overall 'healthiness' of a diet through the use of various DQIs (Gherasim et al, 2020). However, DQIs have limitations. Many DQIs are based on dietary guidelines that are population specific, which may limit their applicability and generalisability. It can also be difficult to compare various scoring systems (Gherasim et al, 2020; Gil et al, 2015). In addition, only a few DQIs have been assessed for validity and reliability in children or have been used to assess childhood growth and other prospective health outcomes (Dalwood et al, 2020).
- 5.131 In contrast to hypothesis-driven dietary patterns, exploratory approaches (also known as a posteriori approaches) do not begin from predefined dietary patterns. Instead, exploratory approaches apply statistical methods to dietary intake data collected from a population sample in order to identify dietary patterns for that population (Gherasim et al, 2020). Commonly used statistical methods are cluster analysis and factor or principal component analysis. The identified dietary patterns are labelled or described based on an (often simplistic) interpretation of the pattern

identified (Schulz et al, 2021). However, some dietary patterns identified through exploratory approaches can also be compared with existing dietary guidelines (which reflect diet quality) for the population for which the dietary pattern was derived. For example, a 'prudent' dietary pattern (characterised by greater consumption of vegetables, fruits, wholegrains, poultry and fish) and a 'Western' dietary pattern (characterised by greater intakes of white bread, red or processed meat, potatoes and high-fat dairy products) are 2 common dietary patterns derived from European population data (Gherasim et al, 2020; Ocke, 2013; Schulz et al, 2021). Dietary patterns derived through exploratory approaches can be challenging to interpret because the analyses not only involve some level of subjectivity but as they are population specific, can be limited in their generalisability to other populations (Schulz et al, 2021).

- 5.132 Hybrid approaches aim to explain the relationship between diet and health through intermediate factors. Hybrid methods consider existing knowledge (for example, known health effects of dietary components) but the grouping of food items is exploratory by design. An example of a hybrid approach is the reduced rank regression method (Gherasim et al, 2020; Ocke, 2013; Schulz et al, 2021).
- 5.133 It has been suggested that no method of studying dietary patterns is superior to any other method and that exploratory approaches and hypothesis-driven approaches may complement each other and could be used simultaneously (Ocke, 2013; Previdelli et al, 2016).
- 5.134 However, data on the validity and reliability of both hypothesis-driven dietary patterns and dietary patterns derived using exploratory approaches are sparse (Jannasch et al, 2021). It has been recommended that the validity of dietary patterns across different countries should be investigated to examine the generalisability of already identified dietary patterns outside the population from which they were derived (Jannasch et al, 2018).

## **Systematic review evidence identified on dietary patterns**

- 5.135 For this report, dietary patterns examined by SRs were divided into 2 categories:
- dietary patterns that could be considered to reflect diet quality (see paragraphs 5.127 to 5.131) (evidence described from page 248)
  - other dietary patterns (evidence described from page 254).

## **Limitations of the systematic review evidence on dietary patterns**

- 5.136 Many of the primary studies included in the SRs identified on dietary patterns did not adjust for key potential confounding factors, mediators and effect modifiers. These include age, sex, socioeconomic status (SES) and maternal education, which is associated with healthier dietary patterns and has a key influence on children's diets (Emmett et al, 2015).
- 5.137 Primary studies that examined outcomes relating to or resulting from effects on energy balance (for example, body mass index [BMI]) did not always adequately account for children's body size at baseline.
- 5.138 Primary studies that examined cognition-related outcomes did not always adjust for child baseline cognition and parental cognition.

## **Diet quality**

### **Systematic review evidence identified on diet quality and health outcomes**

- 5.139 Two SRs without MA (Costa et al, 2018; Tandon et al, 2016) were identified that included studies that examined diet quality. Costa et al (2018) examined the relationship between consumption of 'ultra-processed' foods (UPFs) as defined by the NOVA food classification system (see paragraph 5.93) or dietary patterns characterised by the consumption of groups of UPFs. It should be noted, however, that the primary studies included in the SRs used terminology such as 'junk foods', 'convenience foods' and 'discretionary foods' to describe the dietary patterns they examined.
- 5.140 For the purposes of this report, the dietary patterns examined by the SRs were categorised into 'healthy' and 'unhealthy' dietary patterns (See Annex 8 Table A8.29) according to how they were described by the SRs (or primary studies) as well as how similar they are to current UK dietary recommendations. For example, dietary patterns characterised by the consumption of UPFs (or 'junk foods', 'convenience foods', 'discretionary foods') were categorised as 'unhealthy' because such dietary patterns would also likely be energy dense and high in saturated fats, salt and (free) sugars, and low in dietary fibre, vegetables and fruit. In contrast, dietary patterns described as 'health-conscious' or 'nutrient-dense' were categorised as 'healthy'.
- 5.141 Key outcomes examined by the SRs were body composition (body fat) and cognitive development.

- 5.142 All primary studies included in the SRs were conducted in HICs.
- 5.143 Details of the 2 SRs included in this section can be found in Annex 5 (Table A5.3). Quality assessment of the SRs using the AMSTAR 2 tool can be found in Annex 8 (Tables A8.5). Additional data extracted on the primary studies can be found in Annex 9 (Table A9.25).
- 5.144 The criteria used to grade the evidence are provided in chapter 2 (Table 2.4, paragraphs 2.54 to 2.59). Summary tables of the evidence grading process for this section are provided in Annex 10 (Tables A10.11, A10.12 and A10.36).

## **Diet quality and body fat**

- 5.145 One SR without MA (Costa et al, 2018) (AMSTAR 2 confidence rating: moderate) was identified that examined the relationship between adherence to ‘unhealthy’ dietary patterns (derived using exploratory approaches) that were characterised by the consumption of UPFs (see paragraph 5.93) and body fat. It included 3 PCS that examined this relationship in children aged 1 to 5 years.
- 5.146 One PCS (in 292 participants) reported that children aged 3.8 to 4.8 years who scored in the highest quartile for a dietary pattern that contained mostly UPFs (as described by the SR and identified using reduced rank regression) had higher fat mass (kg) compared with children with scores in the lower quartiles across all age groups that were assessed (older than 4.8 to 5.8 years, older than 5.8 to 6.8 years and older than 6.8 to 7.8 years). The analyses were adjusted for sex, exact age, height, TDEI, calcium intake, accelerometer counts per minute, TV viewing time and outdoor playtime.
- 5.147 One PCS (in 585 participants) reported that among boys (196 included in the analysis), a dietary pattern characterised by ‘convenience food consumption’ (and measured as % TDEI at age 3 years) predicted an increase in % body fat at age 18 years (beta coefficient 0.104; 95% CI NR;  $p=0.0098$ ). However, the same PCS reported no association in girls (170 were included in the analysis). The analyses were adjusted for age, TDEI, physical activity, and maternal BMI and education. It is important to note that the SR did not include ‘convenience foods’ consumed in communal eating environments (such as day-care centres and schools) as the focus of the SR was on eating within family settings.
- 5.148 One PCS (in 4750 participants) reported an association between adherence to a ‘junk food dietary pattern’ (identified by principal components analysis) at age 38 months and increased body fat at age 15 years (beta coefficient 0.06; 95% CI 0.02 to 0.10;  $p=0.002$ ). The analysis was adjusted for sex and age at the time of body composition measurement, TDEI at age 38 months (see chapter 3, paragraphs 3.48 and 3.49), parental factors (maternal and paternal height and BMI, maternal age and parity) and social factors (social class and maternal education).

## Summary: diet quality and body composition

- 5.149 The evidence identified from SRs on the relationship between ‘unhealthy’ dietary patterns (see paragraph 5.140) and body composition in children aged 1 to 5 years is summarised in Table 5.31.

**Table 5.31 Summary of the evidence on diet quality and body composition**

Exposure	Outcome	Direction of association <sup>1</sup>	Certainty of evidence
‘Unhealthy’ dietary patterns	Body fat	↑	Limited

<sup>1</sup> Direction of association for reported outcomes: ↑increase

- 5.150 The available evidence from SRs on the relationship between dietary patterns described as ‘unhealthy’ and body fat in children aged 1 to 5 years is from 1 SR given a moderate confidence rating using the AMSTAR 2 tool.
- 5.151 Evidence from 3 PCS included in the SR without MA by Costa et al (2018) suggests that greater adherence to ‘unhealthy’ dietary patterns (characterised by consumption of ‘UPFs’, ‘convenience foods’ or ‘junk foods’) in children aged 1 to 5 years are associated with greater body fat in childhood and adolescence. The evidence was graded as ‘limited’ based on 3 PCS that provided evidence of a consistent direction of association.

## Diet quality and cognitive outcomes

- 5.152 One SR without MA (Tandon et al, 2016) was identified that examined the relationship between diet quality and cognitive outcomes.
- 5.153 Tandon et al (2016) (AMSTAR 2 confidence rating: critically low) included 5 PCS that examined the relationship between diet quality and various measures of cognitive ability. These included vocabulary, cognitive ability, Key Stage 2 (KS2) performance (see Glossary) and Intelligence Quotient (IQ).
- 5.154 One PCS (in 1346 participants) examined the relationship between diet quality (measured by the Eating Assessment in Toddlers [EAT] diet score; see Glossary) and receptive vocabulary and non-verbal cognitive ability. Receptive vocabulary was measured using the Peabody Picture Vocabulary Test (PPVT III). The PCS reported that a higher EAT score at age 1 year was associated with a higher PPVT II score and better non-verbal cognitive ability at age 10 years. The analysis was adjusted for sex, maternal age and education, family income, a father living with family, reading to the child, maternal mental health distress and breastfeeding (duration not specified).

- 5.155 One PCS (in 7652 participants) examined the relationship between adherence to either a 'discretionary' or 'healthy' dietary pattern (as described by the SR) at ages 15 and 24 months and IQ at age 8 and 15 years. The 'discretionary' dietary pattern included consumption of foods such as biscuits, sweets and crisps. The 'healthy' dietary pattern included consumption of raw vegetables and fruit, cheese and herbs. The PCS reported that the 'discretionary' dietary pattern was associated with lower IQ at age 15 years but not at 8 years (quantitative findings NR) and that the 'healthy' dietary pattern was "weakly" associated with higher IQ at age 8 years but not at age 15 years (quantitative findings NR). The analyses were adjusted for maternal characteristics (age, education, SES, tobacco use during pregnancy), ethnicity, duration of breastfeeding.
- 5.156 Another study (in 1366 participants) using the same dataset as that described in paragraph 5.155 assessed the relationship between adherence to either a 'discretionary' dietary pattern or a 'nutrient-dense' dietary pattern (as described by the SR) at ages 15 and 24 months and Full-Scale Intelligence Quotient (FSIQ) or Verbal Intelligence Quotient (VIQ) at age 8 years. The PCS reported that higher scores for the 'discretionary' dietary pattern in early childhood were associated with lower FSIQ and VIQ and that higher scores for the 'nutrient-dense' dietary pattern in early childhood were associated with higher in FSIQ and VIQ (quantitative findings NR). The analyses were adjusted for maternal characteristics (age, education, SES, tobacco use during pregnancy), ethnicity and duration of breastfeeding.
- 5.157 One PCS (in 3966 participants) examined the relationship between adherence to a 'processed foods' dietary pattern (as described by the SR) at ages 3 and 4 years and IQ at age 8.5 years. The 'processed foods' dietary pattern (derived from principal components analysis) was characterised by higher consumption of foods high in fat or sugar and by processed and convenience foods. IQ was measured using the Wechsler Intelligence Scale for Children (WISC) Version III. The PCS reported that the dietary pattern at age 3 years was associated with a decrease in IQ at age 8.5 years (quantitative findings NR). The analysis was adjusted for age at WISC testing and WISC administrator, dietary pattern scores at that time point, breastfeeding duration, TDEI, maternal education, maternal social class, maternal age, housing tenure, life events, HOME score and all other dietary pattern scores.
- 5.158 One PCS (in 5741 participants) examined the relationship between adherence to either a 'junk food' dietary pattern or a 'health-conscious' dietary pattern (as described by the SR) at ages 38, 54 and 81 months and KS2 results (see Glossary) at ages 10 and 11 years. The 'junk food' dietary pattern was characterised by consumption of high-fat processed foods (sausages, burgers and poultry products), snack foods high in fat or sugar (such as crisps, sweets, chocolate, ice lollies and ice creams), 'fizzy drinks', and the number of takeaway meals eaten per month. The 'health-conscious' dietary pattern was characterised by vegetarian foods, nuts, salad, rice, pasta, fruit, cheese, fish, cereal, water and

fruit juice. The PCS reported that higher scores for a 'junk food' dietary pattern at age 38 months were associated with lower KS2 results (quantitative findings NR) but that adherence to the 'health-conscious' dietary pattern at age 38 months was not associated with KS2 results (quantitative findings NR). Results for ages 54 and 81 months were not reported. The analyses were adjusted for sex, ethnicity, birth order, various socioeconomic measures, and maternal behaviours, breastfeeding duration, television watching, an indicator of cognitive stimulation and emotional warmth in the home environment.

## Summary: diet quality and cognitive outcomes

5.159 The evidence identified from SRs on diet quality and cognitive outcomes in children aged 1 to 5 years is summarised in Table 5.32.

**Table 5.32 Summary of the evidence on diet quality and cognitive outcomes**

Exposure	Outcome	Direction of association	Certainty of evidence
Diet quality measured by score or index	Receptive vocabulary	Not applicable	Insufficient
Diet quality measured by score or index	Non-verbal vocabulary	Not applicable	Insufficient
'Healthy' dietary pattern <sup>1</sup>	Intelligence quotient (IQ)	Not applicable	Insufficient
'Healthy' dietary pattern <sup>1</sup>	Verbal IQ	Not applicable	Insufficient
'Healthy' dietary pattern <sup>1</sup>	Key stage 2 performance	Not applicable	Insufficient
'Unhealthy' dietary pattern <sup>1</sup>	IQ	Not applicable	Insufficient
'Unhealthy' dietary pattern <sup>1</sup>	Verbal IQ	Not applicable	Insufficient
'Unhealthy' dietary pattern <sup>1</sup>	Key stage 2 results	Not applicable	Insufficient

<sup>1</sup> Defined in paragraph 5.140.

5.160 The available evidence from SRs on the relationship between diet quality and cognitive outcomes in children aged 1 to 5 years is from 1 SR without MA given a critically low confidence rating using the AMSTAR 2 tool.

5.161 There was 'insufficient' evidence from SRs to enable conclusions to be drawn on any relationship between diet quality (assessed by score or index) and receptive

vocabulary and non-verbal vocabulary in children aged 1 to 5 years as fewer than 3 primary studies included in the SR examined these relationships.

- 5.162 There was also 'insufficient' evidence from SRs to enable conclusions to be drawn on any relationship between adherence to dietary patterns classified by SRs as 'healthy' and various measures of cognitive development in children aged 1 to 5 years as fewer than 3 primary studies included in the SR examined these relationships.
- 5.163 Although the SR by Tandon et al (2016) included 3 PCS that examined the relationship between adherence to a dietary pattern classified as 'unhealthy' and IQ, 2 of the 3 studies used a dataset from the same longitudinal cohort study. Because there were only 2 independent PCS, the evidence from this SR was graded 'insufficient' and no conclusions can be drawn on.

## **Other dietary patterns**

- 5.164 This section describes the evidence identified from SRs on dietary patterns that did not describe differences in 'diet quality' (defined on page 246). The dietary patterns covered in this section were derived using exploratory approaches and were labelled and defined by the SR or the primary study authors.
- 5.165 This section also provides a short introduction to vegetarian and vegan diets given the increasing popularity of these diets in the UK over recent years. However, no evidence from SRs was identified on these diets in children aged 1 to 5 years for this report.

## **Vegetarian and vegan diets**

- 5.166 Vegetarian and vegan diets have gained in popularity in recent years (Kiely, 2021; Schurmann et al, 2017). In the UK, the Vegan Society reported that the number of vegans practicing in the UK had increased 4-fold between 2014 and 2019 from 150,000 to 600,000 (The Vegan Society, 2022). In 2014, 0.25% of the UK population were reported to follow a vegan diet, whereas in 2019 it was 1.21% (The Vegan Society, 2022).
- 5.167 Vegetarian diets exclude foods derived from animal flesh, such as meat, poultry, seafood and their products; while vegan diets exclude all animal products, including foods that use ingredients derived from animal processing (such as gelatine) (Baroni et al, 2019; Kiely, 2021). Both vegetarian and vegan diets consist of a variety of plant-based foods such as vegetables and fruit, grains, pulses, nuts and seeds (Baroni et al, 2019).
- 5.168 Evidence suggests that well-planned vegetarian and vegan diets can meet the nutritional requirements of preschool children if sufficient care is taken (Baroni et al, 2019; Melina et al, 2016). Special attention needs to be given to protein

quantity and quality, and ensuring adequate intakes of iron, calcium, vitamin D, vitamin B12, iodine and n-3 fatty acids (Baroni et al, 2019; Schurmann et al, 2017) while avoiding excessive intakes of dietary fibre, which can reduce nutrient absorption in young children (Kiely, 2021). Vitamin B12 is especially important given that it is only found in animal products and therefore supplementation is essential among all those who avoid animal products (Baroni et al, 2019).

- 5.169 Young children are at particular risk of adverse effects from highly restrictive diets, such as unsupplemented vegan diets, because their energy and nutritional requirements are higher than the rest of the population due to their rapid growth and development. Highly restrictive unsupplemented diets can lead to poor nutrient intake and nutritional status. In extreme cases, these diets can cause long term malnutrition and adversely affect growth and development (Dagnelie & van Staveren, 1994; DH, 1994b).
- 5.170 Some observational evidence suggests that children following vegetarian diets have a lower risk of childhood obesity, a healthier blood lipid profile, and are leaner and taller in adolescence compared with children who are not vegetarians (Baroni et al, 2019; Sabaté & Wien, 2010). There is also some evidence that adult vegetarians have a lower risk of ischemic heart disease, obesity, diabetes and some cancers compared with adult non-vegetarians (Appleby & Key, 2016; Baroni et al, 2019; Leitzmann, 2014). Considering that dietary patterns in early childhood can track into older age (Craigie et al, 2011; Emmett et al, 2015; Hodder et al, 2018), this may be an important area of research. However, there are few well-characterised, controlled studies on the health effects of different types of vegetarian and vegan diets. It is also difficult to interpret population-level data of contemporary dietary practices as most of the evidence of adverse effects arises from case studies and case series (Kiely, 2021; Lemoine et al, 2020).
- 5.171 The European Society for Paediatric Gastroenterology, Hepatology and Nutrition (ESPGHAN) recommend close monitoring of child growth and development in vegetarian and vegan children (Fewtrell et al, 2017).

## **Systematic review evidence identified on other dietary patterns and health**

- 5.172 One SR without MA (Tandon et al, 2016) was identified that included studies that examined the relationship between adherence to dietary patterns variously described in the SR as 'snacking', 'ready-to-eat', 'freshly-cooked' and 'traditional' dietary patterns, and their relationship with cognitive development.
- 5.173 All primary studies included in the SRs were conducted in HICs.
- 5.174 Details of the SR included in this section can be found in Annex 5 (Table A5.3). Quality assessment of the SRs using the AMSTAR 2 tool can be found in Annex 8

(Table A8.5). Additional data extracted on the primary studies can be found in Annex 9 (Table A9.26).

- 5.175 The criteria used to grade the evidence are provided in chapter 2 (Table 2.4, paragraphs 2.54 to 2.59). Summary tables of the evidence grading process for this section are provided in Annex 10 (Table A10.36).

## **‘Snacking’ and IQ**

- 5.176 Tandon et al (2016) (AMSTAR 2 confidence rating: critically low) included 1 PCS (in 3966 participants) that examined the relationship between ‘snacking’ at ages 3 and 4 years and IQ at age 8.5 years. ‘Snacking’ was characterised by the SR as including foods such as fruit, biscuits, bread and cakes. IQ was measured using WISC Version III. The PCS reported that ‘snacking’ at age 3 years was associated with an increase in IQ at age 8.5 years (quantitative findings NR). The analyses were adjusted for age at WISC testing, dietary pattern scores at that time point, breastfeeding duration, TDEI, maternal characteristics and SES.

## **Summary: ‘Snacking’ and IQ**

- 5.177 The evidence identified from SRs on ‘snacking’ and IQ in children aged 1 to 5 years is summarised in Table 5.33.

**Table 5.33 Summary of the evidence on ‘snacking’ and IQ**

Exposure	Outcome	Direction of association	Certainty of evidence
‘Snacking’ <sup>1</sup>	Intelligence quotient (IQ)	Not applicable	Insufficient

<sup>1</sup>Defined and characterised by the SR (Tandon et al, 2016).

- 5.178 The available evidence from SRs on the relationship between ‘snacking’ and IQ in children aged 1 to 5 years is from 1 SR given a critically low confidence rating using the AMSTAR 2 tool. There was ‘insufficient’ evidence from SRs to enable conclusions to be drawn on any relationship between ‘snacking’ and IQ in children 1 to 5 years as fewer than 3 primary studies included in the SR examined these relationships.

## **‘Ready-to-eat or freshly cooked’ dietary patterns and IQ**

- 5.179 Tandon et al (2016) included 3 PCS that examined the relationship between adherence to ‘ready-to-eat’ or ‘freshly cooked’ dietary patterns by children aged 1 to 5 years and IQ.
- 5.180 One PCS (in 7652 participants) examined the relationship between adherence to a ‘ready-to-eat’ dietary pattern at ages 15 and 24 months and IQ at ages 8 and 15

years. The 'ready-to-eat' dietary pattern was characterised by the consumption of commercially manufactured foods marketed for children at age 15 months; and the consumption of biscuits, bread and breakfast cereals at age 24 months. The PCS reported no association between 'ready-to-eat' dietary pattern and IQ at any age (quantitative findings NR). The analysis was adjusted for maternal characteristics (age, education, SES, tobacco use during pregnancy), ethnicity, and duration of breastfeeding.

- 5.181 The second study (in 1366 participants), which used the same dataset as the study described in the previous (paragraph 5.180), examined the relationship between adherence to a 'ready-to-eat' or 'ready-to-eat baby foods' dietary pattern at ages 15 and 24 months and the FSIQ and VIQ at age 8 years. The study reported that adherence to the 'ready-to-eat' dietary pattern at age 24 months was associated with an increase in FSIQ and VIQ at age 8 years (quantitative findings NR) while adherence to the 'ready-to-eat baby foods' dietary pattern at age 15 months was associated with a decrease in FSIQ and VIQ at age 8 years (quantitative findings NR). The analyses were adjusted for maternal characteristics (age, education, SES, marital status, tobacco use) ethnicity.
- 5.182 One PCS (in 5217 participants) examined the relationship between adherence to a 'freshly cooked' dietary pattern and vocabulary and cognitive performance compared with a 'ready-to-eat' dietary pattern. Both exposure and outcomes were measured at ages 3 and 5 years. The 'freshly cooked' dietary pattern was characterised by 'slow meals' such as sit-down restaurant meals and meals cooked using fresh ingredients. The 'ready-to-eat' dietary pattern was characterised by 'fast' meals such as frozen, ready or takeaway meals. The PCS reported that a 'freshly cooked' dietary pattern at age 3 years was associated with an increase in vocabulary at age 3 and 5 years (quantitative findings NR) and higher cognitive performance at age 5 years (quantitative findings NR) compared with the 'ready-to-eat' dietary pattern. The analyses were adjusted for SES and cognitive ability from earlier assessments. It should be noted that consuming more 'slow' meals compared with 'fast' meals per week partially mediated the effect of SES on cognitive performance at ages 3 and 5 years.

### **Summary: 'ready-to-eat' or 'freshly cooked' dietary patterns and IQ**

- 5.183 The evidence identified from SRs on the relationship between adherence to 'ready-to-eat' or 'freshly cooked' dietary patterns and IQ in children aged 1 to 5 years is summarised in Table 5.34.

**Table 5.34 Summary of the evidence on ‘ready-to-eat or freshly cooked’ dietary patterns and cognitive outcomes**

Exposure	Outcome	Direction of association	Certainty of evidence
‘Ready-to-eat’ dietary pattern <sup>1</sup>	Intelligence quotient (IQ)	Not applicable	Insufficient
‘Ready-to-eat’ dietary pattern <sup>1</sup>	Verbal IQ	Not applicable	Insufficient
‘Ready prepared baby foods’ dietary pattern <sup>1</sup>	IQ	Not applicable	Insufficient
‘Ready prepared baby foods’ dietary pattern <sup>1</sup>	Verbal IQ	Not applicable	Insufficient
‘Freshly cooked’ dietary pattern <sup>1</sup>	Vocabulary	Not applicable	Insufficient
‘Freshly cooked’ dietary pattern <sup>1</sup>	Cognitive performance	Not applicable	Insufficient

<sup>1</sup> Defined and characterised by the SR (Tandon et al, 2016).

- 5.184 The available evidence from SRs on the relationship between adherence to ‘ready-to-eat’ or ‘freshly cooked’ dietary patterns and IQ in children aged 1 to 5 years is from 1 SR given a critically low confidence rating using the AMSTAR 2 tool.
- 5.185 There was ‘insufficient’ evidence from SRs to enable conclusions to be drawn on any relationship between ‘ready-to-eat’ or ‘freshly cooked’ dietary patterns and cognitive outcomes in children 1 to 5 years as fewer than 3 primary studies included in the SR examined these relationships.

### **‘Traditional’ dietary patterns and cognitive outcomes**

- 5.186 Tandon et al (2016) included 1 PCS that examined the relationship between adherence to a ‘traditional’ dietary pattern by children aged 1 to 5 years and IQ in adolescence. ‘Traditional’ dietary patterns were characterised by meat, cooked vegetables, and puddings.
- 5.187 The PCS (in 7652 participants) reported that adherence to a ‘traditional’ dietary pattern at ages 15 and 24 months was associated with lower IQ at age 15 years but not at age 8 years (quantitative findings NR). The analysis was adjusted for

maternal characteristics (age, education, SES, marital status, tobacco use during pregnancy) ethnicity, duration of breastfeeding.

## Summary: ‘traditional’ dietary patterns and IQ

- 5.188 The evidence identified from SRs on the relationship between adherence to a ‘traditional’ dietary pattern and IQ in children aged 1 to 5 years is summarised in Table 5.35.

**Table 5.35 Summary of the evidence on ‘traditional’ dietary patterns and IQ**

Exposure	Outcome	Direction of association	Certainty of evidence
‘Traditional’ dietary pattern <sup>1</sup>	Intelligence quotient (IQ)	Not applicable	Insufficient

<sup>1</sup> Defined and characterised by the SR (Tandon et al, 2016).

- 5.189 The available evidence from SRs on the relationship between a ‘traditional’ dietary pattern and IQ in children aged 1 to 5 years is from 1 SR given a critically low confidence rating using the AMSTAR 2 tool.
- 5.190 There was ‘insufficient’ evidence from SRs to enable conclusions to be drawn on any relationship between ‘traditional’ dietary patterns and IQ in children 1 to 5 years as fewer than 3 primary studies included in the SR examined these relationships.

# Dietary components

- 5.191 This section includes evidence from SRs on dietary (non-nutrient) components that were identified during the literature search process. Although there are no dietary recommendations for these components, they may have effects on health and development in young children and are therefore considered below.

## Probiotics

### Introduction

- 5.192 The term 'probiotic' is defined as 'live microorganisms that, when administered in adequate amounts, confer a health benefit on the host' (Hill et al, 2014). The most common microorganisms considered to be beneficial to health belong to the bacterial genera Lactobacilli and Bifidobacterium (Guarner et al, 2017; Zheng et al, 2020).
- 5.193 While the largest group of food products containing probiotics are fermented dairy products such as yoghurt, kefir and cheese (Douglas & Sanders, 2008), newer products containing probiotics have been developed, including granola bars, fruit juices and ice creams (Vandenplas et al, 2014).
- 5.194 Some infant formula and follow-on formula (see Glossary) are also supplemented with probiotics although little is known of their effects in young children. ESPGHAN reviewed the existing evidence on probiotics in infant and follow-on formula and concluded that there was a lack of data on long term health effects. Although the evidence suggests that probiotic-supplemented formula for healthy infants do not raise safety concerns, ESPGHAN does not recommend routine use of probiotic-supplemented formula (Braegger et al, 2011).
- 5.195 The current UK advice for preparing infant formula (including probiotic-supplemented formula) is to boil fresh tap water and let it cool for no more than 30 minutes in order that it remains at a temperature of at least 70 degrees Celsius (NHS, 2019b).
- 5.196 In Great Britain, individual strains of microorganism present in a product must be listed as ingredients but cannot be described as 'probiotic' unless a specific health claim (set out by the retained [Regulation \(EC\) No 1924/2006](#)) for that organism has been approved and added to the [Great Britain Nutrition and Health Claims \(GB NHC\) Register](#) (equivalent EU regulations apply in Northern Ireland). The product would also have to meet the specific conditions of use of any approved claim. There are currently no authorised claims for probiotic strains on the [GB NHC Register](#).

## **Systematic review evidence identified on probiotics and health**

- 5.197 One SR with MA (Onubi et al, 2015) was identified that included studies that examined the relationship between probiotics and growth (linear growth and weight gain).
- 5.198 Details of the SR included in this section can be found in Annex 5 (Table A5.3). Quality assessment of the SRs using the AMSTAR 2 tool can be found in Annex 8 (Table A8.6). Additional data extracted on the primary studies can be found in Annex 9 (Table A9.27).
- 5.199 The criteria used to grade the evidence are provided in chapter 2 (Table 2.4, paragraphs 2.54 to 2.59). Summary tables of the evidence grading process for this section are provided in Annex 10 (Table A10.36).

### **Probiotics and growth outcomes**

- 5.200 One SR without MA (Onubi et al, 2015) (AMSTAR 2 confidence rating: low) was identified that examined the relationship between probiotics and growth outcomes and included 2 RCTs that examined this relationship in children aged 1 to 5 years. One RCT was conducted in a high income country (HIC) and the other was conducted in an upper-middle income country (UMIC) (defined according to the [World Bank classification system](#)).
- 5.201 One RCT (in 131 participants from a HIC) examined the effect of probiotics in children aged 3 to 24 months on weight-for-age z-score (WHZ), weight-for-length z-score (WLZ) and height-for-age z-score (HAZ). The RCT had 2 intervention groups: one group received a high dose probiotic ( $1 \times 10^7$  Bifidobacterium lactis Bb12 and Streptococcus thermophilus CFU per gram) in a standard milk-based formula while the second group received a low dose probiotic ( $1 \times 10^6$  of the above) in a standard milk-based formula. The control group received a standard milk-based formula with no probiotics. The mean duration of the intervention was 210 days (SD 127 days). The RCT reported no difference in all assessed outcomes between both intervention groups compared to the control group (quantitative findings NR).
- 5.202 The second RCT (in 393 participants from an UMIC) examined the effect of probiotics in children aged 12 months on weight gain (per day), change in weight-for-age z-score (WAZ) and linear growth. The intervention group received a twice-daily dose of Bifidobacterium longum and Lactobacillus rhamnosus with 200ml milk, prebiotics and long-chain polyunsaturated fatty acids (LC-PUFA) alongside the child's 'normal diet' (terminology used in the primary study). The control group received 200ml milk twice daily with a 'normal diet'. The duration of the intervention was 12 months and outcomes were measured between ages 12 and

16 months. The RCT reported that children in the intervention group experienced greater daily weight gain (MD 0.93 grams per day; 95% CI 0.12 to 1.95; p=0.025) and change in WAZ (MD 0.09; 95% CI 0.01 to 0.18; p=0.06) compared with the control group. The SR reported that the changes in both weight gain and WAZ were greater than the growth standards recommended by the WHO for the age group. There was no difference reported in linear growth between groups (quantitative findings NR). As the study was conducted in an UMIC, the generalisability of the results to the UK population may be limited.

## Summary: probiotics and growth outcomes

5.203 The evidence identified from SRs on probiotics and growth outcomes in children aged 1 to 5 years is summarised in Table 5.36.

**Table 5.36 Summary of the evidence on probiotics and growth outcomes**

Exposure	Outcome	Direction of effect	Certainty of evidence
Probiotics	Change in body weight or weight-for-age z-score	Not applicable	Insufficient
Probiotics	Linear growth	Not applicable	Insufficient
Probiotics	Change in weight-for-length z-score	Not applicable	Insufficient

5.204 The available evidence from SRs on the relationship between probiotics and growth outcomes in children aged 1 to 5 years is from 1 SR given a low confidence rating using the AMSTAR 2 tool. There was 'insufficient' evidence from SRs to enable conclusions to be drawn on any relationship between probiotics and growth outcomes in children 1 to 5 years as fewer than 3 primary studies included in the SR examined this relationship.

# Low or no calorie sweeteners

## Introduction

- 5.205 Low or no calorie sweeteners (LNCS) are a range of artificial or nature-derived chemical substances that can be used to sweeten foods and drinks normally in place of using sugars and syrups (Sharma et al, 2016). LNCS include both high-potency and bulk sweeteners. High-potency sweeteners can deliver the sweetness of sugars when used in very small quantities with a negligible calorie content. Bulk sweeteners are used in larger quantities and have a sweetness potency closer to sugars, but with energy values ranging from 0 to 2 kcal per gram (Chattopadhyay et al, 2014; Dills, 1989).
- 5.206 LNCS approved for use in the UK include acesulfame K, aspartame, saccharin, sorbitol, sucralose, steviol glycosides, thaumatin and xylitol (FSA, 2022; NHS, 2019c). The Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT) concluded that the exposures in the diet of children aged 1 to 5 years of the most commonly used sweeteners in the UK (aspartame, acesulfame K, saccharine, sorbitol and xylitol, stevia and sucralose) were not of toxicological concern (COT, 2020).
- 5.207 In principle, if compensatory energy intake is avoided, consumption of foods and drinks sweetened by LNCS could contribute to a reduction in energy intake from free sugars (Rogers et al, 2016).
- 5.208 There is a lack of agreed terminology in the discourse around LNCS. In the section below, the term 'non-nutritive sweeteners' was used when describing SR evidence because this was the terminology used in the SR literature.

## Systematic review evidence identified on low or no calorie ('non-nutritive') sweeteners and health

- 5.209 For this report, 1 SR with MA (Karalexi et al, 2018) was identified that examined the relationship between consumption of 'non-nutritive sweeteners' (terminology used in the SR) and various metabolic health outcomes. An additional SR with MA (World Health Organization et al, 2022) that examined the health effects of the use of 'non-sugar sweeteners' (terminology used by the SR) was identified through the public consultation process and also considered by SACN. However, SACN concluded that the SR did not provide sufficient additional evidence in children aged 1 to 5 years to warrant inclusion in the main report. Therefore, only the findings from Karalexi et al (2018) are described below. Further information on

World Health Organization et al (2022) is provided in Annex 6 (Table A6.1), Annex 9 (Table A9.28) and Annex 10 (A10.13).

- 5.210 All primary studies included in Karalexi et al (2018) were conducted in HIC.
- 5.211 Details of the SR can be found in Annex 5 (Table A5.3). Quality assessment of the SRs using the AMSTAR 2 tool can be found in Annex 8 (Table A8.6). Additional data extracted on the primary studies can be found in Annex 9 (Table A9.28).

### **Low or no calorie ('non-nutritive') sweeteners and BMI**

- 5.212 Karalexi et al (2018) (AMSTAR 2 confidence rating: critically low) included 2 PCS that examined the relationship between consumption of 'non-nutritive sweeteners' in children aged 1 to 5 years and BMI (or BMI z-score). The exposure in both studies was described in the SR as 'diet soda'. Both studies (in a total of 1522 participants) reported no association between consumption of diet soda in children aged 3 to 6 years and BMI (or BMI z-score) after 6 months to 3 years of follow up. For one of the PCS (in 1345 participants), the SR authors calculated a new estimate of association (odds ratio) for pooling into a MA but it was unclear whether this estimate was crude or adjusted. The other PCS (in 177 participants) adjusted for TDEI at age 3 years (see chapter 3, paragraphs 3.48 and 3.49).

### **Summary: low or no calorie ('non-nutritive') sweeteners and BMI**

- 5.213 The evidence identified from SRs on consumption of low or no calorie ('non-nutritive') sweeteners and BMI in children aged 1 to 5 years is summarised in Table 5.37.

**Table 5.37 Summary of the evidence on low or no calorie ('non-nutritive') sweeteners and obesity outcomes**

Exposure	Outcome	Direction of association	Certainty of evidence
Low or no calorie ('non-nutritive') sweeteners (in drinks)	Body Mass Index (BMI) or BMI z-score	Not applicable	Insufficient

- 5.214 The available evidence from SRs on the relationship between low or no calorie ('non-nutritive') sweeteners and BMI in children aged 1 to 5 years is from 1 SR given a low confidence rating using the AMSTAR 2 tool. There was 'insufficient' evidence from SRs to enable conclusions to be drawn on any relationship between low or no calorie ('non-nutritive') sweeteners and BMI (BMI z-score) in children 1 to

5 years as fewer than 3 primary studies included in the SR examined this relationship.

## **Low or no calorie ('non-nutritive') sweeteners and type 1 diabetes**

5.215 One SR without MA (Karalexi et al, 2018) (AMSTAR 2 confidence rating: critically low) included 1 PCS (in 2547 participants) that examined the relationship between 'non-nutritive sweeteners' and predictors of type 1 diabetes (islet autoimmunity and progression to type 1 diabetes) in children (baseline mean age 2 years). The PCS reported no association with islet immunity and progression to type 1 diabetes after 10.2 years' follow up. The analysis adjusted for a genotype associated with autoimmune diseases (see Annex 9, Table A9.28 for details), type 1 diabetes family history, ethnicity (non-Hispanic white vs other), diet survey type (food frequency questionnaire or Young Adolescent Questionnaire) and TDEI. It should be noted that the study included children at increased risk of developing type 1 diabetes.

### **Summary: low or no calorie ('non-nutritive') sweeteners and type 1 diabetes**

5.216 The evidence identified from SRs on low or no calorie 'non-nutritive' sweeteners and outcomes related to type 1 diabetes in children aged 1 to 5 years is summarised in Table 5.38.

**Table 5.38 Summary of the evidence on low or no calorie ('non-nutritive') sweeteners and type 1 diabetes**

Exposure	Outcome	Direction of association	Certainty of evidence
Low or no calorie ('non-nutritive') sweeteners	Islet autoimmunity	Not applicable	Insufficient
Low or no calorie ('non-nutritive') sweeteners	Progression to type 1 diabetes	Not applicable	Insufficient

5.217 The available evidence from SRs on the relationship between low or no calorie ('non-nutritive') sweeteners and outcomes related to type 1 diabetes in children aged 1 to 5 years is from 1 SR given a low confidence rating using the AMSTAR 2 tool. There was 'insufficient' evidence from SRs to enable conclusions to be drawn on any relationship between low or no calorie ('non-nutritive') sweeteners and type 1 diabetes related outcomes in children 1 to 5 years as fewer than 3 primary studies included in the SR examined this relationship.

# 6 Drinks

## Background

- 6.1 This chapter examines the evidence identified on breastfeeding beyond the first year of life, as well as drinks that are commonly consumed by young children in the UK.
- 6.2 This chapter covers
- breast milk
  - formula milks (infant formula, follow-on formula and milks marketed to children over the age of 1 year, also known as ‘toddler milks’ and ‘growing-up milks’)
  - milk (specifically cows’ milk)
  - 100% fruit juice (with no added sugars or sweeteners)
  - sugar-sweetened beverages (SSBs).
- 6.3 Nutritional and toxicological aspects associated with the consumption of plant-based drinks by children aged 1 to 5 years are being considered in a joint risk assessment being undertaken by SACN and COT. Findings are expected to be published in 2024 and will include recommendations on plant-based drink consumption. More information on the work of the joint SACN-COT working group is available [here](#).
- 6.4 For the purposes of this report, SSBs are any beverages (carbonated drinks, fruit-based drinks, squashes, flavoured water) where free sugars have been specifically added as a sweetener (excluding formula milks, which, in this report are treated as a separate category). Where possible, these are distinguished from 100% fruit juices (with naturally occurring levels of sugars) in the assessment of the evidence and the report’s recommendations (see [Recommendations](#)).
- 6.5 Commercially manufactured drinks that are marketed specifically for children aged up to 36 months are covered in chapter 5 (see [Foods and drinks marketed specifically for infants and young children](#)).
- 6.6 This chapter covers all health outcomes for which systematic review (SR) evidence was identified but excludes oral health, which is covered in chapter 9 (see [Oral Health](#)).

# Breastfeeding beyond the first year of life

- 6.7 The composition of breast milk varies between and within women, in part according to the changing needs of the developing child. Reliable sampling of breast milk for comparative studies can be challenging due to changes in composition during and between feeds (Leghi et al, 2020). A large FAO and WHO-commissioned SR with meta-analysis (MA) of studies that examined calcium, zinc and vitamin D concentrations in breast milk found that calcium concentrations were almost constant from birth, with a very slow decline into the second year of an infant's life; while zinc concentrations declined rapidly in the first 100 days before reaching a plateau (Rios-Leyvraz & Yao, 2023). Data were insufficient to do a similar analysis for vitamin D and 25-hydroxyvitamin D, the major circulating metabolite of vitamin D (see [Vitamin D](#) in chapter 4).
- 6.8 In its report 'Feeding in the first year of life', SACN reiterated its support for longstanding advice to breastfeed exclusively for around the first 6 months of an infant's life and to continue breastfeeding for at least the first year of life alongside the introduction of a wide range of solid foods in an age-appropriate form from around age 6 months (SACN, 2018). The WHO additionally recommends continued breastfeeding up to 2 years of age or beyond (WHO, 2021).
- 6.9 The last UK-wide Infant Feeding Survey (IFS), which was conducted in 2010, indicated that only 8% of children aged over 1 year consumed breast milk (McAndrew et al, 2012). At the time of publication of the current report, work was underway on a new IFS, which would provide updated data on breastfeeding rates for England.
- 6.10 Breastfeeding rates are monitored regularly but at different time points by the 4 UK countries. Scotland, Northern Ireland and Wales collect data at birth or soon after, then again at 6 to 8 weeks, 3 months (Northern Ireland), and 6 months (Wales and Northern Ireland). England collects data only at 6 to 8 weeks after birth.
- 6.11 Only Scotland and Northern Ireland collect breastfeeding data into the second year of life (OHID, 2023; Public Health Agency, 2022; Public Health Scotland, 2022a; StatsWales, 2022). Data from Public Health Scotland for 2021 to 2022 indicated that 22% of children aged 13 to 15 months were still receiving breast milk (Public Health Scotland, 2022a). Data from the Northern Ireland Public Health Agency indicated that 11.2% of children born in 2020 were still receiving breast milk at age 12 months (Public Health Agency, 2022).
- 6.12 Data from the Diet and Nutrition Survey in Infants and Young Children (DNSIYC) indicated that 8% of children aged 12 to 18 months received breast milk while average consumption was 290 grams per day. Breast milk provided approximately 2% of total energy intake in children (including non-consumers) in this age group.

- 6.13 In 2018, SACN recommended that, given the rapid decline in the proportion of women breastfeeding over the first few weeks of an infant's life in the UK, greater focus should be given to reducing attrition rates and supporting women who make the informed choice to breastfeed (SACN, 2018). The context of breastfeeding and continued breastfeeding is shaped by the complex interplay of maternal-infant or child attributes; sociocultural factors (including changing social attitudes towards breastfeeding); marketing practices of the formula milk industry; and social and structural barriers within workplaces, healthcare systems, and the wider built environment (Rollins et al, 2016).

## **Systematic review evidence identified on breastfeeding beyond the first year of life and health**

- 6.14 One SR with MA (Delgado & Matijasevich, 2013) was identified that included studies that examined the health impact of breastfeeding beyond the first year of life.
- 6.15 Key outcomes examined were measures of growth (weight gain and linear growth) and cognitive development. SR evidence identified on breastfeeding beyond the first year of life and oral health is covered in chapter 9 (see [Continued breastfeeding and development of dental caries](#)).
- 6.16 Details of the SR included in this section can be found in Annex 5 (Table A5.3). Quality assessment of the SRs using the AMSTAR 2 tool can be found in Annex 8 (Table A8.7). Additional data extracted on the primary studies can be found in Annex 9 (Table A9.29).
- 6.17 The criteria used to grade the evidence are provided in chapter 2 (Table 2.4, paragraphs 2.54 to 2.59). Summary tables of the evidence grading process for this section are provided in Annex 10 (Tables A10.36).
- 6.18 All primary studies included in the SRs were conducted in lower middle income countries (LMICs) and low income countries (LICs) (defined according to the [World Bank classification system](#)). Therefore, the generalisability of findings from these studies to the UK context may be limited.
- 6.19 None of the studies accounted for possible confounding by other aspects of the diet on growth and cognitive development. This is particularly important in the UK context given that breast milk makes only a small contribution to the diet of children aged 1 year and older (see paragraph 6.12).

## **Breastfeeding beyond the first year of life and growth**

- 6.20 One SR with MA (Delgado & Matijasevich, 2013) (AMSTAR 2 confidence rating: critically low) included 2 PCS that examined the association between breastfeeding beyond the first year of life and growth (weight gain or linear growth).
- 6.21 One PCS (in 28,753 participants) reported that children breastfed for at least 2 years gained less weight between ages 2 to 3 years than children who were breastfed for less than 2 years. This relationship was modified by household wealth and maternal education. Children from poor households who were breastfed for at least 2 years gained less weight than children from wealthier households who were breastfed for at least 2 years (MD -205g; 95% CI -279g to -131g versus MD -38g; 95% CI -106g to 30g). Similarly, children of mothers who had a lower level of education who were breastfed for at least 2 years gained less weight than breastfed children of mothers with a higher level of education (MD -133g; 95% CI -193g to -74g versus MD -88g; 95% CI -179g to 4g). It should be noted that the analyses combined children of healthy and low nutritional status (wasting or stunting). Analyses were adjusted for various baseline variables including child age, sex, dietary vitamin A intake, morbidity, household wealth, maternal literacy, availability of water in the house.
- 6.22 One PCS (in 443 participants) reported that children aged 21 to 26 months who were breastfed over the following 6 months experienced greater linear growth over this period than children who had stopped receiving breast milk before this period (MD 0.7cm; SD 0.3cm;  $p < 0.05$ ). Analyses were adjusted for a season (wet or dry), quality of housing, initial age and weight. Housing quality was a key modifier in this association. Breastfeeding was associated with greater linear growth among children living in poor housing, while the opposite association was observed in children living in adequate housing (that is, lower linear growth in children who were still being breastfed compared with those who were no longer being breastfed).

## **Summary: breastfeeding beyond the first year of life and growth**

- 6.23 The evidence identified from SRs on breastfeeding beyond the first year of life and growth is summarised in Table 6.1.

**Table 6.1 Summary of the evidence on breastfeeding beyond 12 months of age and growth**

Exposure	Outcome	Direction of association	Certainty of evidence
Breastfeeding beyond 12 months	Weight gain	Not applicable	Insufficient
Breastfeeding beyond 12 months	Linear growth	Not applicable	Insufficient

- 6.24 The available evidence from SRs on the relationship between breastfeeding beyond the first year of life and growth is from 1 SR given a critically low confidence rating using the AMSTAR 2 tool.
- 6.25 There was insufficient evidence from SRs to enable conclusions to be drawn on any relationship between breastfeeding beyond the first year of life and growth in children 1 to 5 years as fewer than 3 primary studies included in SRs examined these relationships.

## **Breastfeeding beyond the first year of life and cognitive and psychosocial development**

- 6.26 One SR with MA (Delgado & Matijasevich, 2013) (AMSTAR 2 confidence rating: critically low) included 2 PCS that examined the relationship between breastfeeding for 2 years and beyond and cognitive and psychosocial development.
- 6.27 One PCS (in 1979 participants) reported no association between breastfeeding for 2 years or more compared with breastfeeding for less than 6 months and cognitive development as measured by cognitive ability score at ages 8.5 years and 11.5 years (quantitative findings NR). The analyses were adjusted for sex, various measures of SES, maternal age, maternal alcohol use in pregnancy and preterm status of child.
- 6.28 The second PCS (in 2752 participants) reported no difference in psychosocial developmental scores at ages 5 to 6 years between children who were breastfed for 2 years or more compared with children who were breastfed for less than 6 months. The analysis was adjusted for sex, day-care attendance, maternal education, father's presence in the home, hygiene and non-income-producing assets.

## Summary: breastfeeding beyond the first year of life and cognitive and psychosocial development

- 6.29 The evidence identified from SRs on breastfeeding beyond the first year of life and cognitive and psychosocial development is summarised in Table 6.2.

**Table 6.2 Summary of the evidence on breastfeeding beyond 12 months of age and growth and cognitive and psychosocial outcomes**

Exposure	Outcome	Direction of association	Certainty of evidence
Breastfeeding beyond 12 months	Cognitive development	Not applicable	Insufficient
Breastfeeding beyond 12 months	Psychosocial development	Not applicable	Insufficient

- 6.30 The available evidence from SRs on the relationship between breastfeeding beyond the first year of life and cognitive outcomes is from 1 SR given a critically low confidence rating using the AMSTAR 2 tool. There was 'insufficient' evidence from SRs to enable conclusions to be drawn on any relationship between breastfeeding beyond the first year of life and cognitive outcomes in children aged 1 to 5 years as fewer than 3 primary studies included in SRs examined these relationships.

## Use of formula milks beyond the first year of life

### Types of formula milks

- 6.31 For the purposes of this report, 'formula milks' are used to describe (first) infant formula, follow-on formula and milks specifically marketed for children aged 1 year and over.
- 6.32 In the UK, it is recommended that infant formula (based on either cows' or goats' milk) is the only suitable alternative to breast milk for healthy children aged under 12 months (SACN, 2018). Infants diagnosed with cows' milk allergy may be given specialised formulas but only under medical supervision. Once children reach age 12 months, infant formula (including specialised formulas) are not needed

(NHS.uk). Follow-on formula, which is marketed specifically for children aged 6 months and older, and milks marketed to children over the age of 1 year, also known as ‘toddler milks’ and ‘growing-up milks’, are also not needed (NHS, 2023b).

- 6.33 While the composition, labelling and marketing of infant formula and follow-on formula are regulated in Great Britain (GB) by [Commission Delegated Regulation \(EU\) 2016/127](#) (this regulation was retained and amended as GB law after the UK left the EU; Northern Ireland are still required to follow the equivalent EU legislation), there is currently no regulation on the composition, labelling or marketing of milks marketed specifically for children aged 12 months and over.
- 6.34 The Commission Delegated Regulation 2016/127 and the overarching Foods for Specific Groups Regulation 609/2013 together give effect to some but not all of the general principles and ambitions of the [1981 WHO Code on the Marketing of Breast milk Substitutes](#) covering marketing, information and responsibilities of health authorities in relation to infant formula and follow-on formula, as they set provisions which regulate labelling and restrict advertising and presentation of infant and follow-on formula so as not to discourage breastfeeding.
- 6.35 ‘Growing-up milks’ and other milks specifically marketed for children aged 1 year and over are mainly composed of powdered milk or individual milk components, vegetable oils, and free sugars (First Steps Nutrition Trust, 2021). The carbohydrate source of these milks is usually maltodextrins (produced from starch from maize or potatoes), which are easily hydrolysed in the mouth to free sugars (see [Classification of carbohydrates](#)) by salivary amylase, and the addition of lactose (First Steps Nutrition Trust, 2021). Lactose that is naturally present in cows’ milk is not classified as a free sugar, while lactose that is added to a product is (Swan et al, 2018). Specialised formula milks, developed as alternatives to cows’ milk-based formula, also contain higher amounts of free sugars (mainly glucose or sucrose) than standard first infant formula (Mehta et al, 2022).
- 6.36 Globally, the growth in sales of formula milks have been driven by the growth in sales of ‘growing-up’ or ‘toddler’ milks. Between 2005 and 2019, sales of ‘growing-up’ or ‘toddler’ milks more than trebled between 2005 and 2019, while in high income countries, sales of these milks grew by 148% over this period (Baker et al, 2021). In addition, a recent cross-sectional analysis of national prescription data found that prescribed volumes of specialised formula milks increased nearly 3-fold in England between 2007 and 2018, which is well above the expected level given an approximate 1% incidence of cows’ milk allergy in children under the age of 2 years (Mehta et al, 2022). This analysis suggests that specialised formula milks are being overprescribed with unknown short- and long-term health consequences.

## Use of formula milks in the UK

- 6.37 Data from DNSIYC and NDNS (years 2016 to 2019) on the consumption and contribution of formula milks to the diets of children aged 12 to 60 months in the UK are presented in Table 6.3 to 6.4. Values that include non-consumers provide an estimate of the overall contribution of formula milks to the diets of young children, while values in consumers provide an estimate of the quantities consumed.
- 6.38 Formula milks were consumed by 36% of children aged 12 to 18 months and 7% of children aged 18 to 47 months. There were no consumers in the 48 to 60 month age group. For children aged 12 to 18 months, mean consumption was 365 grams per day for consumers (133 grams per day at a population level).

**Table 6.3 Formula milks consumption (grams per day and contribution to TDEI) in children aged 12 to 18 months in the UK (DNSIYC)<sup>1</sup>**

Food group	Mean consumption (SD) in grams per day Includes non-consumers	% Contribution to TDEI Includes non-consumers	% (number) of consumers over 4 days	Mean consumption (SD) in grams per day Consumers only	% Contribution to TDEI Consumers only
Formula milks <sup>2</sup>	133 (211)	9.8	36 (454)	365 (194)	26.9
Number of participants	1275	1275	1275	1275	1275

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake.

<sup>1</sup> Data from DNSIYC 2011 (Lennox et al, 2013).

<sup>2</sup> Formula milks include milks marketed to children over the age of 1 year, also known as 'toddler milks' and 'growing-up milks' (see Glossary).

**Table 6.4 Formula milks consumption (grams per day and contribution to TDEI) in children aged 18 to 47 months in the UK (NDNS years 2016 to 2019)<sup>1</sup>**

Food group	Mean consumption (SD) in grams per day Includes non-consumers	% Contribution to TDEI Includes non-consumers	% (number) of consumers over 4 days	Mean consumption (SD) in grams per day Consumers only	% Contribution to TDEI Consumers only
Formula milks <sup>2</sup>	18.5 (80.4)	1.1	7 (18)	No data <30 consumers	No data <30 consumers
Number of participants	306	306	306	306	306

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake.

<sup>1</sup> Data from NDNS years 2016 to 2019.

<sup>2</sup> Formula milks include milks marketed to children over the age of 1 year, also known as ‘toddler milks’ and ‘growing-up milks’ (see Glossary).

- 6.39 Formula milks provided 27% of total dietary energy intake (TDEI) in consumers aged 12 to 18 months (10% TDEI at population level). This is despite current recommendations that formula milks are not needed once children reach 12 months of age. For children aged 18 to 47 months, formula milks provided 1% TDEI at population level (see chapter 3, Table 3.5). Children aged 48 to 60 months did not consume formula milks.
- 6.40 Formula milks made a sizeable contribution to protein and free sugars intakes in children aged 12 to 47 months (Table 6.5 and Table 6.6).

**Table 6.5 Formula milks consumption (mainly follow-on formula and ‘growing up’ milks) to protein and free sugars intakes in children aged 12 to 18 months in the UK<sup>1</sup>**

	% Contribution to free sugars intake	% Contribution to protein intake
Including non-consumers	18.1	6.6
In consumers only <sup>2</sup>	49.8	18.0

<sup>1</sup> Data in children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013).

<sup>2</sup> There were 454 consumers of formula milks (mainly follow-on formula and ‘growing up’ milks).

**Table 6.6 Formula milks consumption (mainly follow-on formula and milks marketed to children over the age of 1 year, also known as ‘toddler milks’ and ‘growing-up milks’) to protein and free sugars intakes in children aged 18 to 47 months in the UK<sup>1</sup>**

	% Contribution to free sugars intake	% Contribution to protein intake
Including non-consumers	2.8	0.8
In consumers only <sup>2</sup>	No data <30 consumers	No data <30 consumers

<sup>1</sup> Data from NDNS years 2016 to 2019.

<sup>2</sup> There were 18 consumers of formula milks (mainly follow-on formula and ‘growing up’ milks).

- 6.41 For children aged 12 to 18 months, consumers of formula milks (36% of this age group) obtained approximately 50% of their free sugars intake from these products

- 6.42 In children aged 12 to 18 months, formula milks provided 18.0% of daily protein intake in consumers (6.6% at a population level).
- 6.43 Formula milks also made an appreciable contribution to micronutrient intakes in young children. Secondary analysis of NDNS data (year 2008 to 2019) indicated that for children aged 18 to 47 months with iron, zinc and vitamin A intakes that were at or above the dietary recommendations for these nutrients, formula milks provided 10% and 11% of daily iron and zinc intake, respectively, and 9% of daily vitamin A intake (see [Dietary contributors to iron, zinc and vitamin A](#), Table 4.1, 4.3 and 4.5).
- 6.44 It is currently recommended that children aged 6 months to 5 years are given dietary supplements containing vitamins A, C and D except when they consume more than 500ml of formula milk (including follow-on formula and 'growing-up' milks) per day because formula milks are fortified with vitamins A, C and D and other nutrients, including iron and zinc. Children who consume both formula milk and dietary supplements may be at risk of excess intakes of some micronutrients.

## Milk (excluding formula milks)

- 6.45 In this report, 'milk' is used to refer to cows' milk (excluding formula milks). This is in line with European Union regulations that define 'milk' as a mammary secretion of animals obtained from milking, with the most common type being cows' milk (Dougkas et al, 2019). 'Milk' is a protected term (Dougkas et al, 2019).
- 6.46 Milk is a rich source of energy, protein, calcium, vitamin A, riboflavin, iodine, phosphorus, potassium, zinc and vitamin B12, although the exact nutrient composition is dependent on the type of milk, geographical location, season, diet of the animals and husbandry practices (Dougkas et al, 2019; Haug et al, 2007; NHS, 2019a). Milk contains lactose, a sugar that is naturally present in milk and dairy products (Swan et al, 2018).
- 6.47 The current UK recommendation is that children from the age of 1 year can be given whole cows' milk as a main drink, and that from age 2 years, semi-skimmed milk can be gradually introduced to children who are growing well. It is advised that young children should not be given unpasteurised milk because of the higher risk of food poisoning (NHS, 2022).

## Milk consumption in the UK

- 6.48 Data from DNSIYC and NDNS (years 2016 to 2019) on the consumption and contribution of milk to the diets of children aged 12 to 60 months in the UK are presented in Table 6.7 to 6.9. Values that include non-consumers provide an

estimate of the overall contribution of milk to the diets of young children, while values in consumers provide an estimate of the quantities consumed.

- 6.49 Ninety percent of children aged 12 to 18 months consumed milk (which includes all types of cows' milk and other dairy milks) over the 4 day survey period while 96% to 97% of children in the older age groups consumed milk.
- 6.50 At a population level, milk provided 19.0% TDEI, 14.8% TDEI and 9.6% TDEI in children aged 12 to 18 months, 18 to 47 months and 48 to 60 months, respectively. In consumers only, milk provided 21.2% TDEI, 22.3% TDEI and 15.2% TDEI, respectively.

**Table 6.7 Milk consumption (grams per day and contribution to TDEI) in children aged 12 to 18 months in the UK (DNSIYC)<sup>1</sup>**

Food group	Mean consumption (SD) in grams per day Includes non-consumers	% Contribution to TDEI Includes non-consumers	% (number) of consumers over 4 days	Mean consumption (SD) in grams per day Consumers only	% Contribution to TDEI Consumers only
Whole milk (3.8% fat)	258 (237)	17.4	79 (1008)	329 (220)	22.2
Semi-skimmed milk (1.8% fat)	23 (100)	1.1	13 (169)	169 (223)	8.5
Skimmed milk (0.1% fat)	0.9 (17.9)	0.0	1.1 (17)	No data <30 consumers	No data <30 consumers
Total milk <sup>2</sup>	289 (236)	19.0	90 (1149)	322 (228)	21.2

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake.

<sup>1</sup> Data from DNSIYC 2011 (Lennox et al, 2013). Number of participants = 1275

<sup>2</sup> All types of cows' milk and other dairy milks.

**Table 6.8 Milk consumption (grams per day and contribution to TDEI) in children aged 18 to 47 months in the UK (NDNS years 2016 to 2019)<sup>1</sup>**

Food group	Mean consumption (SD) in grams per day Includes non-consumers	% Contribution to TDEI Includes non-consumers	% (number) of consumers over 4 days	Mean consumption (SD) in grams per day Consumers only	% Contribution to TDEI Consumers only
Whole milk (3.8% fat)	185 (204)	11.4	68 (209)	274 (193)	16.8
Semi-skimmed milk (1.8% fat)	56 (120)	2.4	38 (119)	149 (156)	6.4
Skimmed milk (0.1% fat)	0.6 (10)	0.0	1 (4)	No data <30 consumers	No data <30 consumers
Total milk <sup>2</sup>	311 (213)	14.8	96 (295)	322 (208)	22.3

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake.

<sup>1</sup> Data from NDNS years 2016 to 2019. Number of participants = 306

<sup>2</sup> All types of cows' milk and other dairy milks

**Table 6.9 Milk consumption (grams per day and contribution to TDEI) in children aged 48 to 60 months in the UK (NDNS years 2016 to 2019)<sup>1</sup>**

Food group	Mean consumption (SD) in grams per day Includes non-consumers	% Contribution to TDEI Includes non-consumers	% (number) of consumers over 4 days	Mean consumption (SD) in grams per day Consumers only	% Contribution to TDEI Consumers only
Whole milk (3.8% fat)	83 (157)	4.0	45 (50)	186 (190)	8.9
Semi-skimmed milk (1.8% fat)	125 (196)	4.9	64 (65)	198 (215)	7.7
Skimmed milk (0.1% fat)	3 (17)	0.1	2 (3)	No data <30 consumers	No data <30 consumers
Total milk <sup>2</sup>	271 (216)	9.6	97 (99)	281 (214)	15.2

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake.

<sup>1</sup> Data from NDNS years 2016 to 2019. Number of participants =102

<sup>2</sup> All types of cows' milk and other dairy milks.

## Milk consumption by deprivation

- 6.51 Total milk consumption by index of multiple deprivation (IMD) (see Glossary) in children aged 18 to 60 months (including non-consumers) is presented in Table 6.10.
- 6.52 Total milk consumption was highest (288 grams per day) in quintile 1 (least deprived) and lowest in quintiles 4 and 5 (most deprived). However, there was no clear relationship between total milk consumption and IMD (as indicated by overlapping confidence intervals).

**Table 6.10 Total milk consumption by IMD quintile in children aged 18 to 60 months in England (NDNS years 2008 to 2019)<sup>1</sup>**

Consumption Mean (95%CI)	IMD quintile 1 (least deprived)	IMD quintile 2	IMD quintile 3	IMD quintile 4	IMD quintile 5 (most deprived)
Total milk <sup>1</sup> (grams per day)	288 (257 to 319)	271 (243 to 298)	266 (238 to 294)	236 (210 to 262)	237 (258 to 306)
Number of participants	210	212	182	234	277

Abbreviations: IMD, index of multiple deprivation.

<sup>1</sup> Data from NDNS years 2008 to 2019. Includes non-consumers.

<sup>2</sup> Total milk - all types of cows' milk and other dairy milk.

## Milk substitution analyses

- 6.53 Data from DNSIYC were used to model the potential impact on average TDEI and selected nutrients of substituting semi-skimmed cows' milk, 1% fat cows' milk and skimmed cows' milk for whole cows' milk in the diets of children aged 12 to 18 months. These milk substitution analyses considered only milk consumed as a drink or on breakfast cereals. Milk consumed as part of composite recipe dishes and milk products such as cheese and yoghurt, and dried milk were excluded from the analyses. Average nutrient compositions for whole, semi-skimmed, 1% fat and skimmed milks were obtained as average pasteurised values from the Composition of Foods (PHE, 2021a), which takes account of summer and winter values (Annex 12, Table A12.1).
- 6.54 Detailed results are presented in Annex 12 (Tables A12.2 to A12.4). These tables present the results of substituting each lower fat milk type for whole milk for the group as a whole and for high and low milk consumers, using the 5th and 95th percentile to define high and low consumers. Annex 12, Tables A12.5 to A12.7 use

the 5th and 95th percentiles of overall TDEI to present results in children with the highest and lowest TDEI.

- 6.55 In examining the impact of the substitution on high and low consumers of milk, mean and median intakes of all nutrients were above the reference nutrient intake (RNI) or estimated average requirement (EAR) in the case of TDEI, before and after substituting for each type of milk. This remained the case in the highest and lowest consumers of milk and in those reporting no consumption of milk. Intakes of total fat and saturated fats following substitution fell in all groups reporting milk consumption. As expected, this drop was most marked in groups that reported the highest milk consumption Annex 12 (Tables A12.2 to A12.4).
- 6.56 In examining the impact of the substitution on children with high and low TDEI, children with the lowest 5% TDEI were below the EAR for energy intake before and after substitution. Mean intakes of calcium, iodine, vitamin A and riboflavin remained above the RNI after substituting each type of milk for whole milk, in children with high and low TDEI (Tables A12.5 to A12.7).
- 6.57 The milk substitution analysis indicated that replacing whole cows' milk with semi-skimmed cows' milk in children aged 12 to 18 months would be unlikely to have a detrimental effect on nutrient intakes at the population level. However, switching from whole to semi-skimmed milk may have an impact on excess TDEI, although this is not certain because consumption of other foods might increase to conserve overall TDEI.
- 6.58 In contrast, the milk substitution analysis indicated that a move from whole milk to skimmed or 1% milk would result in a greater proportion of children below the LRNI for vitamin A in all groups of TDEI, with the greatest impact in children with the highest milk consumption (Table A12.11) and lowest TDEI (Table A12.12).

## **Systematic review evidence identified on milk consumption and health**

- 6.59 One SR without MA (Dougkas et al, 2019) was identified that included studies that examined the relationship between consuming milk, including milks with different fat content, and body composition and weight status. An additional SR with MA (Vanderhout et al, 2020) that also compared consumption of milks with different fat content on weight status was identified for consideration after the public consultation on the draft report. However, the SR did not provide sufficient additional evidence to warrant inclusion in the main report. Information on Vanderhout et al (2020) is provided in Annex 6 (Table A6.1) and Annex 10 (Table A10.15). Findings from Dougkas et al (2019) are described below.

- 6.60 Dougkas et al (2019) did not specify whether ‘milk’ was of bovine origin only. However, all primary studies included in the SR referred to milk as ‘dairy’ and not as dairy alternatives.
- 6.61 Key outcomes were body composition (BMI, body weight, body fat) and weight status (overweight). For SR evidence on milk consumption and oral health, see [Milk consumption and oral health](#) in chapter 9.
- 6.62 All primary studies included in the SR were conducted in HIC.
- 6.63 Details of the SR can be found in Annex 5 (Table A5.3). Quality assessment of the SR using the AMSTAR 2 tool can be found in Annex 8 (Table A8.40). Additional data extracted on the primary studies can be found in Annex 9 (Table A9.30).
- 6.64 The criteria used to grade the evidence are provided in chapter 2 (Table 2.4, paragraphs 2.54 to 2.59). Summary tables of the evidence grading process for this section are provided in Annex 10 (Tables A10.14 and A10.36).

## **Total milk consumption and body composition or weight status**

- 6.65 One SR without MA (Dougkas et al, 2019) (AMSTAR 2 confidence rating: low) examined the relationship between total milk consumption and body composition (BMI or % body fat) or weight status (incident overweight) in childhood and included 6 PCS that examined this relationship in children aged 1 to 5 years.
- 6.66 Four PCS (in total 11,992 participants) reported no association between total milk consumption and BMI z-score. The follow up period in the 5 PCS ranged from 8 months to 4 years. All 4 PCS adjusted for sex and demographic factors (ethnicity); 3 adjusted for socioeconomic status; 2 adjusted for TDEI (see chapter 3, paragraphs 3.48 and 3.49) and 2 adjusted for consumption of non-dairy beverages. For detailed results, see Annex 9, Table A9.30.
- 6.67 The fifth PCS (in 103 participants) reported that children in the highest tertile of milk consumption (411ml per day) between ages 3 to 5 years had a lower % body fat compared with children in the lowest tertile of consumption (115ml per day) after 12 years’ follow up (MD -7.3%; 95% CI NR; p=0.0095). The analysis was adjusted for age, baseline anthropometry, percentage energy intake from fat, television viewing, beverage consumption, maternal BMI and education.

## **Whole or reduced-fat milk consumption**

- 6.68 Two of the above PCS also considered separately the longitudinal relationship between consumption of milk with different fat content and BMI z-score.

- 6.69 One PCS (in 852 participants) reported that each additional serving per day of whole milk at age 2 years was associated with a reduction in BMI z-score at age 3 years (beta coefficient:  $-0.09$ ; 95% CI  $-0.16$  to  $-0.01$ ;  $p=0.02$ ). However, when the analysis was restricted to children with healthy weight at baseline (defined as having a BMI between the 5th and 85th centiles at age 2 years), the association disappeared, indicating the possibility of reverse causality. The same study reported that neither consumption of whole milk nor reduced fat milk (servings per day) at age 2 years was associated with risk of incident overweight at age 3 years. All analyses were adjusted for TDEI, age, sex, ethnicity, baseline BMI z-score, and maternal education.
- 6.70 The second PCS (in 8300 participants) reported no difference in change in BMI z-scores from ages 2 and 4 years between children who consumed whole milk at both ages and children who consumed reduced fat milk at both ages ( $p=0.6$  for the difference between groups). However, the same PCS reported that children with a healthy weight at baseline who consistently drank 1% fat or skimmed milk at ages 2 and 4 years had a greater odds of becoming overweight or obese during this time period (OR 1.57; 95% CI 1.03 to 2.42;  $p<0.05$ ) compared with children who consistently drank whole or 2% milk. The analyses were adjusted for sex, ethnicity, SES, child's baseline BMI, fruit juice and SSB consumption, daily glasses of milk at age 4 years and maternal BMI.

## Summary: milk consumption and body composition or weight status

- 6.71 The evidence identified from SRs on milk consumption and body composition or weight status in children aged 1 to 5 years is summarised in Table 6.11.

**Table 6.11 Summary of the evidence on milk consumption and body composition or weight status**

Exposure	Outcome	Direction of association	Certainty of evidence
Total milk consumption	Body fat	Not applicable	Insufficient
Total milk consumption	Body Mass Index (BMI) z-score	No association	Limited
Whole milk consumption	BMI or incident overweight	Not applicable	Insufficient
Reduced-fat milk consumption	BMI or incident overweight	Not applicable	Insufficient
Whole milk versus reduced-fat milk consumption	Odds of overweight or obesity	Not applicable	Insufficient

- 6.72 The available evidence from SRs on the relationship between milk consumption and growth and body composition or weight status in children aged 1 to 5 years is from 1 SR given a low confidence rating using the AMSTAR 2 tool.
- 6.73 There was insufficient evidence from SRs to enable conclusions to be drawn on any relationship between total milk consumption and body fat or incident overweight as fewer than 3 primary studies included in the SR examined these relationships.
- 6.74 Evidence from 4 PCS in the SR by Dougkas et al (2019) suggests that there is no association between total milk consumption and BMI in children aged 1 to 5 years. The evidence was graded limited due to the lack of reporting of confidence intervals and inconsistency in adjustment for confounders.

## Fruit juice

- 6.75 In the UK, it is recommended that fruit juice consumption should be limited to 1 portion of 150ml a day because of high levels of free sugars (see [Classification of carbohydrates](#) in chapter 3). This recommendation applies from age 2 years and older. It is also advised that children aged 6 years and under should minimise consumption of sugars-containing foods and drinks to prevent dental caries (DHSC, 2021a).

## Fruit juice consumption in the UK

- 6.76 Data from DNSIYC and NDNS (years 2016 to 2019) on the consumption and contribution of fruit juice (100% fruit juice and smoothies) to the diets of children aged 12 to 60 months in the UK are presented in Table 6.12 to 6.14. Values that include non-consumers provide an estimate of the overall contribution of fruit juice to the diets of young children, while values for consumers provide an estimate of the quantities consumed.
- 6.77 Twenty-six percent of children aged 12 to 18 months and over 40% of children aged 18 to 60 months consumed fruit juice (100% fruit juice and smoothies) over the 4 day survey period. Fruit juice (100% fruit juice and smoothies) contributed between 2% TDEI in consumers aged 12 to 18 months (0.5% TDEI at a population level) and approximately 3% TDEI in consumers aged 18 to 60 months (approximately 1% TDEI at a population level).

**Table 6.12 Fruit juice consumption (grams per day and contribution to TDEI) in children aged 12 to 18 months in the UK (DNSIYC)<sup>1</sup>**

Food group	Mean consumption (SD) in grams per day Includes non-consumers	% Contribution to TDEI Includes non-consumers	% (number) of consumers over 4 days	Mean consumption (SD) in grams per day Consumers only	% Contribution to TDEI Consumers only
Fruit juice <sup>2</sup>	13 (36)	0.5	26 (326)	50 (57)	2.1

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake.

<sup>1</sup> Data from DNSIYC 2011 (Lennox et al, 2013). Number of participants = 1275

<sup>2</sup> Fruit juice covers 100% fruit juice and smoothies.

**Table 6.13 Fruit juice consumption (grams per day and contribution to TDEI) in children aged 18 to 47 months in the UK (NDNS years 2016 to 2019)<sup>1</sup>**

Food Group	Mean consumption (SD) in grams per day Includes non-consumers	% Contribution to TDEI Includes non-consumers	% (number) of consumers over 4 days	Mean consumption (SD) in grams per day Consumers only	% Contribution to TDEI Consumers only
Fruit juice <sup>2</sup>	38 (79)	1.3	44 (128)	86 (101)	2.9

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake.

<sup>1</sup> Data from NDNS 2016 to 2019. Number of participants = 306

<sup>2</sup> Fruit juice covers 100% fruit juice and smoothies.

**Table 6.14 Fruit juice consumption (grams per day and contribution to TDEI) in children aged 48 to 60 months in the UK (NDNS years 2016 to 2019)<sup>1</sup>**

Food Group	Mean consumption (SD) in grams per day Includes non-consumers	% Contribution to TDEI Includes non-consumers	% (number) of consumers over 4 days	Mean consumption (SD) in grams per day Consumers only	% Contribution to TDEI Consumers only
Fruit juice <sup>2</sup>	33 (58)	1.0	40 (42)	[81 (65)]	2.5

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake.

<sup>1</sup> Data from NDNS 2016 to 2019. Number of participants = 102

<sup>2</sup> Fruit juice covers 100% fruit juice and smoothies.

- 6.78 Fruit juice (and smoothies) contributed, on average, 5%, 11% and 7% to free sugars intakes in children aged 12 to 18 months, 18 to 47 months and 48 to 60 months, respectively, at the population level (see chapter 3, Table 3.13).
- 6.79 Time trend analysis of NDNS data (years 2008 to 2017) in children aged 18 to 36 months indicated no significant change in the percentage of consumers of fruit juice (average change per year  $-0.6\%$ ; 95% CI  $-2.1\%$  to  $0.9\%$ ) for the 9-year period (Bates et al, 2019). No time trend data was available for the other age groups.

## Fruit juice consumption and deprivation

- 6.80 Fruit juice consumption by index of multiple deprivation (IMD) (see Glossary) in children aged 18 to 60 months (including non-consumers) is presented in Table 6.15.
- 6.81 There is no association between fruit juice consumption and IMD quintile, as indicated by overlapping confidence intervals.

**Table 6.15 Fruit juice consumption by IMD quintile in children aged 18 to 60 months in England (NDNS years 2008 to 2019)<sup>1</sup>**

Consumption Mean (95%CI)	IMD quintile 1 (least deprived)	IMD quintile 2	IMD quintile 3	IMD quintile 4	IMD quintile 5 (most deprived)
Fruit juice (grams per day)	65 (51 to 78)	54 (41 to 68)	57 (45 to 69)	61 (48 to 73)	46 (36 to 56)
Number of participants	210	212	182	234	277

Abbreviations: IMD, index of multiple deprivation.

<sup>1</sup> Data from NDNS years 2008 to 2019. Includes non-consumers.

## Systematic review evidence identified on fruit juice consumption and health

- 6.82 One SR without MA (Frantsve-Hawley et al, 2017) was identified that examined the health impact of consuming 100% fruit juice (with no added or free sugars) in childhood.
- 6.83 The outcome covered in this chapter is BMI.
- 6.84 All primary studies included in the SR were conducted in HIC.

- 6.85 Details of the SR can be found in Annex 5 (Tables A5.4). Quality assessment of the SR using the AMSTAR 2 tool can be found in Annex 8 (Tables A8.7). Additional data extracted on the primary studies can be found in Annex 9 (Table A9.31). The criteria used to grade the evidence are provided in chapter 2 (Table 2.4, paragraphs 2.54 to 2.59). Summary tables of the evidence grading process for this section are provided in Annex 10 (Tables A10.16).

## **Fruit juice consumption and BMI**

- 6.86 One SR without MA (Frantsve-Hawley et al, 2017) (AMSTAR 2 confidence rating: moderate) examined the relationship between consumption of 100% fruit juice and BMI. It included 7 PCS that examined this relationship in children aged 1 to 5 years. For one of the PCS, analyses at 2 different time points (age 4 months and 1 year) were reported (in 2 publications). Only the result for the later time point (age 1 year) is described below.
- 6.87 Six of the seven PCS examined the relationship between fruit juice consumption and change in BMI (or BMI z-score). Of the 6 PCS, 3 PCS (in a total of 10,938 participants) reported that fruit juice consumption was associated with an increase in BMI (or BMI z-score); the other 3 PCS (in a total of 16,854 participants) reported no association.
- 6.88 Of the 3 PCS that reported an association, 1 PCS (in 1163 participants) reported a dose-response relationship. Compared with no juice consumption, the mean change in BMI increased from 0.08 kg/m<sup>2</sup> (95% CI -0.05 to 0.20 kg/m<sup>2</sup>) for consumption of 1 to 7 ounces of juice per day to 0.36 kg/m<sup>2</sup> (95% CI 0.08 to 0.64 kg/m<sup>2</sup>) for consumption over 16 ounces per day after 6 years' follow-up.
- 6.89 The 3 PCS that reported an association tended to have longer follow-up durations (2 to 6 years) than the 3 PCS that reported no association (6 months to 4 years).
- 6.90 None of the PCS that reported an association adjusted for TDEI while 2 of 3 PCS that reported no association did (see chapter 3, paragraphs 3.48 and 3.49). The difference between these analyses indicates that any effect of fruit juice consumption on later BMI may be mediated by its contribution to increasing TDEI.
- 6.91 One additional PCS (in 10,904 participants) reported no association between fruit juice consumption (in servings per day) at ages 2 to 3 years in children with healthy weight at baseline and odds of incident obesity 1 year later, adjusted for TDEI (see Annex 9, Table A9.31 for details).
- 6.92 Most of the 7 PCS adjusted for multiple potential confounding factors including sex, a measure of baseline body size, ethnicity or SES.

## Summary: Fruit juice consumption and BMI

- 6.93 The evidence identified from SRs on fruit juice consumption and BMI in children aged 1 to 5 years is summarised in Table 6.16.

**Table 6.16 Summary of the evidence on fruit juice consumption and BMI**

Exposure	Outcome	Direction of association <sup>1</sup>	Certainty of evidence
Fruit Juice	Change in Body Mass Index (BMI) or BMI z-score	↑(non-TDEI adjusted) No association (TDEI-adjusted)	Limited <sup>2</sup>

Abbreviations: TDEI, total dietary energy intake.

<sup>1</sup> Direction of association for reported outcomes: ↑increase

<sup>2</sup> Findings both unadjusted and adjusted for TDEI were graded separately as limited.

- 6.94 The available evidence from SRs on the relationship between fruit juice consumption in children aged 1 to 5 years and BMI in children aged 1 to 5 years is from 1 SR without MA given a moderate confidence rating using the AMSTAR 2 tool.
- 6.95 Evidence from 3 PCS included in the SR by Frantsve-Hawley et al (2017) indicated that higher fruit juice consumption in children aged 1 to 5 years is associated with increased BMI in childhood, when unadjusted for TDEI, compared with lower fruit juice consumption. A dose-response relationship demonstrated by one of these PCS suggests the relationship maybe causal. In contrast, evidence from 4 PCS from the same SR indicated that there is no association between fruit juice consumption and BMI in childhood after adjusting for TDEI. The difference between these analyses indicates that any effect of fruit juice consumption on later BMI may be mediated by its contribution to increasing TDEI (see chapter 3, paragraphs 3.48 and 3.49).
- 6.96 The evidence that fruit juice consumption is directly associated with BMI, when unadjusted for TDEI, was graded 'limited'. The evidence that fruit juice consumption is not associated with BMI, when adjusted for TDEI, was also graded 'limited'.

## Sugar-sweetened beverages

- 6.97 For the purposes of this report, a sugar-sweetened beverage (SSB) is any (non-dairy) beverage (carbonated drinks, fruit-based drinks, squashes, flavoured water) where sugars have been specifically added as a sweetener. This definition is based on what is used in the NDNS as well as in the SRs identified on this topic

area. Where possible, SSBs are distinguished from formula milks, flavoured milks (for example, milkshakes, flavoured milk based drinks, hot chocolate, evaporated and condensed milks) and 100% fruit juices (with naturally occurring levels of sugars) (see [Fruit juice](#)).

- 6.98 In its report '[Carbohydrates and Health](#)', SACN found that consumption of SSBs, compared with non-calorically sweetened beverages, resulted in greater weight gain and increases in BMI in children aged 5 years and older (including adolescents) (SACN, 2015). The hypothesised mechanisms that link consumption of SSBs to weight gain include low satiety of liquid calories and incomplete compensation in energy intake at subsequent meals, leading to an increase in TDEI (Malik & Hu, 2011). SACN also found moderate evidence that greater consumption of SSBs is detrimental to oral health in primary dentition (see [Sugar-sweetened beverages and development of dental caries](#) for additional evidence in children aged 1 to 5 years identified for this report).
- 6.99 SACN therefore recommended that SSB consumption be minimised in children (SACN, 2015).

## **Sugar-sweetened beverage consumption in the UK**

- 6.100 Data from DNSIYC and NDNS (years 2016 to 2019) on the consumption and contribution of SSBs to the diets of children aged 12 to 60 months in the UK are presented in Table 6.17 to 6.19. Values that include non-consumers provide an estimate of the overall contribution of SSBs to the diets of young children, while values in consumers provide an estimate of the quantities consumed.
- 6.101 Twenty-six percent of children aged 12 to 18 months and over 20% of children aged 18 to 60 months consumed SSBs over the 4 day survey period.
- 6.102 SSBs contributed 1.6% TDEI in consumers aged 12 to 18 months (0.5% TDEI at a population level) and 1.7% TDEI in consumers aged 18 to 47 months (0.4% TDEI at a population level). SSBs contributed 0.5% TDEI at a population level in children aged 48 to 60 months (data were insufficient to present for consumers only in this age group).

**Table 6.17 Sugar-sweetened beverage consumption<sup>1</sup> (grams per day and contribution to TDEI) in children aged 12 to 18 months in the UK (DNSIYC)<sup>1</sup>**

Food group	Mean consumption (SD) in grams per day Includes non-consumers	% Contribution to TDEI Includes non-consumers	% (number) of consumers over 4 days	Mean consumption (SD) in grams per day Consumers only	% Contribution to TDEI Consumers only
SSB <sup>2</sup>	42 (119)	0.4	26 (329)	158 (186)	1.6

Abbreviations: SD, standard deviation; SSB, sugar-sweetened beverage; TDEI, total dietary energy intake.

<sup>1</sup> Data in children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013). Number of participants = 1275

<sup>2</sup> Includes carbonated drinks, concentrates and ready to drink products with added sugars.

**Table 6.18 Sugar-sweetened beverage consumption<sup>1</sup> (grams per day and contribution to TDEI) in children aged 18 to 47 months in the UK (NDNS years 2016 to 2019)<sup>1</sup>**

Food Group	Mean consumption (SD) in grams per day Includes non-consumers	% Contribution to TDEI Includes non-consumers	% (number) of consumers over 4 days	Mean consumption (SD) in grams per day Consumers only	% Contribution to TDEI Consumers only
SSB <sup>2</sup>	19 (62)	0.4	20 (68)	95 (108)	1.8

Abbreviations: SD, standard deviation; SSB, sugar-sweetened beverage; TDEI, total dietary energy intake.

<sup>1</sup> Data from NDNS 2016 to 2019. Number of participants = 306

<sup>2</sup> Includes carbonated drinks, concentrates and ready to drink products with added sugars.

**Table 6.19 Sugar-sweetened beverage consumption<sup>1</sup> (grams per day and contribution to TDEI) in children aged 48 to 60 months in the UK (NDNS years 2016 to 2019)<sup>1</sup>**

Food group	Mean consumption (SD) in grams per day Includes non-consumers	% Contribution to TDEI Includes non-consumers	% (number) of consumers over 4 days	Mean consumption (SD) in grams per day Consumers only	% Contribution to TDEI Consumers only
SSB <sup>2</sup>	35 (97)	0.5	23 (23)	No data <30 consumers	No data <30 consumers

Abbreviations: SD, standard deviation; SSB, sugar-sweetened beverage; TDEI, total dietary energy intake.

<sup>1</sup> Data from NDNS 2016 to 2019. Number of participants = 102

<sup>2</sup> Includes carbonated drinks, concentrates and ready to drink products with added sugars.

- 6.103 SSBs contributed, on average, 2.5%, 2.8% and 3.8% to free sugars intakes in children aged 12 to 18 months, 18 to 47 months and 48 to 60 months, respectively, at a population level (see chapter 3, Table 3.13)
- 6.104 Time trend analysis of NDNS data (years 2008 to 2019) in children aged 18 to 36 months indicated a decrease in the percentage of consumers of SSBs (average change per year  $-2.9\%$ ; 95% CI  $-3.8\%$  to  $-2.0\%$ ) for the 11-year period (Bates et al, 2019). No time trend data were available for the other age groups.

## Sugar-sweetened beverage consumption and deprivation

- 6.105 SSB consumption by index of multiple deprivation (IMD) (see Glossary) in children aged 18 to 60 months (including non-consumers) is presented in Table 6.20.
- 6.106 There was no association between SSB consumption and IMD quintile, as indicated by overlapping confidence intervals.

**Table 6.20 SSB<sup>1</sup> consumption by IMD quintile in children aged 18 to 60 months in England (NDNS years 2008 to 2019)<sup>1</sup>**

Consumption Mean (95%CI)	IMD quintile 1 (least deprived)	IMD quintile 2	IMD quintile 3	IMD quintile 4	IMD quintile 5 (most deprived)
SSB (grams per day)	45 (30 to 61)	42 (30 to 54)	54 (35 to 73)	46 (34 to 59)	59 (45 to 74)
Number of participants	210	212	182	234	277

Abbreviations: IMD, index of multiple deprivation; SSB, sugar-sweetened beverage.

<sup>1</sup> Data from NDNS years 2008 to 2019. Includes non-consumers.

<sup>2</sup> Includes carbonated drinks, concentrates and ready to drink products with added sugars.

## Systematic review evidence identified on sugar-sweetened beverage consumption and health

- 6.107 Four SRs without MA (Frantsve-Hawley et al, 2017; Luger et al, 2017; Perez-Morales et al, 2013; Tandon et al, 2016) and 1 with MA (Te Morenga et al, 2012), were identified that examined the health impact of SSB consumption in childhood.

- 6.108 Key outcomes were measures of body composition (BMI, BMI z-score, weight-for-height z-score, body fat) and weight status (overweight or obesity) and cognitive development. For SR evidence on the impact of consuming drinks containing free sugars, including SSBs, on oral health, see chapter 9.
- 6.109 'Sugar-sweetened beverage' was defined differently in each SR that included this as an exposure. In Frantsve-Hawley et al (2017), SSBs included all sugar-sweetened (non-dairy) beverages and 100% fruit juice. In Te Morenga et al (2012), SSB did not include fruit juice, but the category was otherwise undefined. In Luger et al (2017), SSBs included soft drinks, 'fruit juice drinks' (undefined), syrup-based drinks, flavoured water with (added) sugar, and sports drinks. In Perez-Morales et al (2013), SSBs included soft drinks, 'soda', 'fruit drinks' (undefined), sports drinks, sweetened iced tea and lemonade. In Tandon et al (2016), SSBs included soft drinks, cordial and 'fruit drinks' (undefined).
- 6.110 Most SRs did not discuss the implications of findings adjusted for TDEI against those that were not when outcomes relating to or resulting from effects on energy balance were investigated (paragraph 3.49).
- 6.111 The majority of primary studies included in the SRs were conducted in HIC.
- 6.112 Details of the SRs can be found in Annex 5 (Tables A5.3 and A5.4). Quality assessment of the SRs using the AMSTAR 2 tool can be found in Annex 8 (Tables A8.5 and A8.7). Additional data extracted on the primary studies can be found in Annex 9 (Tables A9.32). The criteria used to grade the evidence are provided in chapter 2 (Table 2.4, paragraphs 2.54 to 2.59). Summary tables of the evidence grading process for this section are provided in Annex 10 (Tables A10.17 and Table A10.36).

## **Sugar-sweetened beverages and body composition or weight status**

- 6.113 One SR with MA (Te Morenga et al, 2012) and 2 SRs without MAs (Frantsve-Hawley et al, 2017; Luger et al, 2017) examined the relationship between SSB consumption in children aged 1 to 5 years and body composition or weight status. Outcome measures were odds or risk of overweight or obesity, and changes in body composition (BMI, BMI z-score, weight-for-height z-score) over time.

## **Sugar-sweetened beverages and odds or risk of overweight or obesity**

- 6.114 One SR with MA (Te Morenga et al, 2012) and one SR without MA (Frantsve-Hawley et al, 2017) examined the relationship between SSB consumption in children aged under 5 years and odds or risk of overweight or obesity.
- 6.115 Te Morenga et al (2012) (AMSTAR 2 confidence rating: moderate) reported that higher consumption of SSBs (servings per day or per week) in children mostly aged 1 to 5 years was associated with a greater odds of overweight or obesity 1 to 8 years later compared with lower consumption of SSBs (OR 1.55; 95% CI 1.32 to 1.82;  $p < 0.001$ ;  $I^2 = 0$ ; random-effects model; 7 estimates from 5 PCS; 7225 participants). All 7 estimates were from PCS that adjusted for multiple key confounding factors (age, sex, baseline BMI and physical activity). Six of the seven estimates were from 4 PCS that adjusted for TDEI, indicating that SSBs may independently contribute to later odds of overweight or obesity.
- 6.116 Frantsve-Hawley et al (2017) (AMSTAR 2 confidence rating: moderate) included 2 additional PCS that examined the relationship between SSB consumption in children aged 1 to 5 years and later odds or risk of overweight. One PCS (in 568 participants) reported that children aged 3 to 6 years who consumed  $>65$ ml per day of SSBs had a greater odds of being overweight 30 months later compared with children who consumed  $<65$ ml per day, unadjusted for TDEI, but with a wide confidence interval (OR 1.36; 95% CI 0.77 to 2.40). The PCS adjusted for baseline BMI, SES and physical activity.
- 6.117 The second PCS (in 4169 participants) reported that the risk of children with normal weight who consumed SSBs at ages 4 to 5 years becoming overweight 6 years later was not greater than children who did not consume SSBs (RR 0.97, SE 0.05;  $p = 0.57$ ), unadjusted for TDEI. The analysis adjusted for sex, ethnicity, sedentary behaviour, parental BMI and SES.

## **Sugar-sweetened beverages and change in BMI or weight-for-height z-score**

- 6.118 Two SRs without MAs (Frantsve-Hawley et al, 2017; Luger et al, 2017) examined the relationship between SSB consumption and change in BMI (or BMI z-score) or weight-for-height z-score (WHZ) in children.
- 6.119 Frantsve-Hawley et al (2017) included 5 PCS in children aged 1 to 5 years. Of the 5 PCS, 3 PCS (in a total of 29,187 participants) reported that higher SSB consumption at age 1 to 5 years was associated with a greater increase in BMI (or BMI z-score), unadjusted for TDEI. The other 2 PCS (in a total of 1381 participants) reported no association, adjusted for TDEI.

- 6.120 Of the 3 PCS that reported an association, 1 PCS (in 15,418 participants) reported that consuming any SSBs at age 4 years was associated with a 0.138 (SE 0.037;  $p < 0.01$ ) increase in BMI over the next 2 years, compared with not consuming any SSBs. Another PCS (in 4169 participants) reported that each additional intake of SSB per day was associated with a 0.015 increase in BMI z-score (95% CI 0.004 to 0.25;  $p < 0.01$ ) 6 years later.
- 6.121 Compared with the PCS that reported no association, the PCS that reported an association were larger and tended to have longer follow-up durations (2 years versus 6 months). None of the PCS that reported an association adjusted for TDEI. One of the 2 PCS that reported no association adjusted for TDEI while another reported that adjusting for TDEI did not change the findings.
- 6.122 The PCS that reported an association did not adjust for baseline BMI and therefore reverse causality cannot be ruled out (see chapter 3, paragraph 3.53).
- 6.123 Luger et al (2017) (AMSTAR 2 confidence rating: low) included 2 additional PCS in children aged 1 to 5 years. Both PCS (in a total of 294 participants) reported that higher SSB consumption (units NR) in children aged 1 to 2 years was associated with a greater increase in weight-for-height z-score (WHZ) 6 months later (in 1 study) or higher BMI 13 years later (in the other study), unadjusted for TDEI. Quantitative details for both studies were not reported. Only 1 of the studies adjusted for baseline weight status.

## **Sugar-sweetened beverages and body fat**

- 6.124 Perez-Morales et al (2013) (AMSTAR 2 confidence rating: critically low) included 1 PCS that examined the relationship between SSB consumption in children aged 1 to 5 years and body fat. The PCS (in 135 participants) reported that an increase in energy intake from SSB between ages 3 to 5 years was associated with a larger waist circumference at ages 5 to 6 years (beta coefficient 0.04cm; 95% CI NR;  $p = 0.001$ ). The study adjusted for TDEI at baseline and change in waist circumference at ages 3 to 5 years.

## **Summary: sugar-sweetened beverage consumption and body composition or weight status**

- 6.125 The evidence on SSB consumption and body composition or weight status in children aged 1 to 5 years is summarised in Table 6.21.

**Table 6.21 Summary of the evidence on sugar-sweetened beverage consumption and body composition or weight status**

Exposure	Outcome	Direction of association <sup>1</sup>	Certainty of evidence
Sugar-sweetened beverage consumption	Odds of overweight or obesity (mostly adjusted for total dietary energy intake)	↑	Adequate
Sugar-sweetened beverage consumption	Change in Body Mass Index (BMI) (or BMI z-score or weight-for-height z-score) (unadjusted for total dietary energy intake)	↑	Moderate
Sugar-sweetened beverage consumption	Body fat	Not applicable	Insufficient

<sup>1</sup> Direction of association for reported outcomes: ↑increase

- 6.126 The available evidence from SRs on the relationship between SSB consumption in children aged 1 to 5 years and body composition or weight status is from 3 SRs (1 with MA), of which 2 were given a moderate confidence rating using the AMSTAR 2 tool and 1 was given a low confidence rating.
- 6.127 Evidence from the SR with MA by Te Morenga et al (2012) suggests that higher SSB consumption in children aged 1 to 5 years is associated with a greater odds of overweight or obesity in childhood compared with lower SSB consumption, adjusted for baseline weight status. Six of the seven estimates included in the MA adjusted for TDEI, indicating that SSBs may contribute to later odds of overweight or obesity independent of their contribution to increasing TDEI. The evidence was graded ‘adequate’ given the large association, no statistical heterogeneity, and adequate accounting for key confounding factors by the PCS included in the MA.
- 6.128 Evidence from 5 additional PCS included in 2 SRs without MAs by Frantsve-Hawley et al (2017) and Luger et al (2017) suggests that higher SSB consumption in children aged 1 to 5 years is associated with a greater increase in BMI (or BMI z-score or WHZ) in childhood, unadjusted for TDEI, compared with lower SSB consumption. However, most of the PCS did not adjust for baseline weight status, a key confounding factor. Therefore, the possibility of reverse causality cannot be ruled out. For this reason, the evidence was graded ‘moderate’ rather than ‘adequate’ but nevertheless strengthens the findings from the SACN report ‘Carbohydrates and Health’ (SACN, 2015).

- 6.129 Together with the findings from Te Morenga et al (2012) (paragraph 6.127), the evidence from Frantsve-Hawley et al (2017) and Luger et al (2017) indicates that the effect of SSBs on later weight gain or excess weight may be partially mediated by its contribution to increasing TDEI (see paragraph 3.49) and partially independent of its contribution to increasing TDEI.
- 6.130 There was insufficient evidence from SRs to enable conclusions to be drawn on any relationship between SSB consumption and body fat as fewer than 3 primary studies included in the SRs examined this relationship.

## Sugar-sweetened beverages and cognitive development

- 6.131 One SR without MA (Tandon et al, 2016) (AMSTAR 2 confidence rating: critically low) included 1 PCS that examined the relationship between SSB consumption in children aged 1 to 5 years and cognitive development. The PCS (in 1445 participants) reported that higher SSB consumption (per serving) at age 1 year was associated with lower nonverbal reasoning ability at age 10 years (quantitative findings NR). The analysis was adjusted for sex, breastfeeding duration, maternal characteristics (age, education and mental health distress), family income, and reading to the child.

## Summary: sugar-sweetened beverages and cognitive development

- 6.132 The evidence on SSB consumption and cognitive development in children aged 1 to 5 years is summarised in Table 6.22.

**Table 6.22 Summary of the evidence on sugar-sweetened beverage intake and cognitive development**

Exposure	Outcome	Direction of association	Certainty of evidence
Sugar-sweetened beverage consumption	Cognitive development	Not applicable	Insufficient

- 6.133 The available evidence from SRs on the relationship between SSB consumption and cognitive development in children aged 1 to 5 years is from 1 SR without MA, given a critically low confidence rating using the AMSTAR 2 tool.
- 6.134 There was insufficient evidence from SRs to enable conclusions to be drawn on any relationship between SSB consumption in children aged 1 to 5 years and

cognitive development as fewer than 3 primary studies included in the SR examined this relationship.

- 6.135 No evidence from SRs was identified on the relationship between other types or sources of carbohydrate and cognitive development in children aged 1 to 5 years.
- 6.136 No evidence from SRs was identified on the relationship between carbohydrate intake and any other health outcomes in children aged 1 to 5 years.

# 7 Eating and feeding behaviours

## Background

- 7.1 There are a number of biological, social and environmental factors that influence food acceptance and preferences during infancy and early childhood. There is evidence to indicate that some food acceptance outcomes may have their origins in utero when tastes and smells from the maternal diet may be transmitted via amniotic fluid (Freitas et al, 2018; Wood et al, 2020). Breastfed infants are also exposed to flavours from the maternal diet in breast milk and may accept a wider variety of foods than those fed infant formula (Freitas et al, 2018).
- 7.2 Infants readily accept sweet tastes (Desor et al, 1977; Desor et al, 1973). For salty tastes, acceptance increases between 3 and 12 months old (Schwartz et al, 2017) and preference for salt is determined, in part, by salt exposure (Stein et al, 2012; Sullivan & Birch, 1990). Bitter and sour tastes are the least accepted tastes in infancy (Schwartz et al, 2009) and it has been speculated that the infant's predisposition to reject these tastes represents an innate response that has evolved to protect infants from potential toxins (Rozin, 1976). However, early exposure to bitter tastes may improve later acceptance (Nehring et al, 2015). Innate responses to the basic tastes can nevertheless be modified by exposure to different flavours in early life (Beauchamp & Mennella, 2009; Mennella & Trabulsi, 2012).
- 7.3 As young children become more independent around food, certain avoidant behaviours begin to emerge, including food neophobia, which is the avoidance of new foods, and food fussiness ('picky' eating), where a child eats a reduced variety of foods and rejects many foods even if familiar and previously liked by the child (Dovey et al, 2008).
- 7.4 While interrelated, food neophobia and food fussiness are behaviourally distinct, with different factors predicting the severity and expression of each (Galloway et al, 2003). Both tend to emerge around ages 18 to 24 months and typically diminish during the preschool period, although both can persist in some children (Cole et al, 2017; Wood et al, 2020). Children who display food neophobia or food fussiness tend to reject foods such as meat, vegetables and fruit, which can negatively impact on micronutrient status, but growth is not usually affected (Wright et al, 2007).
- 7.5 Finding from the Gemini birth cohort study in England and Wales has suggested that food fussiness and liking for certain foods have a genetic basis (Fildes et al, 2016), and that common genetic factors predict both food fussiness and preferences for vegetables and fruit (Fildes et al, 2016). There also appears to be

a genetic component to the development of other eating traits, including food responsiveness, satiety responsiveness and slowness in eating (Freitas et al, 2018).

- 7.6 Other intrinsic behavioural traits may also be important in determining eating behaviour and weight status (Stifter & Moding, 2019). For example, poorer self-regulation of energy intake, including eating past the point of satiety or eating in the absence of hunger, are potential behavioural pathways to excess weight gain (Brugailleres et al, 2019; Lansigan et al, 2015; Miller et al, 2016).
- 7.7 It has been suggested that caregiver practices such as restriction or pressure to eat, may contribute to disrupting a child's ability to respond to internal hunger or satiety cues and thereby may indirectly contribute to weight gain (Wood et al, 2020). However, a child's behaviour in relation to food or their nutritional status may evoke certain responses from their caregiver which in turn may affect how the child subsequently responds (Stifter & Moding, 2019; Wood et al, 2020). In the Twins Early Development Study, it was found that caregiver practices such as restricting or encouraging food intake was determined, in part, by the child's genetic predisposition to a higher or lower BMI (Selzam et al, 2018). This bi-directional interaction between children and their caregivers is not always considered in research.
- 7.8 A distinction has been made between general caregiver or parenting styles and practices (Peters et al, 2012; Vollmer & Mobley, 2013; Wood et al, 2020).
- 7.9 General caregiver or parenting styles encapsulate the emotional climate around caregiver-child interactions and are defined along 2 dimensions: demandingness (that is, the extent to which the parent or caregiver makes demands on the child) and responsiveness (that is, the extent to which the parent or caregiver is responsive to the child's needs) (Hurley et al, 2011; Vollmer & Mobley, 2013). Combinations of high or low demandingness and responsiveness give rise to 4 distinct parenting styles: authoritative, authoritarian, indulgent, and uninvolved. For example, an authoritative parenting style (high responsiveness and demandingness) is when the parent or caregiver is responsive to the child's needs, involved and makes appropriate demands on the child; while an authoritarian parenting style (low responsiveness and high demandingness) is when the parent or caregiver is highly directive but unresponsive to the child's needs (Hughes et al, 2011).
- 7.10 When applied to specific eating and feeding interactions, caregiver or parenting styles have been termed 'feeding styles' (Vollmer & Mobley, 2013).
- 7.11 In contrast, feeding practices describe specific goal-oriented behaviours of the caregiver (for example, getting the child to eat their vegetables). Practices include those related to coercion or control (for example, pressuring a child to eat), structure (setting mealtimes and boundaries around food), and supporting and encouraging a child to eat (Wood et al, 2020). For example, a caregiver with an

authoritative feeding style might set boundaries around food while encouraging the child to respond to their internal cues of hunger or satiety, while a caregiver with an authoritarian feeding style might employ directive strategies to alter the child's behaviour such as by using food to soothe or reward, or restricting access to certain foods.

- 7.12 More generally, parents or caregivers create food environments that may foster the development of healthy or unhealthy eating behaviours. Factors that contribute to the shaping of these environments may include parental or caregiver attitudes and beliefs about foods and eating behaviours (Schwartz et al, 2011), which are in turn influenced by the caregiver's cultural beliefs and practices (Wood et al, 2020), mental health (Lindsay et al, 2016; McPhie et al, 2014), and physical resources (food security or insecurity) (Wood et al, 2020) (also see chapter 1, paragraphs 1.33 and 1.34 on wider environmental influences of food preferences and eating behaviours). Formal childcare settings (for example, nurseries and preschools) and informal childcare arrangements (for example, relatives) may also shape child eating behaviours (Alberdi et al, 2016).
- 7.13 The context around eating occasions also influences children's eating behaviours, diet quality and body weight. More frequent family mealtimes may be associated with better overall diet quality and lower BMI in children and adolescents (Dallacker et al, 2018). Yet there has been a move towards more informal eating patterns in high income countries. For example, US survey data show a marked increase in snacking among children since the 1970s, while those snacks have become more energy-dense and nutrient poor (Larson & Story, 2013).
- 7.14 This chapter focusses on 2 main areas of evidence. Consideration is first given to the evidence identified from systematic reviews (SRs) (with or without meta-analyses [MAs]) on children's eating behaviours at ages 1 to 5 years and any relationship these may have with child weight status. This is followed by an examination of the evidence identified from SRs (with or without MAs) on the impact of caregiver feeding styles and practices on acceptance and intake of foods (primarily vegetables and fruit) in children aged 1 to 5 years and weight status. Interventions to reduce the risk of obesity in children in childcare settings were considered out of scope of this risk assessment unless they had a specific dietary or feeding practice component of interest (see [Exclusion criteria](#)).

# **Systematic review evidence identified on children’s eating behaviours and health outcomes**

- 7.15 Four SRs without MAs (Blondin et al, 2016; Brown et al, 2016; Caleza et al, 2016) and 1 SR with MA (Kininmonth et al, 2021) were identified that examined the impact of children’s eating behaviours on body composition (BMI) and weight status in childhood and adolescence.
- 7.16 Among the types of eating behaviours examined, both Brown et al (2016) and Kininmonth et al (2021) investigated food fussiness (also known as ‘picky’ eating) and the evidence from both SRs is described in this section.
- 7.17 Kininmonth et al (2021) also examined other children’s eating behaviours that are captured in the Child Eating Behaviour Questionnaire (such as satiety responsiveness and enjoyment of food). However, the SR did not provide sufficient evidence on any of these behaviours and their potential impact on health and are therefore not described in the main report (for more details, see Annex 9, Table A9.33).
- 7.18 Caleza et al (2016) examined the impact of young children’s ability to delay gratification and weight status while Blondin et al (2016) examined the impact of breakfast consumption in young children and weight status.
- 7.19 Details of the SRs can be found in Annex 5 (Table A5.5) and Annex 6 (Table A6.2). Quality assessment of the SRs using the AMSTAR 2 tool can be found in Annex 8 (Table A8.8). Additional data extracted on the primary studies can be found in Annex 9 (Table A9.33). The criteria used to grade the evidence are provided in chapter 2 (Table 2.4, paragraphs 2.54 to 2.59). Evidence grades are provided in Annex 10 (Table A10.18) and summarised at the end of this section.

## **Limitations of the systematic review evidence on eating behaviours**

- 7.20 Across the SRs identified on eating behaviours, there was a paucity of large, adequately powered randomised controlled trials (RCTs) of sufficient length to capture habitual behaviour.
- 7.21 Many prospective cohort studies (PCS) included in the SRs did not adjust for potential confounding factors, which include socioeconomic status (SES) measures (parental education, household income) and baseline child weight status. At the same time, some studies adjusted for independent predictors and mediators (including household food insecurity, parenting styles, family or home

environment, community characteristics), potentially removing the mechanism by which children's eating behaviours may impact their body weight.

- 7.22 Primary studies also lacked consistent use of terminology or standardised definitions for key exposures (for example, 'picky eating', 'food fussiness', 'food neophobia') as well as standardised instruments to measure or assess eating behaviours. This limited the ability to combine data for meta-analysis or draw overarching conclusions.
- 7.23 The areas covered by the SRs that was specific to children aged 1 to 5 years was limited. For example, no SR evidence was identified on the impact of food neophobia, eating in the absence of hunger and informal eating behaviours (including snacking and eating while watching television) in children aged 1 to 5 years on dietary intake or weight status in the short or longer term.

## **Children's eating behaviours and body composition or weight status**

### **Food fussiness ('picky' eating)**

#### **Food fussiness and later BMI (BMI z-score)**

- 7.24 One SR without MA (Brown et al, 2016) and 1 SR with MA (Kininmonth et al, 2021) examined the relationship between food fussiness (or 'picky eating') in children aged 1 to 5 years and BMI (or BMI z-score). Although Kininmonth et al (2021) performed MAs, findings from PCS in children aged 1 to 5 years were not pooled into a single MA and were therefore considered separately.
- 7.25 Primary studies included in the SRs measured food fussiness using the caregiver-administered Child Eating Behaviour Questionnaire (CEBQ) or adaptations of it.
- 7.26 Brown et al (2016) (AMSTAR 2 confidence rating: moderate) included 1 PCS (in 156 participants) that reported no association between food fussiness (or any other eating behaviours) at ages 2 to 4 years and BMI z-score at ages 3 to 5 years ( $R^2_{\text{Change}}=0.01$ ;  $p=0.707$ ), after adjusting for baseline child BMI z-score, age, sex and maternal characteristics (age, BMI and education).
- 7.27 Kininmonth et al (2021) (AMSTAR 2 confidence rating: critically low) included an additional 2 PCS that reported no association between food fussiness at ages 14 months to 5 years and BMI z-score 12 to 30 months later, after adjusting for child baseline BMI z-score and maternal characteristics (BMI and education) and household income. Quantitative details were not reported for either study.

## **Food fussiness and change in BMI or standardised weight (weight-for-length z-score)**

- 7.28 Brown et al (2016) included an additional 2 PCS that examined the relationship between food fussiness and change in BMI or standardised weight over time.
- 7.29 One PCS (in 486 participants) reported no association between food fussiness (identified through cluster analysis) at age 1 year and change in standardised weight (weight-for-length z-score) from ages 1 to 3 years (mean 0.48; SD 1.25;  $p=0.4$ ), adjusted for sex.
- 7.30 The second PCS (in 135 participants) reported no association between food fussiness (measured by the Stanford Feeding Questionnaire) at ages 4 and 5 years and change in BMI at ages 4 to 5 years in the overall sample but did report that girls with food fussiness at age 4 years experienced a greater increase in BMI over 1 year (from 15.3 to 15.7kg/m<sup>2</sup>) compared with girls without food fussiness (from 16.4 to 16.3kg/m<sup>2</sup>). There was no evidence of a difference in change in BMI between boys who exhibited food fussiness and those who did not. The analyses from both PCS were not adjusted for potential confounding factors other than sex. However, in both PCS, children who exhibited food fussiness at baseline were lighter than children who did not.

## **Food fussiness and later in weight status**

- 7.31 Brown et al (2016) included an additional PCS (in 1498 participants) that reported that children who exhibited food fussiness at ages 2.5, 3.5 and 4.5 years had a greater odds of being underweight at age 4.5 years compared with children who were never fussy (OR 2.4; 95% CI 1.4 to 4.2;  $p$ -value NR). However, there was no association with weight status if children were fussy at 1 or 2 of the ages when measurements were taken compared with children who were never fussy (quantitative findings NR). The study did not adjust for baseline child weight status but did adjust for multiple other potential confounding factors, including the child's sex, maternal characteristics (age, immigrant status, education, smoking status during pregnancy), and family characteristics (type of household, income, number of parents with obesity).

## **Ability to delay gratification**

- 7.32 One SR without MA (Caleza et al, 2016) (AMSTAR 2 confidence rating: critically low) examined the relationship between children's ability to delay gratification and weight status and included 2 PCS that examined this relationship in children aged 1 to 5 years. Both PCS reported an association between the inability to delay gratification and later BMI or risk of being overweight.
- 7.33 One PCS (in 805 participants) reported that children who failed a task that tested their ability to delay gratification (and involved their preferred food) at age 4 years

had a greater risk of being overweight at age 11 years (RR 1.29; 95% CI 1.06 to 1.58; p-value NR) compared with children who passed the task. The analysis was adjusted for baseline BMI z-score, sex, ethnicity, SES and maternal marital status.

- 7.34 One PCS (in 1061 participants) reported that children who scored low on tasks designed to test their self-regulatory ability (involving food and non-food items) at the ages of 3 and 5 years experienced the highest gains in BMI z-score from ages 3 to 12 years compared with children with higher self-regulatory capacity (change in age- and sex-standardised BMI z-score from ages 3 to 12 years: 0.57; SD 0.05). Analyses were conducted separately in boys and girls and were adjusted for maternal education and household income. As this study did not adjust for children's weight status at baseline, the possibility of reverse causality (whereby children with a higher BMI are prone to poorer self-regulation behaviour than children with a lower BMI) cannot be ruled out.

## **Breakfast consumption**

- 7.35 One SR without MA (Blondin et al, 2016) (AMSTAR 2 confidence rating: critically low) examined the relationship between breakfast consumption and weight status in children and adolescents and included 1 PCS in children aged 1 to 5 years. The PCS (in 1366 participants) reported no association between skipping breakfast at ages 2 and 5 years and odds of being overweight at age 5 years compared with eating breakfast at these ages (OR 0.72; 95% CI 0.15 to 3.49; p-value NR). The analysis was adjusted for birth weight, maternal education, parental BMI when children were aged 2 and 5 years, and household type.

## **Summary: children's eating behaviours and body composition or weight status**

- 7.36 The evidence identified from SRs on the relationship between children's eating behaviours and body composition or weight status is summarised in Table 7.1.

**Table 7.1. Summary of the evidence on children’s eating behaviours and body composition or weight status.**

Exposure	Outcome	Direction of association	Certainty of evidence
Food fussiness	Body Mass Index (BMI) z-score	No association	Limited
Food fussiness	Change in BMI or standardised weight	Not applicable	Insufficient
Food fussiness	Odds of underweight	Not applicable	Insufficient
Inability to delay gratification	Risk of overweight	Not applicable	Insufficient
Inability to delay gratification	Change in BMI z-score	Not applicable	Insufficient
Skipping breakfast versus eating breakfast	Odds of overweight	Not applicable	Insufficient

- 7.37 The available evidence from SRs examining the association between eating behaviours in children aged 1 to 5 years and body composition or weight status is from 3 SRs without MAs, 1 given a moderate confidence rating using the AMSTAR 2 tool, and 2 given critically low confidence ratings.
- 7.38 Evidence from 3 PCS included in Brown et al (2016) and Kininmonth et al (2021) suggests that there is no association between food fussiness in children aged 1 to 5 years and BMI z-score, adjusted for child baseline BMI. The evidence was graded ‘limited’ due to small number of PCS and lack of reporting of quantitative findings to judge confidence intervals.
- 7.39 There was ‘insufficient’ evidence from SRs to enable conclusions to be drawn on any relationship between the other eating behaviours examined (ability to delay gratification and skipping breakfast versus eating breakfast) in children aged 1 to 5 years and body composition or weight status in childhood or adolescence as fewer than 3 primary studies included in the SRs examined these relationships.

# Systematic review evidence identified on caregiver feeding practices and styles on children's food acceptance, dietary intake and health outcomes

- 7.40 Two SRs with MAs (Hodder et al, 2020; Nekitsing et al, 2018) and 8 SRs without MAs (Appleton et al, 2018; Bergmeier et al, 2015; Hurley et al, 2011; Mikkelsen et al, 2014; Mura Paroche et al, 2017; Osei-Assibey et al, 2012; Russell et al, 2016; Ward et al, 2015) were identified that examined the effect of caregiver or parental feeding practices and styles on children's acceptance of foods, dietary intake, and body composition or weight status.
- 7.41 Most of the primary studies included in the SRs examined the efficacy of interventions to increase children's acceptance, preference or consumption of vegetables or fruit. Interventions included: repeated exposure (taste or visual) to the target food(s); pairing of the target food(s) with liked foods, additional flavours or dietary energy; modelling the eating of target food(s) by adults or peers; and use of rewards (food and non-food) to reinforce or encourage eating of the target food(s).
- 7.42 Studies took place in a mix of settings including the child's home, childcare centres or preschools, as well as laboratory settings. Study designs varied considerably: RCTs, non-randomised controlled trials, quasi-experimental designs, and pre-post designs.
- 7.43 Several SRs (Bergmeier et al, 2015; Hurley et al, 2011; Russell et al, 2016) also included PCS that examined the potential longitudinal influence of parental feeding practices on children's dietary intake or body composition or weight status.
- 7.44 Most of the studies included in the SRs in children aged 1 to 5 years were conducted in high-income countries (HIC) (defined according to the [World Bank classification system](#)), including the UK.
- 7.45 Details of the SRs can be found in Annex 5 (Table A5.4). Quality assessment of the SRs using the AMSTAR 2 tool can be found in Annex 8 (Table A8.8). Additional data extracted on the primary studies can be found in Annex 9 (Tables A9.34 to A9.37). The criteria used to grade the evidence are provided in chapter 2 (Table 2.4, paragraphs 2.54 to 2.59). Summary tables of the evidence grading process for this section are provided in Annex 10 (Tables A10.19 to A10.23 and Table A10.36).

## Limitations of the systematic review evidence on feeding practices and styles

- 7.46 All the primary studies included in the SRs or MAs examined the effect of caregiver or parental feeding practices on children's eating behaviours and body weight in the short term (less than 12 months). While these studies may be useful in demonstrating the possibility of influencing children's eating behaviours in the short term, they do not enable conclusions to be drawn on the longer term impact of such practices.
- 7.47 Most primary studies had small sample sizes. Whether they were adequately powered was either not considered or reported in the SR.
- 7.48 Risk of publication bias was evident in 1 SR with MA (Nekitsing et al, 2018) that formally assessed this, indicating that statistically significant findings may have had a greater likelihood of being published and included in SRs (Higgins et al, 2022).
- 7.49 SRs did not always report or consider whether non-randomised studies of interventions (NRSI) and PCS adjusted for potential confounding factors, such as child weight status and eating behaviours at baseline. For example, PCS that report associations between parental restrictive practices around food and higher risk of overweight in children may be interpreted as evidence that parental restrictive practices increase the risk of overweight in children. Yet the opposite may be the case (reverse causality); parents may be more likely to employ restrictive practices around food if their child overeats and has overweight at baseline. The same may be said of associations between pressuring a child to eat and risk of child underweight. Children with underweight at baseline may be more likely to be pressured to eat rather than the other way around.
- 7.50 Primary studies were limited in some instances by not using validated methods to measure parental or caregiver feeding styles or practices. In addition, self-report questionnaires that are validated may still be subject to reporting bias and impression management. Even the most widely used self-report measure of parental feeding practices, the Child Feeding Questionnaire (Birch et al, 2001), was found not to be aligned with observational measures of parental feeding (Bergmeier et al, 2015; Hurley et al, 2011).
- 7.51 Ideally, mealtime interactions should be assessed in relation to both parent and child responsiveness during feeding (Bergmeier et al, 2015). However, none of the primary studies examined mutual parent-child responsiveness during feeding (Bergmeier et al, 2015).
- 7.52 Despite survey data showing that young children from lower SES backgrounds in HIC (including the UK) eat, on average, fewer vegetables and fruit than children from the least deprived households (see [Vegetables and fruit consumption in the](#)

[UK](#), chapter 5), only 1 SR (Hodder et al, 2020) specifically examined interventions to increase vegetable and fruit consumption in these children.

- 7.53 No SR evidence was identified on the influence of parental feeding styles (authoritative, authoritarian, indulgent, uninvolved) on children's acceptance or consumption of food and only 1 SR (Bergmeier et al, 2015) included some evidence on the impact of parental feeding styles on children's body composition.

## **Caregiver feeding practices on increasing children's acceptance or consumption of fruit or vegetables**

- 7.54 Two SRs with MAs (Hodder et al, 2020; Nekitsing et al, 2018) examined the effectiveness of feeding practices on increasing vegetable consumption in children aged up to 5 years. Intervention strategies included repeated taste exposure, pairing vegetables with positive stimuli, and general advice on introducing solid foods.
- 7.55 Hodder et al (2020)(AMSTAR 2 confidence rating: high) included 38 RCTs in children aged up to 5 years. Findings from 19 RCTs were pooled into a MA. The MA reported that feeding practices collectively increased children's vegetable consumption compared with no intervention, with medium heterogeneity (SMD 0.50; 95% CI 0.29 to 0.71;  $p < 0.00001$ ;  $I^2 = 77\%$ ; random-effects model; 19 RCTs, 2140 participants). The SR stated that this effect size was equivalent to an increase of 5.30g as-desired consumption of vegetables. Seventy-six percent of the weighting of the MA was from RCTs in children aged 1 to 5 years. The intervention duration was up to 6 months (mean duration: 8.3 weeks).
- 7.56 Findings from a sensitivity analysis that excluded trials that were at high risk of bias were similar (SMD 0.54; 95% CI 0.18 to 0.90;  $p$ -value NR;  $I^2 = 77\%$ ; random-effects model; 8 RCTs, 701 participants) as were the findings of a sensitivity analysis in trials with low attrition or high attrition with intention-to-treat analysis (SMD 0.49; 95% CI 0.22 to 0.77;  $p$ -value NR;  $I^2 = 71\%$ ; random-effects model; 11 RCTs, 971 participants). There was no evidence of publication bias.
- 7.57 Hodder et al (2020) also performed a subgroup MA in children aged >12 months and up to 5 years, and reported a larger effect size (SMD 0.58; 95% CI 0.34 to 0.83;  $p < 0.00001$ ;  $I^2 = 72\%$ ; random-effects model; 15 RCTs, participants NR).
- 7.58 Nekitsing et al (2018) (AMSTAR 2 confidence rating: low) also reported that feeding practices collectively increased vegetable consumption in children under 5 years compared with the control group, with medium heterogeneity (SMD: 0.40; 95% CI 0.31 to 0.50;  $p < 0.001$ ;  $I^2 = 73.4\%$ ; random-effects model; 30 intervention studies, 4017 participants). The intervention duration was up to 8 months. The

effect was slightly larger when estimates from 44 intervention arms across the 30 studies were pooled (SMD: 0.42; 95% CI 0.33 to 0.51;  $p < 0.001$ ;  $I^2 = 69.1\%$ ; random-effects model; 4017 participants). Intervention strategies included repeated taste or visual exposure, pairing vegetables with liked foods or additional flavours or dietary energy, modelling of vegetable consumption and offering non-food rewards (for example, praise or a toy or sticker).

- 7.59 However, these findings should be interpreted with caution given evidence of publication bias that may have inflated the effect size. By the review authors' estimation, correcting for this bias would likely reduce the effect size to SMD 0.31 (95% CI 0.21 to 0.41;  $p$ -value NR;  $I^2$  NR; random-effects model).
- 7.60 A subgroup analysis conducted by Nekitsing et al (2018) reported that feeding practices increased consumption of unfamiliar or previously disliked vegetables to a greater extent than consumption of familiar or liked vegetables (SMD: 0.58; 95% CI 0.44 to 0.73; 9 studies, 1058 participants versus SMD: 0.31; 95% CI 0.21 to 0.40; 21 studies, 2959 participants;  $p = 0.002$  for difference between subgroups). However, 8 of the 9 studies that examined unfamiliar or previously disliked vegetables used repeated taste exposure in their intervention. The SR concluded that it was not possible to determine whether the observed increase in consumption of unfamiliar or previously disliked vegetables was due to the type of vegetable that was tested (unfamiliar or previously disliked) or due to the intervention strategy employed (repeated taste exposure).

## **Feeding practices to increase vegetable or fruit consumption and deprivation**

- 7.61 Hodder et al (2018) included 2 RCTs that specifically recruited children aged 1 to 5 years from predominantly economically or socially disadvantaged backgrounds. Due to methodological reasons, the results of these 2 studies were not included in a MA and quantitative results were not reported for either study.
- 7.62 One RCT (in 216 participants, aged 4 to 5 years, eligible for free school meals in the UK) reported that a 3 week intervention that involved repeated food exposure coupled with a non-food reward significantly increased the consumption of a target vegetable in a school setting. The other RCT (in 240 participants, aged 3 to 5 years, from low-income households) reported that two 8-week interventions that included either provision of vegetables and fruit alone or together with parental and child nutrition education (which included tastings of target fruits and vegetables) increased fruit and vegetable consumption (assessed via skin carotenoid levels compared with no intervention).

## Repeated taste exposure on children's vegetable consumption

- 7.63 The SACN report '[Feeding in the first year of life](#)' reported that repeated exposure to a variety of vegetables in infants during complementary feeding improved later acceptance of vegetables (SACN, 2018).
- 7.64 For this report, 1 SR with MA (Nekitsing et al, 2018) and 1 SR without MA (Mura Paroche et al, 2017) were identified that examined the short term (less than 12 months) effectiveness of repeated taste exposure on increasing vegetable consumption in children up to age 5 years.
- 7.65 Nekitsing et al (2018) (AMSTAR 2 confidence rating: low) reported that repeated taste exposure (alone or combined with other intervention strategies) increased children's vegetable consumption compared with the control group in the shorter term (up to 8 months) (SMD 0.57; 95% CI 0.43 to 0.70; p-value NR; I<sup>2</sup>=52%; 10 intervention studies, participants NR). The effect size was larger when intervention arms that included repeated taste exposure only were pooled (SMD 0.79; 95% CI 0.53 to 1.05; p-value NR; I<sup>2</sup> NR; 5 intervention arms, 134 participants).
- 7.66 A meta-regression analysis suggested that the number of taste exposures was associated with increased vegetable consumption (beta coefficient 0.035; 95% CI 0.00 to 0.06; p=0.01; 10 intervention studies, participants NR). For a significant improvement in vegetable consumption (a moderate effect size of 0.5), children would require approximately 8 to 10 taste exposures.
- 7.67 As most of the 10 studies exposed children to a single vegetable, the findings do not indicate whether increased acceptance or consumption of one vegetable after repeated taste exposure generalises to acceptance or consumption of another vegetable.
- 7.68 The findings also do not demonstrate whether taste exposure is the most effective strategy in children who score high for food neophobia or food fussiness (paragraphs 7.3 and 7.4) or whether taste exposure is equally effective at increasing acceptance or consumption of vegetables across the 1 to 5-year age group.
- 7.69 Mura Paroche et al (2017) (AMSTAR 2 confidence rating: critically low) included 1 additional multi-centre intervention study (in 332 participants, aged 4 to 38 months) that reported that 5 to 10 taste exposures to an unfamiliar vegetable increased intake of that vegetable 2 weeks after the intervention (quantitative findings NR). The study also reported that the effectiveness of repeated taste exposure appeared to diminish after the child reached age 24 months.

## **Repeated taste exposure to a variety of textures (vegetables or fruit)**

- 7.70 Mura Paroche et al (2017) included 2 intervention studies (design unspecified) that examined the effect of repeated taste exposure on children's acceptance of new textures (vegetable or fruit). Both studies (in a total of 82 participants, aged 12 to 22 months) reported that repeated taste exposure to a variety of textures increased subsequent acceptance of complex textures (chopped or lumpy) compared with simpler textures (purée) (quantitative findings not reported).

## **Repeated taste exposure and pairing on children's vegetable consumption**

- 7.71 One SR with MA (Nekitsing et al, 2018) and 1 SR without MA (Mura Paroche et al, 2017) examined whether repeated exposure to vegetables paired with liked foods, additional flavours or dietary energy increased vegetable consumption in the short term (less than 12 months) in children aged 5 years and under.
- 7.72 Nekitsing et al (2018) (AMSTAR 2 confidence rating: low) reported that, in the short term (less than 8 months), repeated taste exposure to vegetables paired with liked foods, additional flavours or dietary energy, increased vegetable consumption compared with no intervention (SMD: 0.43; 95% CI 0.26 to 0.61; I<sup>2</sup> NR; 8 intervention arms, 358 participants). However, pairing the vegetables with liked foods, additional flavours or dietary energy was less effective at increasing vegetable consumption than repeated exposure to the vegetable in its plain form (see paragraph 7.65). The comparison between the two intervention strategies should be interpreted with caution because Nekitsing et al (2018) did not report performing a formal statistical comparison between the two.
- 7.73 Mura Paroche et al (2017) (AMSTAR 2 confidence rating: critically low) included 1 additional multicentre intervention study (in 332 participants, age 4 to 38 months) that reported that repeated taste exposure to a vegetable paired with added dietary energy (144kcal per 100g from sunflower oil) was less effective at increasing vegetable consumption than repeated taste exposure to the vegetable in plain form (quantitative findings not reported).

## **Repeated visual exposure on children's acceptance or taste preference for fruit or vegetables**

- 7.74 Mura Paroche et al (2017) (AMSTAR confidence rating: critically low) included 2 intervention studies (design unspecified) that examined the effect of repeated visual exposure on acceptance of or preference for unfamiliar fruit in children aged 1 to 5 years. No quantitative data was reported for either study. One study (in 20 participants, aged 21 to 24 months) reported that visual exposure to an unfamiliar fruit increased children's willingness to taste the fruit compared with no visual exposure. The other study (in 43 children, aged 23 to 69 months) reported that

visual exposure to an unfamiliar fruit enhanced children's visual preference for the fruit but did not correlate with their taste preferences for that fruit. This finding indicates that to enhance taste preferences, exposure to a food may need to occur in the relevant sense modality.

- 7.75 One of the studies (in 20 participants, described in paragraph 7.74), and an additional intervention study (design unspecified, from the same research group), also examined the effect of repeated visual exposure on children's acceptance of, or preference for, vegetables (familiar and unfamiliar). Both studies (in a total of 68 participants, aged 20 to 24 months) reported that children were more easily persuaded to eat the target (exposed) vegetable than a control (non-exposed) vegetable, and that the effect was strongest for initially unfamiliar vegetables (compared with initially familiar and liked or disliked vegetables). One of the studies (in 20 participants) also reported that children unexpectedly decreased their willingness to taste a familiar vegetable after repeated visual exposure, although the reasons for this were not explored by Mura Paroche et al (2017).

## **Summary: Feeding practices on increasing children's consumption of fruit or vegetables (short term, up to 8 months)**

- 7.76 The evidence identified from SRs on the effect of feeding practices (collectively and specific feeding practices) on increasing children's consumption of vegetables or fruit in the short term (up to 8 months) is summarised in Table 7.2.
- 7.77 The available evidence from SRs examining the effect of feeding practices (collectively and specific feeding practices) on increasing children's vegetable or fruit consumption in children aged 1 to 5 years is from 3 SRs (2 with MAs), 1 given a high confidence rating using the AMSTAR 2 tool, 1 given a low confidence rating, and 1 given a critically low rating.

**Table 7.2. Summary of the evidence of the effect of feeding practices on increasing children’s vegetable or fruit consumption in the short term (up to 8 months)**

Intervention <sup>1</sup>	Outcome	Direction of effect <sup>5</sup>	Certainty of evidence
Feeding practices (collectively) <sup>2</sup>	Vegetable consumption	↑	Moderate
Feeding practices <sup>3</sup> and social or economic deprivation	Vegetable or fruit consumption	Not applicable	Insufficient
Repeated taste exposure	Vegetable consumption	↑	Moderate
Repeated taste exposure	Acceptance of textures (vegetable or fruit)	Not applicable	Insufficient
Repeated taste exposure plus pairing <sup>4</sup>	Vegetable consumption	↑	Moderate
Repeated visual exposure	Preference or acceptance (fruit)	Not applicable	Insufficient
Repeated visual exposure	Preference or acceptance (vegetable)	Not applicable	Insufficient

<sup>1</sup> Compared with a control group (no intervention, usual care, or treatment received after the intervention phase).

<sup>2</sup> Includes repeated taste or visual exposure, pairing with positive stimuli such as liked foods, modelling of vegetable consumption, offering the child non-food rewards (for example, praise or a sticker or toy).

<sup>3</sup> Includes repeated taste exposure, non-food rewards, vegetable and fruit provision, child and parent nutrition education.

<sup>4</sup> Repeated taste exposure to vegetables that were paired with liked foods, or additional flavours or dietary energy.

<sup>5</sup> Direction of effect for reported outcomes: ↑increase

## **Summary: feeding practices (collectively)**

7.78 Evidence from the MA conducted by Hodder et al (2020) suggests that feeding practices (repeated exposure, pairing vegetables with positive stimuli, and general infant and young child feeding practices) can increase vegetable consumption in children up to the age of 5 years in the short term (up to 6 months) by approximately 5.30g of as-desired vegetable consumption. These findings were supported by a subgroup MA in children aged 1 to 5 years, and sensitivity analyses that excluded studies at high risk of bias or included studies with low attrition or high attrition with intention-to-treat analysis conducted by Hodder et al

(2020), as well as the MA of feeding practice interventions (lasting up to 8 months) that was conducted by Nekitsing et al (2018). The evidence was graded 'moderate'. Evidence of publication bias reported in Nekitsing et al (2018) together with a small effect size and non-specificity of interventions prevented the evidence from being graded 'adequate'.

- 7.79 There was 'insufficient' evidence from SRs to enable any conclusions to be drawn on the effectiveness of feeding practice interventions in children from socially or economically disadvantaged backgrounds (in school settings) as fewer than 3 primary studies included in the SRs examined these relationships.
- 7.80 No evidence from SRs was identified on the longer term impact of feeding practices on children's vegetable or fruit consumption.

### **Summary: repeated exposure**

- 7.81 Evidence from a subgroup MA conducted by Nekitsing et al (2018) suggested that repeated taste exposure is the most effective feeding practice at increasing vegetable consumption in children aged up to 5 years in the short term (up to 8 months). Nekitsing et al (2018) estimated that 8 to 10 taste exposures are required for a significant improvement in vegetable consumption and that the average increase in vegetable consumption after repeated taste exposure is 67g of vegetables (or approximately 1.5 portions for a child aged 2 to 5 years). However, the effect size may have been overestimated given evidence of publication bias. In addition, as most of the studies included in the MA exposed children to a single vegetable, the findings do not reveal whether increased acceptance or consumption of one vegetable after repeated taste exposure generalises to acceptance or consumption of another vegetable. The findings also do not demonstrate whether taste exposure is the most effective strategy in children who score high on food neophobia or food fussiness (paragraphs 7.3 and 7.4) or whether taste exposure is equally effective at increasing acceptance or consumption of vegetables across the 1 to 5-year age group. The evidence on repeated taste exposure and increasing vegetable consumption was graded 'moderate'. Evidence of publication bias prevented the evidence from being graded 'adequate'.
- 7.82 There was also 'moderate' evidence that repeated taste exposure to vegetables paired with liked foods or additional flavours or nutrients increases vegetable consumption. This strategy may be less effective in increasing vegetable consumption than repeated taste exposure to vegetables in their plain form. However, without a formal statistical comparison between the 2 strategies, firm conclusions cannot be drawn.
- 7.83 There was 'insufficient' evidence from SRs to enable conclusions to be drawn on any effect of repeated taste exposure on the acceptance of new textures (vegetable or fruit) or repeated visual exposure on increasing acceptance of or

preference for vegetables or fruit as there were fewer than 3 primary studies included in the SRs that examined these relationships.

## **Caregiver feeding practices on children's acceptance or consumption of food**

- 7.84 Four SRs without MAs (Mikkelsen et al, 2014; Mura Paroche et al, 2017; Osei-Assibey et al, 2012; Ward et al, 2015) included studies that examined the effect of feeding practices on the acceptance or consumption of foods in children aged 1 to 5 years. Feeding practices were divided into those intended to restrict food consumption (parental restriction) and those intended to increase food acceptance or consumption (for example, modelling, use of rewards, verbal encouragement, offering choice). Target foods included fruit (dried or fresh), vegetables, grains, and snack foods (for example, crackers).

### **Caregiver feeding practices to restrict food consumption**

#### **Restriction**

- 7.85 Osei-Assibey et al (2012) (AMSTAR 2 confidence rating: low) included 1 nested non-randomised controlled trial (in 70 participants, aged 4 to 6 years) that reported that parental restriction (measured by the Child Feeding Questionnaire) was not associated with children's total dietary energy intake (TDEI) during an ad libitum meal in a laboratory setting ( $p=0.5$ ; other quantitative data not reported).

### **Caregiver feeding practices to increase food acceptance or consumption**

#### **Adult modelling**

- 7.86 Ward et al (2015) (AMSTAR confidence rating: moderate) included 2 quasi-experimental studies that examined the effect of adults modelling the eating of familiar or unfamiliar foods (including vegetables and fruit) in silence ('silent modelling') compared with visually exposing children to the target foods in a preschool setting in the short term (less than 12 months). Both studies (in a total of 71 participants, preschool age not specified) reported that silent modelling by a teacher was not more effective than visual exposure for increasing acceptance of familiar or unfamiliar foods (see Annex 9, Table A9.36 for detailed results). One of these studies (in 40 participants, preschool age not specified) also reported that enthusiastic modelling by a teacher was more effective in increasing acceptance of unfamiliar foods (including vegetables and fruit) than simple exposure (MD 5.08 bites of new foods; 95% CI not reported;  $p<0.03$ ). However, after adjusting for the

effect of modelling by the children's peers, any independent effect of enthusiastic modelling disappeared (effect size NR;  $p=0.35$ ).

- 7.87 Mura Paroche et al (2017) (AMSTAR confidence rating: critically low) included 3 additional intervention studies (design unspecified) and 1 PCS that examined the effect of adult modelling on children's food acceptance or consumption of unfamiliar foods in the short term. Quantitative data were not reported for any of the 4 studies.
- 7.88 Two of the intervention studies (in a total of 107 participants, aged 14 to 48 months in 1 study; and aged 2 to 5 years in the second study) reported that adult modelling of unfamiliar foods (unspecified in one study, semolina in the second study) increased children's acceptance or consumption of those foods compared with simple exposure, not modelling, or modelling a different food, independently of setting (home or school). One of the studies reported that the modelling effect did not differ by the child's age or early feeding practices while the other study reported that the effect was strongest in girls and when the modeller was the child's mother (rather than a 'visitor'). The third intervention study (in 60 families with children aged 12 to 36 months) reported that parental modelling of an unfamiliar vegetable or fruit in a home setting was not more effective in increasing consumption of the target food compared with a 'neutral' prompt (for example, "eat your peas" spoken in a neutral or positive tone of voice). The PCS (in 156 participants, mean age 3.3 years at baseline) reported that maternal modelling of healthy eating was inversely associated with child food fussiness (paragraphs 7.3 and 7.4) 1 year later after adjusting for food fussiness at baseline, age, sex and maternal characteristics (age, BMI and education). Maternal modelling of healthy eating was assessed through self report rather than observation.
- 7.89 These results should be interpreted with caution as modelling consumption of familiar or unfamiliar foods, including vegetables and fruit, under experimental conditions is systematic but exaggerated and does not reflect everyday modelling of food consumption in the home. Observational evidence indicates that parental modelling at home can be a potent predictor of children's vegetable and fruit consumption (Brown & Ogden, 2004; Hart et al, 2010; Palfreyman et al, 2014).

### **Peer modelling**

- 7.90 Two SRs without MAs (Mikkelsen et al, 2014; Mura Paroche et al, 2017) included studies that examined the effect of peer modelling on food acceptance or consumption in children aged 1 to 5 years in the short term.
- 7.91 Mikkelsen et al (2014) (AMSTAR confidence rating: low) included 1 quasi-experimental study (in 38 participants, aged 3 to 6 years, duration unclear) that reported that female peer models were more effective than male peer models at increasing acceptance of a selection of unfamiliar fruit (measured by the number of bites taken of the fruit) in children of either gender in a school setting (quantitative

findings not reported). However, the effect disappeared 1 month after the study completed.

- 7.92 Mura Paroche et al (2017) (AMSTAR confidence rating: critically low) included 2 additional intervention studies (design unspecified) in a school setting. Both studies (in a total of 93 participants, aged 2 to 6.5 years, 2 to 4 day duration) reported that peer modelling increased children's preference for or consumption of the modelled food (vegetables in 1 study, crackers in the other study), although in one of the studies (in 39 children, aged 2 to 4 years, 4 day duration), the effect was stronger in the younger children (age unspecified) enrolled in the study compared with the older children (quantitative findings not reported).

### **Use of rewards (food or non-food)**

- 7.93 Ward et al (2015) (AMSTAR confidence rating: moderate) included 2 intervention studies (1 quasi-experimental, 1 pre-post design) that examined the effect of using rewards (food or non-food) on increasing acceptance or consumption of vegetables or fruit in preschool children (exact age not specified) in the short term. Both studies (in a total of 33 participants) reported that use of rewards (food or non-food) increased acceptance or consumption of unfamiliar vegetables or fruits compared with either simple exposure or no reward. One study (in 14 participants, 3-day duration) reported a mean difference in the total number of bites of unfamiliar vegetables and fruit (across 3 meal occasions) of 11.55 (95% CI NR;  $p < 0.02$ ). The other study (in 19 participants, 3-week duration) reported a mean difference in consumption ranging from 14 to 21g of different vegetables (95% CI NR;  $p < 0.05$ ).

### **Verbally encouraging a child to eat**

- 7.94 Ward et al (2015) (AMSTAR 2 confidence rating: moderate) included 1 quasi-experimental study (in 14 participants, preschool age not specified, in a school setting, 3 day duration) that reported that teachers who asked children to "try one bite" of a selection of unfamiliar vegetables and fruit were: more effective at increasing the number of foods children sampled with at least 1 bite (MD 1.85; 95% CI NR;  $p < 0.007$ ); number of meals during which at least 1 of the unfamiliar foods was sampled (MD 1.45; 95% CI NR;  $p < 0.001$ ); and total number of bites of new foods (across 3 study meals) (MD 5.55; 95% CI NR;  $p < 0.02$ ) compared with simply exposing the children to the target foods.

### **Choice offering**

- 7.95 Ward et al (2015) (AMSTAR confidence rating: moderate) included 1 quasi-experimental study (in 10 participants, preschool age not defined, 3 day duration) that reported that children given a choice of unfamiliar vegetables and fruit in a school setting, increased the number of foods they sampled with at least 1 bite (MD 1.7; 95% CI NR;  $p < 0.007$ ), number of meals during which at least 1 of the

unfamiliar foods was sampled (MD 1.0; 95% CI NR;  $p < 0.02$ ), and total number of bites of unfamiliar foods (across 3 study meals) (MD 21.75; 95% CI NR  $p < 0.007$ ) compared with simply exposing the children to the unfamiliar foods.

## Summary: Caregiver feeding practices on children’s food acceptance or consumption

- 7.96 The evidence identified from SRs on the effect of caregiver feeding practices on children’s food acceptance or consumption in the short term (less than 12 months) is summarised in Table 7.3.

**Table 7.3. Summary of the evidence on caregiver feeding practices on children’s food acceptance or consumption (short term, less than 12 months)**

Intervention	Outcome	Direction of effect	Certainty of evidence
Restriction	Dietary energy intake	Not applicable	Insufficient
Adult modelling	Food acceptance or consumption	Inconsistent	Inconsistent
Peer modelling	Food acceptance or preference or consumption	Not applicable	Insufficient
Use of rewards	Food acceptance or consumption	Not applicable	Insufficient
Verbal encouragement	Food acceptance or consumption	Not applicable	Insufficient
Choice offering	Food acceptance or consumption	Not applicable	Insufficient

- 7.97 The available evidence from SRs examining the effect of feeding practices on food acceptance or consumption in children aged 1 to 5 years in the short term (less than 12 months) is from 4 SRs without MAs, 1 given a moderate confidence rating using the AMSTAR 2 tool, and 3 given low confidence ratings.
- 7.98 Evidence from 5 small intervention studies and 1 PCS included in 2 SRs by Ward et al (2015) and Mura Paroche et al (2017) on the effect of adult modelling on children’s food acceptance or consumption in the short term was ‘inconsistent’. Three intervention studies reported no difference in effect on children’s food acceptance or consumption between adult modelling compared with simple exposure or a neutral prompt while 2 intervention studies reported that adult modelling increased children’s food acceptance or consumption compared with

simple exposure or modelling with foods different from the target food. The PCS reported an inverse association between adult modelling of healthy eating and children's food fussiness (paragraphs 7.3 and 7.4).

- 7.99 Evidence from 3 intervention studies included in 2 SRs by Mikkelsen et al (2014) and Mura Paroche et al (2017) on the effect of peer modelling on increasing children's food acceptance or consumption in the short term was graded 'insufficient' due to the lack of quantitative data to judge effect sizes, small sample sizes, and lack of information on study power, publication bias and confounding.
- 7.100 There was 'insufficient' evidence from SRs to enable conclusions to be drawn on the effect of parental feeding practices to reduce or increase children's food or energy intake as fewer than 3 primary studies included in the SRs examined these relationships.

## **Caregiver feeding practices on children's preference for and consumption of sweet foods and beverages**

- 7.101 Infants readily accept sweet taste and have the ability to distinguish quantitative differences between different sugar solutions, demonstrating a preference for sweeter solutions and those with higher sweetening power (Desor et al, 1977; Desor et al, 1973; Ganchrow et al, 1983).
- 7.102 One SR without MA (Appleton et al, 2018) examined whether exposure to sweet taste in early childhood maintains or even promotes a generalised desire for sweet foods and beverages.
- 7.103 Appleton et al (2018) (AMSTAR 2 confidence rating: moderate) identified 2 controlled trials that examined the effect of exposure to sweet foods on subsequent generalised preference for the same or other sweet foods in the short term (2 days to 9 weeks). Quantitative data were not reported for either study.
- 7.104 In one trial (in 39 participants, mean age 55 months), children's preference for an unfamiliar sweet food increased over 15 exposures to that food, however, the increased preference for the sweet food had no effect on preference for other unfamiliar sweet foods. The other trial (in 53 children, mean age 3 years) reported that unrestricted access to a sweet food decreased preoccupation with the food (in terms of demanding and consumption of the food) by the end of the 2 day experiment. This decrease was greater than in children whose access to the sweet food was restricted over the same period. However, children with unrestricted access to the target sweet food increased their demands for (but not consumption of) other sweet foods compared with children with restricted access to the target food.

- 7.105 In addition, Appleton et al (2018) identified 2 PCS that examined whether exposure to sugar-sweetened beverages (SSBs) and fruit juice was associated with later consumption of these beverages. Quantitative data were not reported for either study.
- 7.106 One PCS (in 1163 participants) reported that higher consumption of fruit juice (but not water, in ounces per day) at age 1 year was associated with increased consumption of SSBs and fruit juice (in servings per day) at ages 3 and 7 years after adjusting for baseline child weight-for-length z-score, age, sex, ethnicity, SES and maternal characteristics (age, education). The other PCS (in 493 participants) reported that higher SSB consumption (frequency of consumption) at ages 16 to 24 months was associated with increased SSB consumption (grams per 1000 kcal per day) approximately 2 years later, after adjusting for age, sex, current but not baseline body weight, SES and multiple maternal characteristics.
- 7.107 While these PCS may demonstrate that consumption of SSBs or fruit juice at an early age tracks onto consumption of these beverages in later childhood, it is unclear whether the early exposure to SSBs or fruit juice is associated with increased preference or liking for sweet-tasting foods and beverages. The SR commented that differences in dietary consumption of sweet beverages may have reflected parenting practices and household food offerings rather than preferences for specific sensory attributes. Preferences for sweet taste, though innate, may also reduce with age, and therefore effects demonstrated in childhood may not transfer to adulthood. Appleton et al (2018) did not identify any studies that examined whether exposure to sweet taste in childhood shapes taste preferences in the longer term.

## **Summary: Caregiver feeding practices on children's preference for and consumption of sweet foods and beverages**

- 7.108 The evidence identified from SRs on the effect of feeding practices on children's preference for, and consumption of, sweet foods and beverages in the short term (less than 12 months) is summarised in Table 7.4.

**Table 7.4. Summary of the evidence on caregiver feeding practices on children’s preference for and consumption of sweet foods and beverages (short term, less than 12 months)**

Intervention or exposure	Outcome	Direction of association	Certainty of evidence
Exposure to sweet food	Preference for or consumption of sweet foods	Not applicable	Insufficient
Exposure to sugar-sweetened beverages or fruit juice	Consumption of sugar-sweetened beverages or fruit juice	Not applicable	Insufficient

- 7.109 The available evidence on the effect of sweet taste exposure on the development of children’s preferences for, or consumption of, sweet foods and beverages in the diet is from 1 SR without MA given a moderate confidence rating using the AMSTAR 2 tool.
- 7.110 There was ‘insufficient’ evidence from SRs to enable conclusions to be drawn on any effect of exposure to sweet foods in children aged 1 to 5 years on subsequent generalised preference of sweet foods or any relationship between exposure to SSBs or fruit juice in early childhood and consumption of SSBs or fruit juice in later childhood, as fewer than 3 primary studies included in the SRs examined these relationships.

## Caregiver feeding practices on children’s body composition

### Restrictive feeding practices

- 7.111 Two SRs without MAs (Hurley et al, 2011; Russell et al, 2016) included studies that examined the effect of caregiver or parental restrictive feeding practices on body composition of children aged 1 to 5 years. All adjusted for baseline child body composition reducing the likelihood that observed associations reflect reverse causality (see paragraph 7.49).
- 7.112 Russell et al (2016) (AMSTAR 2 confidence rating: moderate) included 1 PCS (in 1797 participants, aged 1 to 5 years) that reported that parental restrictive feeding practices were not associated with the monthly change in children’s BMI z-scores from age 1 to 5 years (quantitative findings NR). The analysis was adjusted for sex, ethnicity, baseline weight-for-height z-score and food consumption (servings per day).

- 7.113 Hurley et al (2011) (AMSTAR 2 confidence rating: critically low) included 1 PCS (in 62 mother-child dyads) that reported that parental restriction at age 1 year (measured by the Child Feeding Questionnaire) predicted lower child standardised weight at age 2 years, after adjusting for baseline child weight at age 1 year (quantitative findings NR).

## **Pressuring a child to eat**

- 7.114 Two SRs without MAs (Bergmeier et al, 2015; Hurley et al, 2011) included studies that examined the effect of pressuring a child to eat on their body composition when aged 1 to 5 years.
- 7.115 Bergmeier et al (2015) (AMSTAR 2 confidence rating: critically low) included 1 PCS (in 1218 participants) that reported that assertive prompting to eat during video-recorded eating sessions between mother and child at ages 15, 24 and 36 months was directly associated with child adiposity across those ages (quantitative findings NR). Assertiveness was defined in the study as prompting a child to eat using verbal or physical encouragement. Child adiposity was defined in the study as the weight-for-length z-score (WLZ) at age 15 months combined with BMI z-scores at ages 24 and 36 months. The analyses were adjusted for sex, age, ethnicity, SES, and maternal characteristics (education, weight status and depressive symptoms).
- 7.116 Hurley et al (2011) (AMSTAR 2 confidence rating: critically low) included 1 PCS (in 62 mother-child dyads) that reported that pressuring a child to eat (measured by the Child Feeding Questionnaire) at age 1 year predicted lower child standardised weight at age 2 years, after adjusting for baseline child weight at age 1 year (quantitative findings NR).

## **Caregiver feeding styles on children's body composition**

- 7.117 One SR without MA (Bergmeier et al, 2015) was identified that examined the effect of maternal feeding styles on child body composition during mother-child mealtime interactions.
- 7.118 Bergmeier et al (2015) (AMSTAR 2 confidence rating: critically low) included 1 PCS (in 1218 participants, aged 15 to 36 months) that reported that maternal intrusiveness during video-recorded eating sessions between mother and child at ages 15, 24 and 36 months was directly associated with child adiposity across those ages (quantitative findings NR). Intrusiveness was defined in the study as maternal behaviour that imposed the mother's agenda on the child (that is, was adult- rather than child-centred). Child adiposity was defined in the study as the weight-for-length z-score at age 15 months combined with BMI z-scores at ages

24 and 36 months. The analyses were adjusted for sex, age, ethnicity, SES, and maternal characteristics (education, weight status and depressive symptoms), but not child baseline weight status, indicating that the association may be a case of reverse causality.

7.119 No evidence from SRs was identified on the effect of responsive feeding styles (see paragraphs 7.9 and 7.10).

## Summary: Caregiver feeding practices and styles on children’s body composition

7.120 The evidence identified from SRs on the effect of caregiver feeding practices and styles on children’s body composition (short term, less than 12 months) is summarised in Table 7.5 and Table 7.6.

**Table 7.5. Summary of the evidence on caregiver feeding practices on children’s body composition**

Exposure	Outcome	Direction of association	Certainty of evidence
Restrictive feeding practices	Change in Body Mass Index (BMI) z-score	Not applicable	Insufficient
Pressuring a child to eat	Weight-for-length z-score and BMI z-score	Not applicable	Insufficient

**Table 7.6. Summary of the evidence on caregiver feeding styles on children’s body composition**

Exposure	Outcome	Direction of association	Certainty of evidence
Non-responsive feeding (intrusiveness)	Weight-for-length z-score and Body Mass Index (BMI) z-score	Not applicable	Insufficient
Responsive feeding	Body composition measures	Not applicable	No systematic review evidence identified

7.121 The available evidence from SRs examining the effect of feeding practices or styles on the body composition of children aged 1 to 5 years is from 3 SRs without

MAs, 1 given a moderate confidence rating using the AMSTAR 2 tool, and 2 given critically low confidence ratings.

- 7.122 There was 'insufficient' evidence from SRs to enable conclusions to be drawn on any effect of parental feeding practices (including parental restriction and pressuring a child to eat) or feeding styles on children's body composition as fewer than 3 primary studies included in the SRs examined these relationships.

# 8 Excess weight and obesity

## Background

- 8.1 Overweight and obesity are conceptually defined as 'abnormal or excessive fat accumulation that presents a risk to health' (SACN and RCPCH, 2012; WHO, 2020).
- 8.2 The most widely used indicator of overweight and obesity status is body mass index (BMI), calculated as body weight divided by height-squared. However, children and adolescents undergo a number of physiological changes as they grow, making it difficult for a single index to accurately represent weight-for-height across all age groups. Also, BMI provides no information about body shape, pattern of fat distribution or fat-to-lean mass ratio (SACN, 2011b). Research analysing body composition data in young children indicate limited agreement between high BMI categories and high body fat status (Wright et al, 2021). Nevertheless, BMI is still accepted as the most appropriate measure of weight status in children above age 2 years and adolescents (as in adults).
- 8.3 Although evidence suggests that higher BMI values in childhood are associated with adverse short- and long-term health effects (Reilly et al, 2003), data are currently insufficient to demonstrate a link between specific BMI values and levels of excess body fat that may lead to adverse health outcomes (SACN and RCPCH, 2012). Therefore, in practice, overweight and obesity in children are defined as having BMI values in the higher extremes of the general population.
- 8.4 As BMI in children varies with age and sex, standardised BMI centiles and Z-scores, which indicate the number of standard deviations (SD) a value is from the median, are generated by comparing BMI values against growth references that describe the normal distribution of BMI by both age and sex (Cole et al, 1995; Kuczmarski et al, 2002).
- 8.5 In the UK, the reference data used for BMI are the [UK-WHO growth charts](#) (RCPCH, 2013), which are based on the WHO Child Growth Standards, from 2 weeks to 4 years (WHO Multicentre Growth Reference Study Group 2006) and the UK 1990 (UK90) growth reference from ages 4 to 18 years (Freeman et al, 1995; Wright et al, 2010).
- 8.6 Differing BMI thresholds or cut-offs to define overweight and obesity are used for children in the UK. For individual children, the UK-WHO growth charts display lines for the 91st centile (+1.33 SD) and the 98th centile (+2 SD), and BMI values at or above these thresholds indicate overweight and very overweight (clinical obesity). Alternatively, public health surveillance programmes that monitor the prevalence of overweight and obesity in the UK (for example, the National Child Measurement

Programme [NCMP] in England) use the 85th centile (+1 SD) of the UK90 reference population data to indicate risk of overweight and the 95th centile (+1.65 SD) to indicate risk of obesity. These less stringent BMI thresholds maximise the statistical power to detect geographical and secular differences. It is important to note that the 85th and 95th centiles are intended for population monitoring use only and do not provide the number or percentage of individual children clinically defined as overweight or obese (PHE, 2016b). In addition, the NCMP; Scottish Health Survey (SHeS) and Public Health Scotland, which publishes BMI statistics for Scotland; and Child Measurement Programme in Wales; also classify children at or above the 99.6th centile (+2.67 SD) as having clinically severe obesity.

- 8.7 The WHO recommends using the less stringent BMI threshold for overweight (+1 SD) in school aged children and the more stringent threshold for obesity (+2 SD). For children younger than age 2 years, the WHO recommends even more stringent thresholds for overweight (+2 SD) and obesity (+3 SD) on the premise that obesity is intrinsically less prevalent in this age group.

## **Early life determinants of obesity**

- 8.8 Early life determinants of overweight or obesity in childhood and adulthood can be divided into those that are potentially modifiable and those that are not.
- 8.9 Several nutrition- and diet-related modifiable determinants, including infant feeding practices (breastfeeding relative to infant formula feeding) and maternal nutrition, were previously reviewed by SACN (SACN, 2011b; SACN, 2018). Other modifiable determinants include maternal characteristics (such as maternal weight status, gestational weight gain, smoking, physical activity and stress), the characteristics of the child's household (such as household socioeconomic status, food insecurity, access to healthy foods), as well as the wider sociocultural and physical environment (exposure to marketing and advertising of unhealthy foods, childcare attendance, and environmental toxins) (Brisbois et al, 2012; Monasta et al, 2010; Woo et al, 2016). However, it is not always clear whether such determinants are causally related to or merely predictive of a child's later risk of overweight or obesity.
- 8.10 Genetic susceptibility is a non-modifiable determinant of overweight and obesity (Elks et al, 2014). Genome-wide association studies indicate that common genetic variants associated with child BMI overlap with those associated with adult BMI (Alves et al, 2019) and that genetic variants associated with adult obesity risk begin to influence weight gain and body composition of children from infancy onwards (Alves et al, 2019; Elks et al, 2014). Even though genetic susceptibility is not modifiable, environmental factors can alter the effect of these genetic factors. For example, the Gemini birth cohort twin study in England and Wales reported that the heritability of BMI in children aged under 5 years was higher among those living in more obesogenic environments compared with less obesogenic

environments (Schrempft et al, 2018). It has therefore been suggested that modifying the early home environment so that it promotes a healthy weight may be particularly important for children with a genetic susceptibility to becoming overweight or obese.

- 8.11 For children and adolescents with overweight or obesity, evidence suggests that a range of diet, exercise and behavioural therapy interventions may help to reduce BMI or body weight (Salam et al, 2020). Furthermore, interventions that are home based and that include parents or families may be more effective in preventing childhood obesity than interventions in other community settings (Flynn et al, 2022).

## **Excess weight and obesity in young children in the UK**

- 8.12 All 4 UK countries (England, Scotland, Wales and Northern Ireland) also carry out regular surveillance and monitoring of the prevalence of overweight and obesity in preschool or school children.

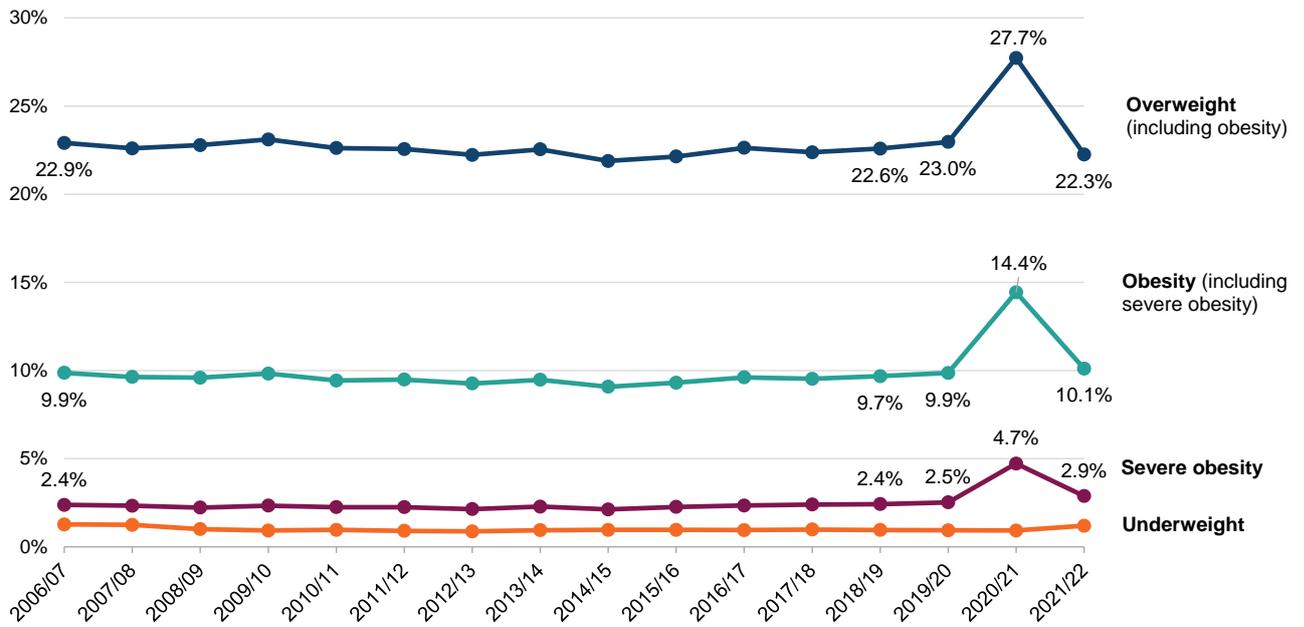
### **England**

- 8.13 In England, the National Child Measurement Programme (NCMP) measures the height and weight of children in reception year (aged 4 to 5 years) and year 6 (aged 10 to 11 years) to assess prevalence of overweight and obesity in children attending primary school. BMI values (derived from height and weight data) are compared against the UK90 reference population data to calculate age- and sex-adjusted centiles. The latest available NCMP data on child weight status are presented in Figure 8.1 to Figure 8.11 and in Annex 13 (Tables A13.1 to A13.3).
- 8.14 For NCMP collection year 2006 to 2007 to the collection year 2019 to 2020, the combined prevalence of overweight and obesity (using public health definitions, see paragraph 8.6) in children aged 4 to 5 years was fairly stable, at 22.0% to 23.0% (see Figure 8.1) (OHID, 2022c). A temporary uptick to 27.7% was observed during the first year of the COVID-19 pandemic (NCMP collection year 2020 to 2021), before declining to 22.3% the following year.
- 8.15 The prevalence of obesity (including severe obesity) prior to the COVID-19 pandemic (NCMP collection year 2006 to 2007 to collection year 2019 to 2020) ranged from 9.1% to 9.9% (NHS Digital, 2021). This was followed by a sharp increase to 14.4% in year 2020 to 2021, before declining to 10.1% in year 2021 to 2022, with the prevalence being somewhat higher in boys (10.3%) than girls (9.9%) (see Figure 8.2) (NHS Digital, 2022).
- 8.16 The prevalence of severe obesity ranged from 2.1% to 2.5% prior to pandemic (NCMP collection year 2006 to 2007 to collection year 2019 to 2020) (NHS Digital,

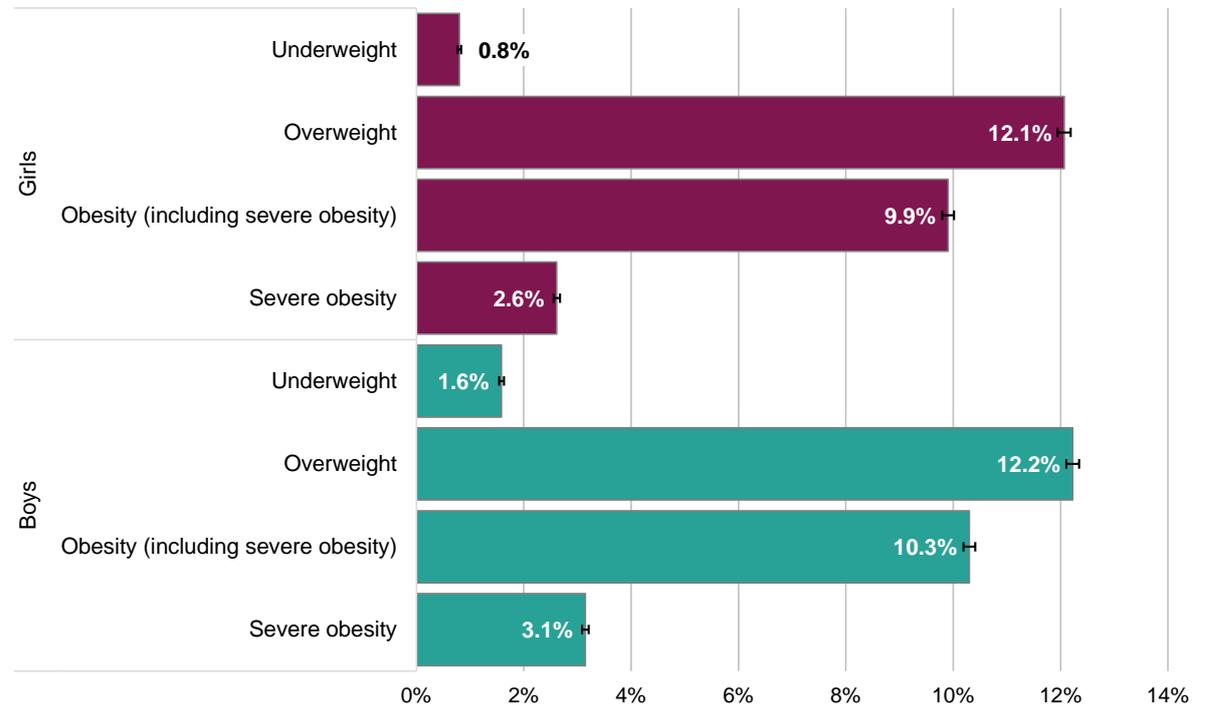
2021). This was followed by an increase to 4.7% in the collection year 2020 to 2021 and then a decline to 2.9% in the collection year 2021 to 2022, with the prevalence being higher in boys (3.1%) than girls (2.6%) (see Figure 8.2) (NHS digital, 2022).

8.17 The increase in prevalence of overweight and obesity during NCMP collection year 2020 to 2021 may have been partly due to a decrease in physical activity levels during the UK’s first national lockdown during the COVID-19 pandemic (Sport England, 2021a; Sport England, 2021b; Sport England, 2021c). Data are currently insufficient to determine whether total dietary energy intake (TDEI) also increased in young children during this time. However, NDNS data in older children (aged 2 to 10 years) indicated that TDEI was not significantly different from previous years (PHE, 2020c).

**Figure 8.1 Prevalence of overweight, obesity, severe obesity and underweight in children aged 4 to 5 years in England for NCMP collection year 2006 to 2007 to collection year 2021 to 2022 (OHID, 2022c)**



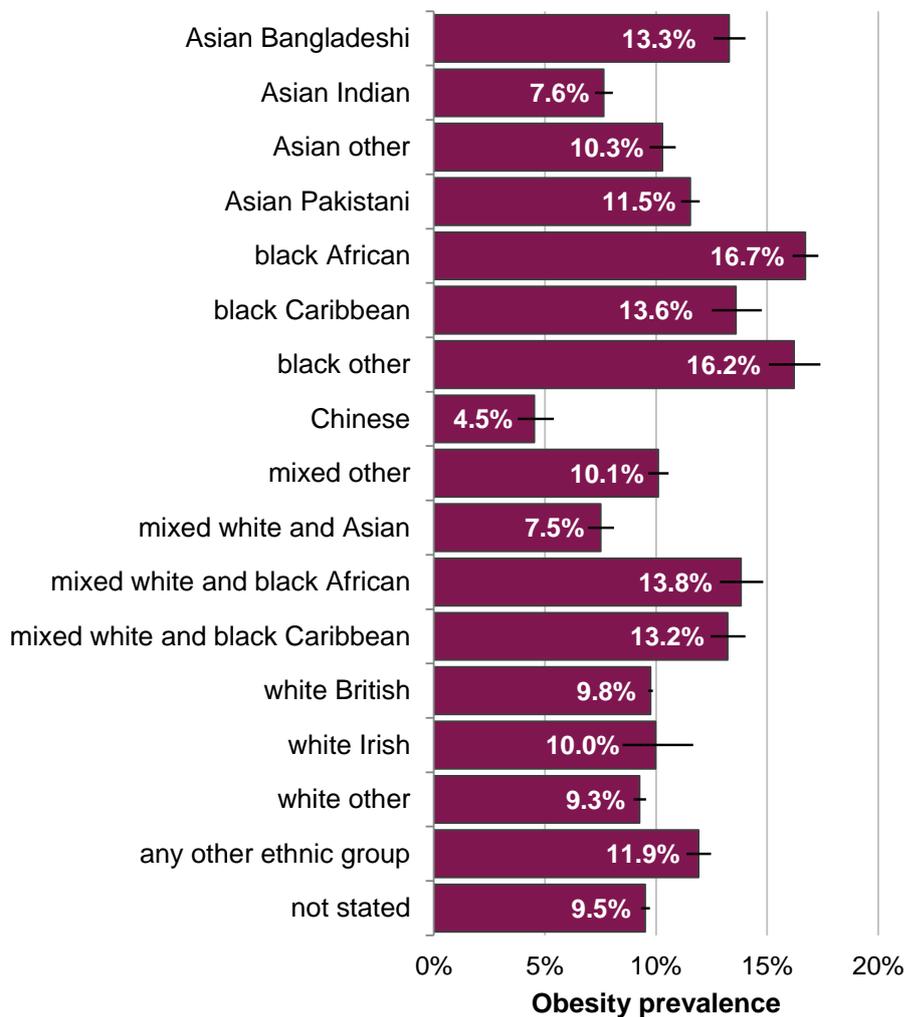
**Figure 8.2 Weight status prevalence of boys and girls aged 4 to 5 years in England for NCMP collection year 2021 to 2022 (NHS digital, 2022)**



## Weight status by ethnic group

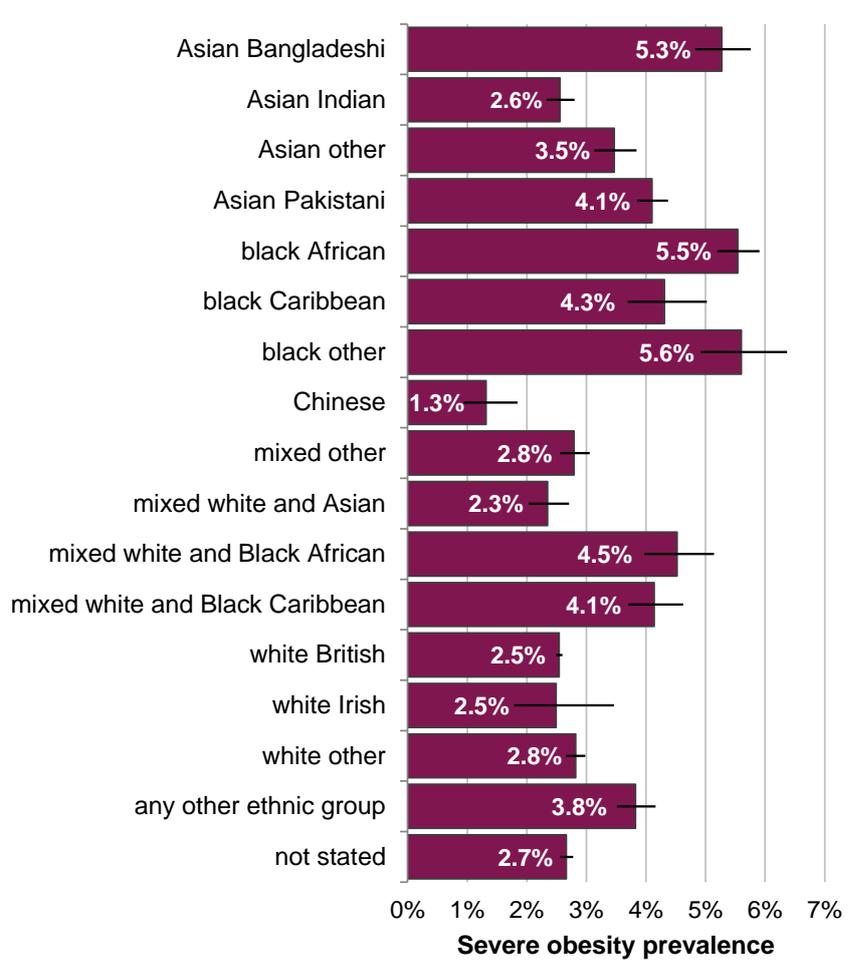
8.18 Obesity prevalence (including severe obesity) in children aged 4 to 5 years by ethnic group is presented in Figure 8.3 (OHID, 2022c). The prevalence of obesity was highest in children categorised as Black African (16.7%), Black other (16.2%), mixed White and Black African (13.8%), Black Caribbean (13.6%), Bangladeshi (13.3%) and mixed White and Black Caribbean (13.2%). Obesity prevalence was lowest in children categorised as Chinese (4.5%), mixed White and Asian (7.5%) and Indian (7.6%).

**Figure 8.3 Prevalence of obesity (including severely obese) in children aged 4 and 5 years by ethnic group for NCMP collection year 2021 to 2022 (OHID, 2022c).**



8.19 The prevalence of severe obesity was highest in children categorised as Black other (5.6%), Black African (5.5%) and Bangladeshi (5.3%) (see Figure 8.4) (OHID, 2022c). Prevalence was lowest in children categorised as Chinese (1.3%), mixed White and Asian (2.3%), White British or White Irish (both 2.5%) and Indian (2.6%).

**Figure 8.4 Prevalence of severe obesity in children aged 4 and 5 years by ethnic group for NCMP collection year 2021 to 2022 (OHID, 2022c)**



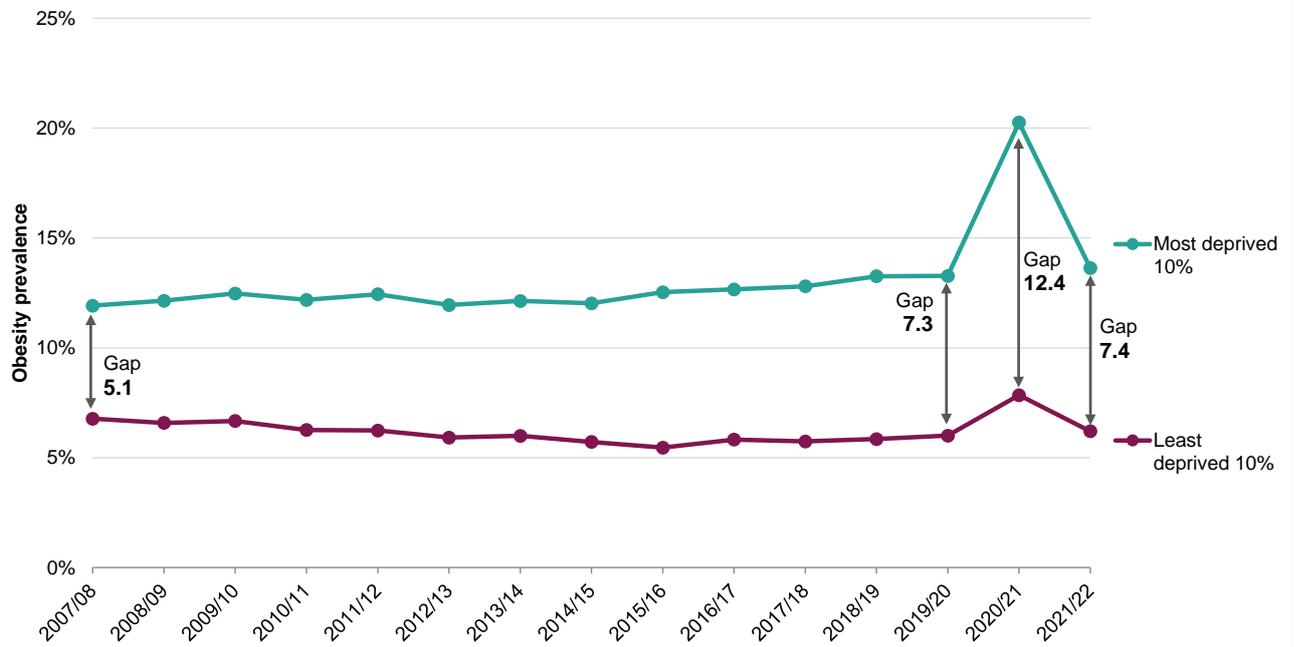
8.20 More detailed data on prevalence by weight status (from underweight to severe obesity) by ethnic group is available in Annex 13, Table A13.1.

### **Weight status and deprivation**

8.21 Weight status by Index of Multiple Deprivation (IMD) (see Glossary) in children aged 4 to 5 years is presented in Figure 8.5 to Figure 8.7, and in Annex 13, Tables A13.2 and A13.3.

8.22 Obesity prevalence in the 10% of children who lived in the least or most deprived areas for NCMP collection year 2007 to 2008 to collection year 2021 to 2022 is presented in Figure 8.5. For all years, obesity prevalence was substantially higher in children who lived in the most deprived areas. The gap in obesity prevalence between children who lived in the most and least deprived areas increased from 5.1% in collection year 2007 to 2008 to 7.4% in collection year 2021 to 2022, with a temporary widening of this gap to 12.4% during the first year of the COVID-19 pandemic (2020/21) (OHID, 2022c).

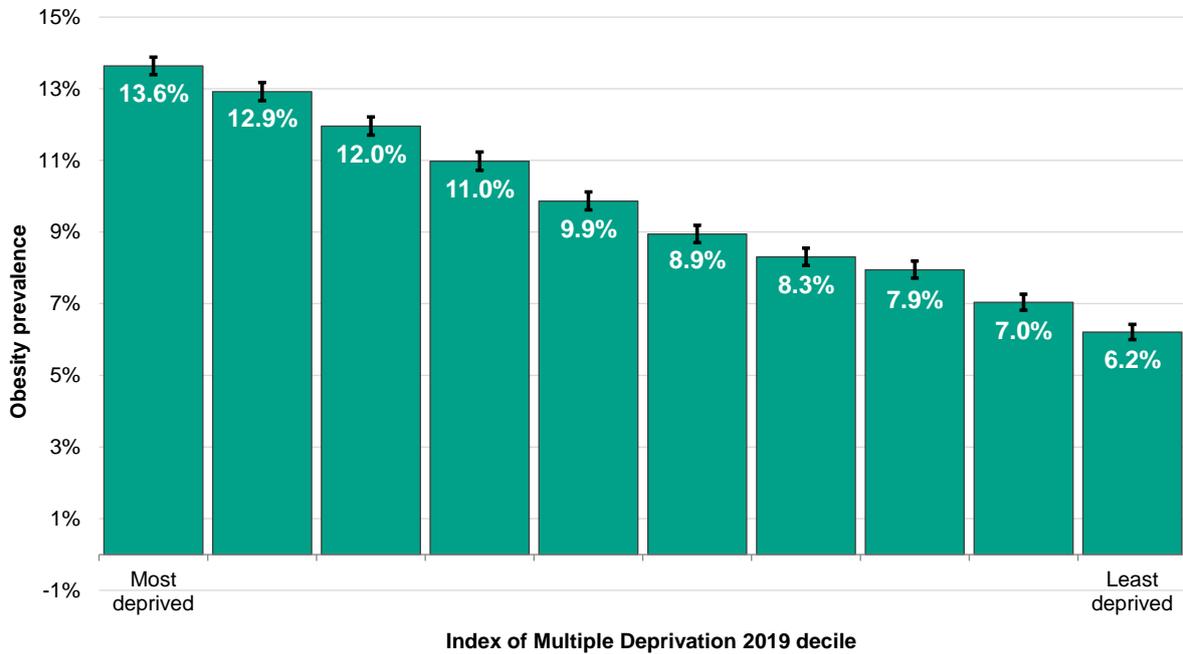
**Figure 8.5 Gap in the prevalence of obesity in children aged 4 to 5 years in England between children living in the least and most deprived areas based on Index of Multiple Deprivation (IMD) decile based on the postcode of the child, from collection year 2007 to 2008 to collection year 2021 to 2022 (OHID, 2022c)**



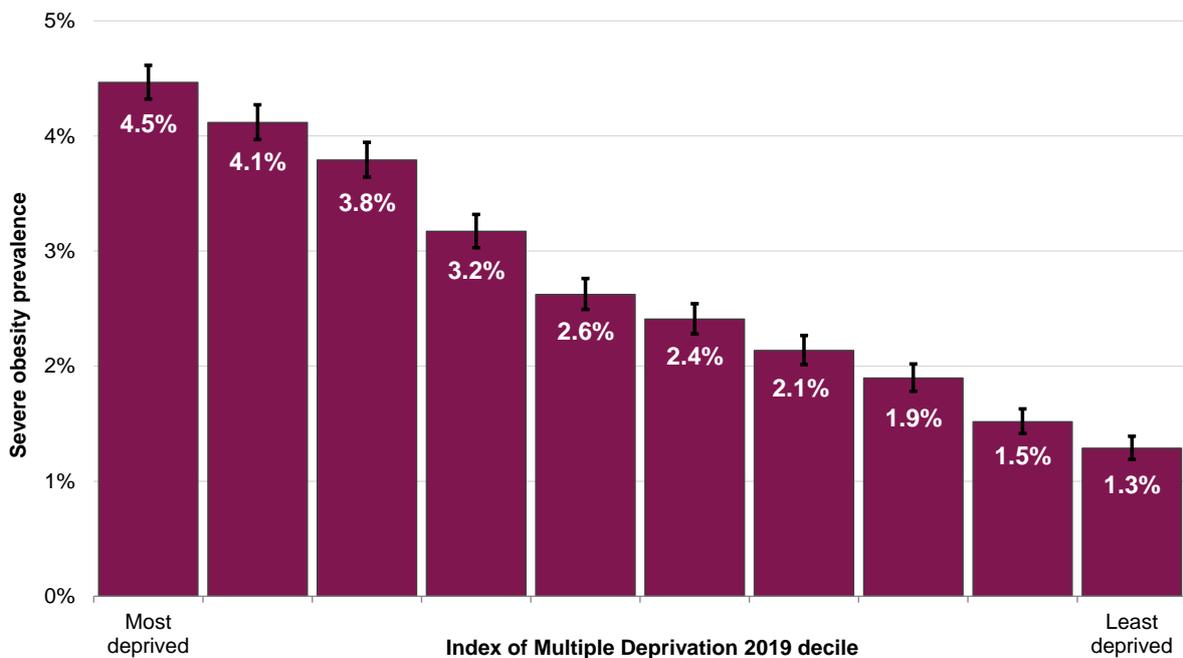
The data for 2021/22 indicated a strong relationship between the prevalence of obesity and deprivation (OHID, 2022c) (see Figure 8.6). Obesity prevalence (including severe obesity) increased with each IMD decile, from the least deprived (6.2%, in decile 10) to the most deprived decile (13.6%, in decile 1). For severe obesity (see

8.23 Figure 8.7), prevalence was over 3 times higher in children living in the most deprived areas (4.5%) than those in the least deprived areas (1.3%).

**Figure 8.6 Prevalence of obesity (including severe obesity) in children aged 4 to 5 years in England by Index of Multiple Deprivation (IMD) decile (based on the postcode of the child) (NCMP collection year 2021 to 2022) (OHID, 2022c)**



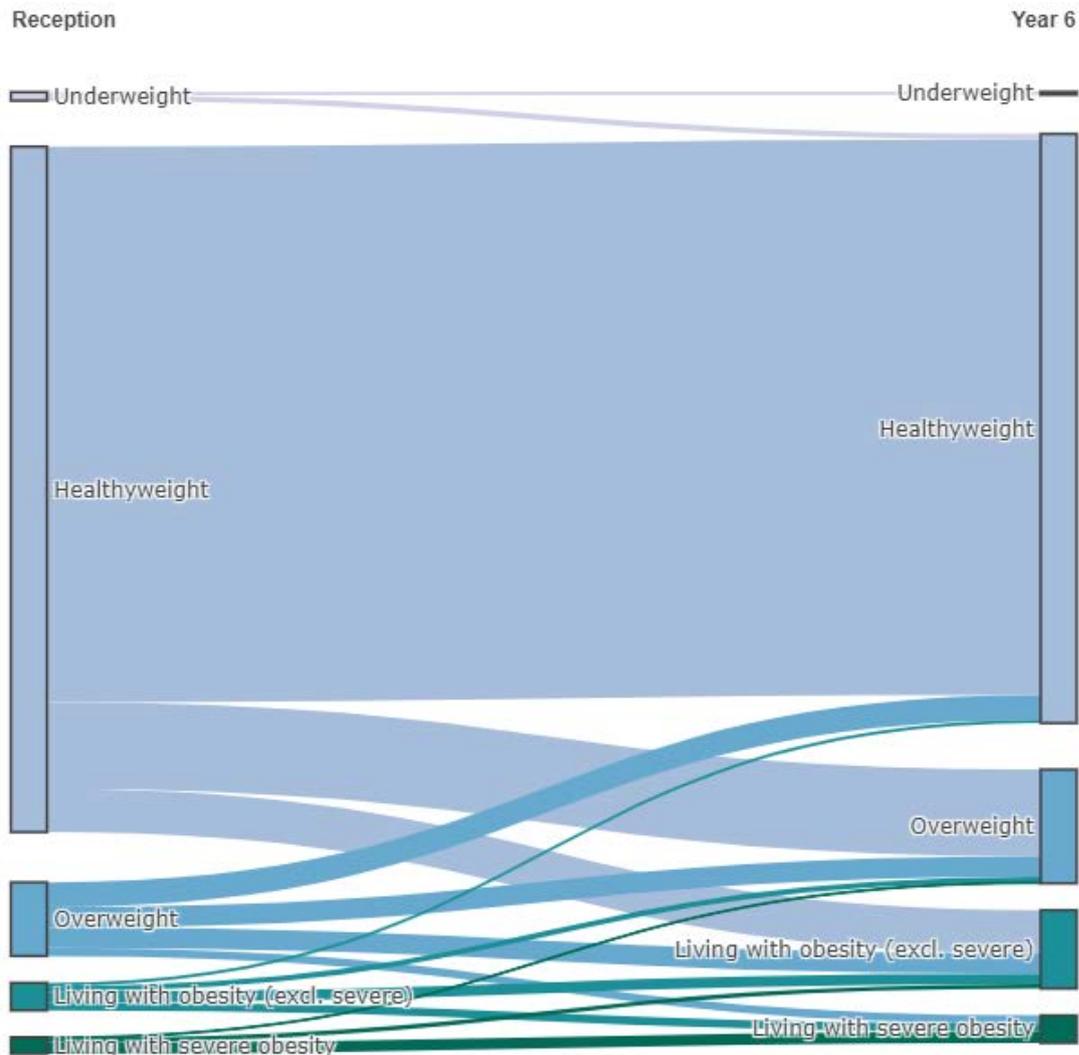
**Figure 8.7 Prevalence of severe obesity in children aged 4 to 5 years in England by Index of Multiple Deprivation (IMD) decile (based on the postcode of the child) (NCMP collection year 2021 to 2022) (OHID, 2022c)**



## Longitudinal trends in weight status

- 8.24 Changes in individual children's weight status between reception year (children aged 4 to 5 years in collection year 2013 to 2014) and year 6 (same children aged 10 to 11 years in collection year 2019 to 2022) of primary school are illustrated in Figure 8.8 below (OHID, 2022a).
- 8.25 In reception year, 84.7% of children were classified as having a healthy weight. Of these, 78.8% remained a healthy weight in year 6 while 19.7% had moved to a higher weight category (overweight or living with obesity or severe obesity) by year 6 (OHID, 2022a).
- 8.26 Of the children classified as living with overweight (excluding obesity) in reception year (8.9%), 66.3% remained in the overweight category or had moved to a higher weight category (living with obesity or severe obesity) by year 6, while 33.7% of children had moved to a healthy weight.
- 8.27 Of the children classified as living with overweight, obesity or severe obesity in reception year (14.2%), 75.9% remained in these higher weight categories in year 6, while 24.1% had moved to a healthy weight.
- 8.28 Of the children classified as living with obesity (excluding severe obesity) in reception year (3.3%), 68.6% remained in this weight category or had moved to living with severe obesity by year 6.
- 8.29 Of the children classified as living with severe obesity in reception year (2.1%), 65.6% remained living with severe obesity (65.5%) in year 6.
- 8.30 Taken together, the data indicate that BMI in young childhood is strongly predictive of BMI in later childhood.

**Figure 8.8 Changes in children’s weight status at age 4 to 5 years (reception) compared to age 10 to 11 years (year 6) (OHID, 2022a)**

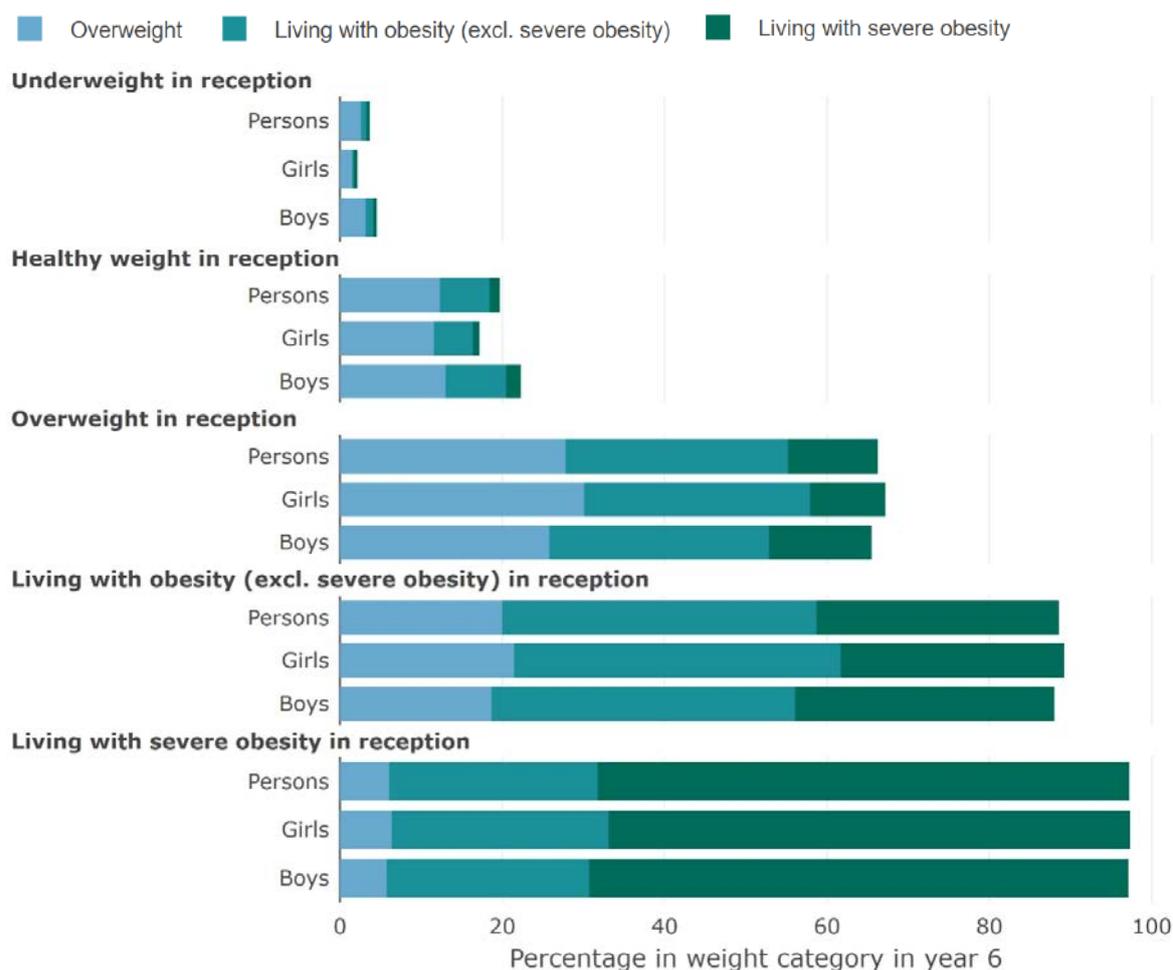


8.31 Changes in child weight status between reception year and year 6 by sex are illustrated in Figure 8.9. The figures show the percentage of children who either moved to or remained in the overweight, obesity or severe obesity categories when measured in year 6.

8.32 A larger proportion of girls (30.1%) than boys (25.8%) who were classified as living with overweight in reception year remained in the overweight category in year 6, while a larger proportion of boys (12.6%) than girls (9.3%) had moved to living with severe obesity by year 6 (OHID, 2022a).

8.33 A larger proportion of boys (31.9%) than girls (27.5%) who were classified as living with obesity (excluding severe obesity) in reception year had moved to living with severe obesity by year 6 (OHID, 2022a).

**Figure 8.9 Changes in children’s weight status at age 4 to 5 years (reception) compared to age 10 to 11 years (year 6), by sex (OHID, 2022a)**



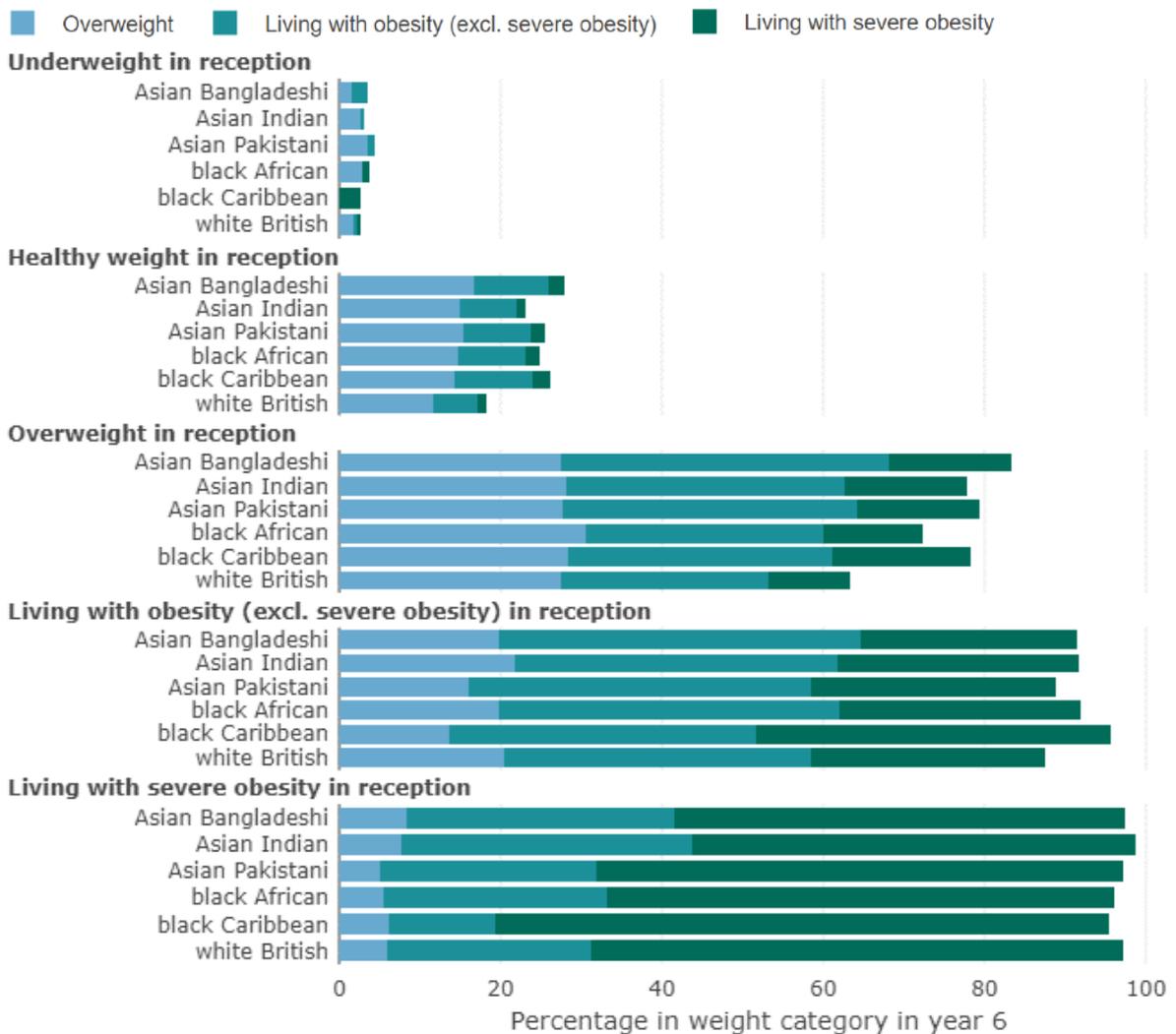
### Changes in weight status by ethnic group

- 8.34 Changes in children’s weight status between reception year (aged 4 to 5 years) and year 6 (aged 10 to 11 years) by ethnic group (Bangladeshi, Black African, Black Caribbean, Indian, Pakistani, or White British) are illustrated in Figure 8.10. The figures show the percentage of children who either moved to or remained in the overweight, obesity or severe obesity categories when measured in year 6.
- 8.35 Of the children classified as having a healthy weight in reception year, the proportion who had moved to living with overweight, obesity or severe obesity in year 6 ranged from 18.3% for White British children to 27.8% for Bangladeshi children (OHID, 2022a).
- 8.36 Of the children classified as living with overweight in reception year, a lower proportion of White British children moved to living with obesity (excluding severe obesity) (25.7%) and severe obesity (10.1%) in year 6, while higher proportions of Bangladeshi (40.6% and 15.2%), Black Caribbean (32.7% and 17.2%), Indian (34.3% and 15.3%) and Pakistani children (36.4% and 15.2%) moved to higher

weight categories (obesity [excluding severe obesity] and severe obesity, respectively) (OHID, 2022a).

8.37 Of the children classified as living with obesity (excluding severe obesity) in reception year, a higher proportion of Black Caribbean children moved to living with severe obesity (44.0%) compared with all other ethnic groups (which ranged from 26.9% for Bangladeshi children to 30.4% Pakistani children) (OHID, 2022a). The proportion of Black Caribbean children who remained living with severe obesity between reception year and year 6 (76.0%) was also markedly higher than that of all other ethnic groups (which ranged from 55.0% for Indian children to 66.0% for White British children).

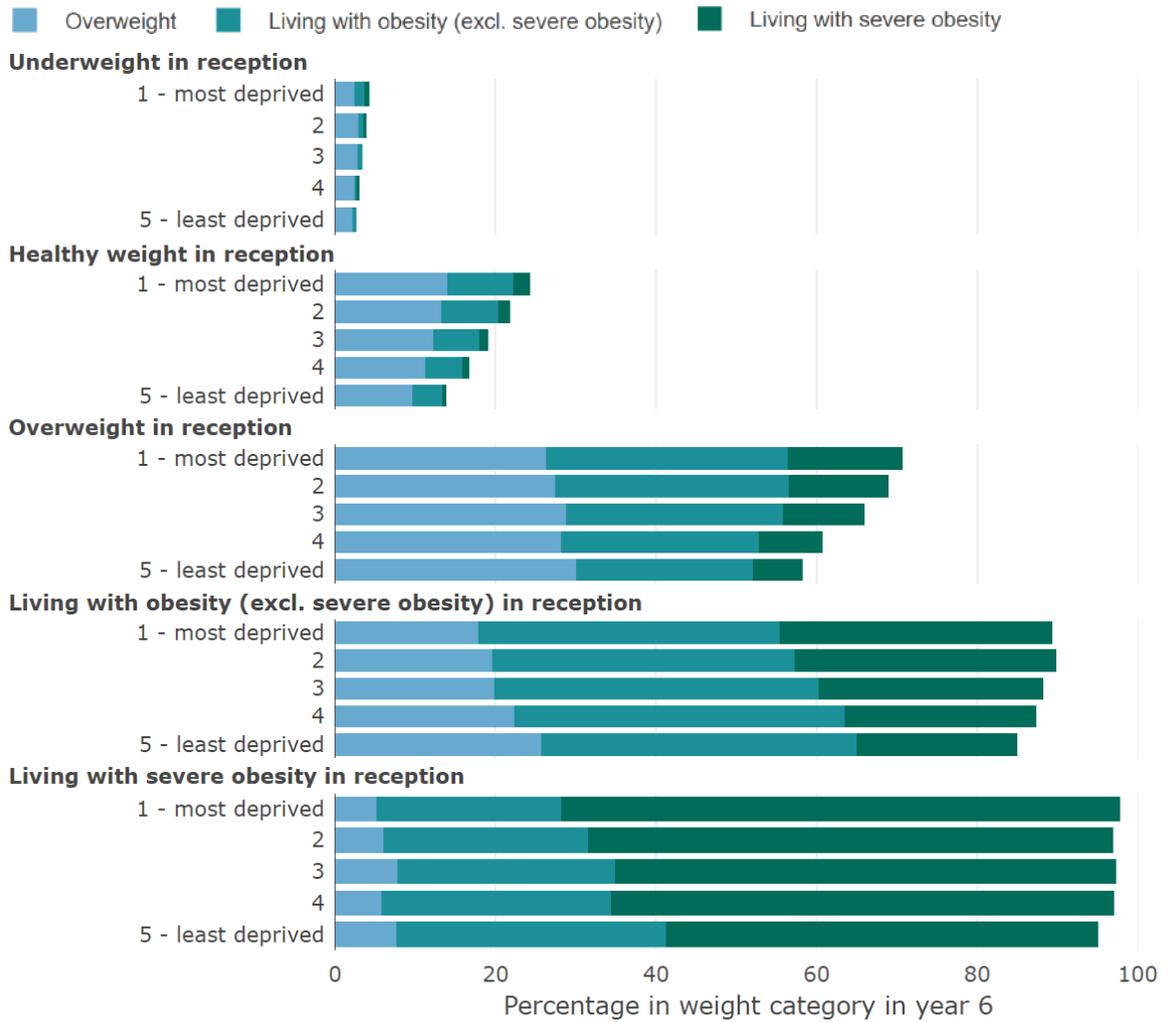
**Figure 8.10 Changes in children’s weight status at age 4 to 5 years (reception) compared to age 10 to 11 years (year 6), by ethnic group (OHID, 2022a)**



## Changes in weight status and deprivation

- 8.38 Changes in child weight status between reception year (aged 4 to 5 years) and year 6 (aged 10 to 11 years) by deprivation quintile (IMD by postcode of child), are illustrated in Figure 8.11 (OHID, 2022a). The figure shows the percentage of children who either moved to or remained in the overweight, obesity or severe obesity categories when measured in year 6.
- 8.39 Of the children with a healthy weight in reception year, a significantly higher proportion of children who lived in more deprived areas moved to living with obesity (excluding severe obesity) or severe obesity compared with children who lived in less deprived areas. For children who lived in the most deprived areas (quintile 1), over 2 times as many had moved to living with obesity (excluding severe obesity) (8.2%) and 4 times as many had moved to living with severe obesity (2.0%) in year 6 compared with children who lived in the least deprived areas (quintile 5) (3.7% and 0.5% respectively) (OHID, 2022a).
- 8.40 Of the children classified as living with obesity (excluding severe obesity) in reception year, a higher proportion of children who lived in the most deprived areas moved to living with severe obesity in year 6 (34.0% in quintile 1) compared with children who lived in the least deprived areas (20.0% in quintile 5). Meanwhile, a lower proportion of children who lived in the most deprived areas moved to living with overweight (17.8%) in year 6 compared with children who lived in the least deprived areas (25.7%).
- 8.41 Of the children classified as living with severe obesity in reception year, a higher proportion of children who lived in the most deprived areas remained in the severe obesity category in year 6 (69.6% in quintile 1) compared with children who lived in the least deprived areas (53.8% in quintile 5).

**Figure 8.11 Changes in children’s weight status at age 4 to 5 years (reception) compared to age 10 to 11 years (year 6), by deprivation quintile (by postcode of child) (OHID, 2022a)**



## Scotland

- 8.42 In Scotland, national statistics on weight status in children are captured annually through the [Scottish Health Survey \(SHeS\)](#) and the Scottish child health programme. The SHeS monitors the BMI of children aged 2 to 6 years (and ages 7 to 11 years and ages 12 to 15 years) while the Child Health Surveillance Programme School system (CHSP-S), which supports the delivery of the child health programme to school aged children, records height and weight measurements for Primary 1 school children (those aged around 5 years). National statistics on BMI in Primary 1 school children are published annually by Public Health Scotland.
- 8.43 Data collection for the most recently published SHeS (year 2021) was impacted by the COVID-19 pandemic (that is, the sample was smaller, and height and weight measures were parent-reported rather than collected by survey staff). In contrast, data collected for the CHSP-S was more recent, complete and employed the same methods as that used during the pre-pandemic years (Public Health Scotland, 2022b). For this reason, only statistics based on the data collected by the CHSP-S are reported below.
- 8.44 Data on BMI for Primary 1 school children (those aged around 5 years) for the collection year 2021 to 2022 indicated that 12.4% of children were at risk of overweight not including obesity (defined as a BMI at or above the 85<sup>th</sup> centile but less than the 95<sup>th</sup> centile) (Public Health Scotland, 2022b). This was similar to that observed in the period between collection years 2011 to 2012 and 2019 to 2020 (range 12.0% to 12.4%) but was lower than the value for collection year 2020 to 2021 (14.0%). The proportion of children at risk of obesity (defined as a BMI at or above the 95<sup>th</sup> centile) for collection year 2021 to 2022 was 11.7%. This was lower than the previous collection year (15.5%) but remained higher than that observed in the collection year 2019 to 2020 (10.4%). Overall, the BMI distribution of primary 1 children in collection year 2021 to 2022 appeared to be more similar to that from the pre-pandemic years than that observed in collection year 2020 to 2021. However, the proportion of children with a healthy weight (defined as a BMI below the 85<sup>th</sup> centile and above the 2<sup>nd</sup> centile) in collection year 2021 to 2022 (74.7%) was lower than that observed between the collection years 2011 to 2012 and 2019 and 2020 (range 76.1% to 77.5%). This was largely due to an increase in the proportion of children at risk of obesity.
- 8.45 Children living in the most deprived areas of Scotland (as indicated by the Scottish Index of Multiple Deprivation) were more than twice as likely to be at risk of obesity (15.5%) than children living in the least deprived areas (7.3%). They were also more likely to be at risk of overweight (13.5% in the most deprived areas compared with 10.3% in the least deprived areas) (Public Health Scotland, 2022b).

## Wales and Northern Ireland

- 8.46 The [Child Measurement Programme for Wales](#) measures the height and weight of children in Reception year (aged 4 to 5 years) and summarises their BMI values using public health thresholds for overweight and obesity (Public Health Wales, 2017). The most latest available statistics are from the collection year 2020 to 2021. However, due to the COVID-19 pandemic, data were not available for Wales overall. Sufficient data were available for only 2 Health Boards; these indicated that the proportion of children at risk of obesity (at or above the 95<sup>th</sup> centile) was approximately 18%, a significant increase of 5 to 6 percentage points from collection year 2018 to 2019 (Public Health Wales, 2021).
- 8.47 Statistics on BMI in children aged 2 to 15 years in Northern Ireland are published in the annual Health Survey Northern Ireland (NI). However, the Health Survey does not publish disaggregated statistics in children aged under 5 years.

# Systematic review evidence identified on excess weight and obesity and health

- 8.48 This section is divided into 2 parts. Consideration is first given to the evidence identified from systematic reviews (SRs) on child growth trajectory and its relationship with adult BMI or weight status. This is followed by an examination of the evidence identified from SRs on the relationship between BMI in children aged 1 to 5 years and adult health outcomes.

## Limitations of the systematic review evidence on excess weight and obesity

- 8.49 Much of the SR evidence identified on excess weight and obesity was informed by cohort studies that commenced in the mid- to late-20th century. As the obesity epidemic is a relatively recent phenomenon (since the 1990s), the environmental determinants of obesity are likely to have changed, potentially limiting the generalisability of findings to the present day.
- 8.50 There are well-known limitations regarding the use of BMI as a measure of overweight and obesity, including that it does not distinguish between lean and fat mass (see paragraph 8.2). Primary studies used widely differing cut-offs for overweight and obesity, which is not surprising given that there is no single accepted threshold for defining young child overweight and obesity (see paragraphs 8.6 to 8.7).
- 8.51 The reporting of outcomes varied between primary studies. Some reported outcomes on a continuous scale while others reported the proportion of children with overweight (variously defined) either combined with or separated from the proportion of children with obesity (variously defined).
- 8.52 Despite strong evidence of substantial disparities in child overweight and obesity based on differences in socioeconomic status (SES) and ethnicity (see [Excess weight and obesity in young children in the UK](#)), much of the SR evidence was derived from populations of mostly affluent, white children.
- 8.53 Primary studies rarely accounted for baseline BMI when examining the relationship between the age at adiposity rebound and later risk of obesity. Reverse causality, whereby there is uncertainty as to which factor is the exposure and which factor is the outcome, is highly possible in this area of research.
- 8.54 Prospective cohort studies (PCS) that reported a relationship between child BMI and adult BMI or weight status may have been able to describe the natural

development or history of becoming overweight and obesity. But these studies were not able to provide mechanistic insights to allow causal inferences to be made due to the great number of potential confounding factors that were often not measured or adjusted for (see paragraphs 8.52 and 8.53).

- 8.55 While there was potential for publication bias in this area of research, it was not assessed by the identified SRs.

## **Systematic review evidence identified on child growth trajectory and adult BMI or weight status**

- 8.56 'Child growth trajectory' describes the tracking of a child's growth from infancy and early childhood into later childhood and adulthood. The trajectory describes how a child may become overweight or obese and provides a way to connect early growth patterns to weight status in later life. It also allows investigation of common determinants of later weight status.
- 8.57 Potential confounding factors that should be considered when interpreting the evidence in this topic area include % body fat (BF), bottle-feeding status, in utero tobacco exposure, maternal weight status and gestational weight gain, parental BMI and SES. Potential variables that could modify any association between child growth trajectory and later weight status that should also be accounted for include standardised BMI (BMI SDS) at birth, gestational age, parity and season of birth.
- 8.58 One SR without meta-analysis (MA) was identified that examined the relationship between child growth trajectory and adult BMI or weight status (Brisbois et al, 2012). Details of the SR included in this section can be found in Annex 5 (Table A5.6). Quality assessment of the SRs using the AMSTAR 2 tool can be found in Annex 8 (Table A8.9). Additional data extracted on the primary studies can be found in Annex 9 (Table A9.38). The criteria used to grade the evidence are provided in chapter 2 (Table 2.4, paragraphs 2.54 to 2.59). Summary tables of the evidence grading process for this section are provided in Annex 10 (Tables A10.24 to A10.26, and Table A10.36).
- 8.59 Indicators of child growth trajectory covered by the SR were 'rapid early growth' (a phrase used in the SR), age at adiposity rebound (AR), and BMI or weight status of children aged 1 to 5 years. These indicators are considered in turn below.

### **Rapid early growth and adult BMI**

- 8.60 'Rapid early growth' describes the increase in body size, usually measured by BMI, beyond what would normally be expected at a particular stage of growth. There is a body of observational evidence indicating that rapid weight gain in

infancy (children under 12 months) may predict later obesity but also predicts tall stature (SACN, 2018).

- 8.61 In this report, consideration was given to rapid growth occurring beyond the age of 1 year.
- 8.62 One SR without MA (Brisbois et al, 2012) examined the relationship between rapid early growth and adult BMI.
- 8.63 Brisbois et al (2012) (AMSTAR 2 confidence rating: critically low) included 2 PCS which examined the relationship between rapid growth beyond the age of 1 year and adult BMI. Rapid early growth was defined in 1 PCS as the deviance from the average predicted growth rate (kg per year); and in the other PCS as an increase in the percentile rank across 2 major reference growth percentiles (defined by the US Centers of Disease Control and Prevention growth charts).
- 8.64 Both PCS (in a total of 940 participants) reported that rapid early growth between ages 1 and 7 years was associated with higher adult BMI, with 1 PCS reporting an association with higher BMI at ages 20 and 40 years (estimate of association not reported (NR);  $p < 0.001$ ). One PCS adjusted for birth weight, postnatal growth rate (percentile change) from birth to age 4 months and from age 4 months to age 1 year, maternal BMI and maternal weight gain during pregnancy. For the other PCS, all statistical models adjusted for adult age, child sex and gestational age, while a subset of models also adjusted for SES, parental weight and height and maternal smoking during pregnancy (it was unclear which findings from which model was cited in the SR).
- 8.65 It should be emphasised that while these findings may highlight the natural history of becoming overweight and obesity in adulthood, they do not provide mechanistic insights to allow causal inferences to be made.

### **Summary: rapid early growth and adult BMI**

- 8.66 The evidence identified from SRs on rapid early growth and adult BMI is summarised in Table 8.1.

**Table 8.1. Summary of the evidence on rapid early growth and adult body mass index (BMI)**

Exposure	Outcome	Direction of association	Certainty of evidence
Rapid early growth	Adult Body Mass Index (BMI)	Not applicable	Insufficient

- 8.67 The available evidence from SRs on the relationship between rapid early growth and adult BMI is from 1 SR without MA, given a critically low confidence rating using the AMSTAR 2 tool.

- 8.68 Although evidence from 2 PCS included in the SR by Brisbois et al (2012) suggested that rapid early growth at age 1 to 7 years was associated with higher adult BMI, the evidence from this SR was graded 'insufficient' given the small number of primary studies identified.

## **Age at adiposity rebound (AR) and adult BMI or risk of obesity**

- 8.69 Adiposity rebound (AR) describes the second rise in BMI that occurs in early childhood. An early AR may be a potential risk factor for obesity in later life. However, using age at AR as a determinant of later obesity risk has major limitations, as it can only be detected some years after it has occurred. It is therefore an unmodifiable risk factor and not useful when it comes to obesity prevention. It is also strongly associated with baseline BMI, as a higher BMI in early childhood results in a shallower, earlier rebound in BMI (Cole, 2004). Compared with AR, BMI in early childhood is a stronger predictor of BMI in later life as well as being measurable at a much earlier age (Freedman et al, 2022). Adjusting for baseline BMI is therefore critical in studies examining the relationship between age at AR and later BMI. Yet adjustment for baseline BMI has not been common practice. Given these limitations, age at AR is not considered a robust indicator of obesity risk in later life.
- 8.70 For this report, 1 SR without MA was identified that examined the relationship between age at AR and adult BMI or risk of adult obesity (Brisbois et al, 2012).
- 8.71 Brisbois et al (2012) (AMSTAR 2 confidence rating: critically low) included 4 PCS that examined this relationship in children who experienced AR at age 5 years or earlier. Three PCS (in a total of 948 participants) reported that early AR was associated with higher adult BMI and 1 PCS (in 458 participants) reported that early AR was associated with higher risk of obesity by age 26 years (relative risk [RR] 5.91; 95% CI 3.03 to 11.55; p-value NR), adjusted for sex. However, it was unclear whether any of the PCS adjusted for baseline BMI.

## **Summary: age at AR and BMI or risk of obesity**

- 8.72 The evidence identified from SRs on age at AR and obesity is summarised in Table 8.2.

**Table 8.2. Summary of the evidence on age at adiposity rebound and Body Mass Index (BMI) or risk of obesity**

Exposure	Outcome	Direction of association <sup>1</sup>	Certainty of evidence
Early adiposity rebound (occurring before age 5 years)	Adult Body Mass Index (BMI) or risk of obesity	↑	Limited

<sup>1</sup> Direction of association for reported outcomes: ↑increase

- 8.73 The available evidence from SRs examining the relationship between age at AR in children aged 1 to 5 years and adult BMI or risk of adult obesity is from 1 SR without MA, given a critically low confidence rating using the AMSTAR 2 tool.
- 8.74 Evidence from 4 PCS included in the SR by Brisbois et al (2012) suggests that AR occurring before age 5 years is associated with higher BMI or risk of obesity in adulthood. The evidence was graded ‘limited’ given the small number of studies identified and the lack of adjustment for baseline BMI.

### **Child BMI or weight status and adult BMI or weight status**

- 8.75 One SR without MA examined the relationship between child BMI or weight status and adult BMI or weight status (Brisbois et al, 2012). The SR included 11 PCS in children aged 1 to 5 years at baseline. Ten of the eleven PCS (in a total of 3590 participants) reported that a higher BMI (or a BMI above the 75<sup>th</sup> or 85<sup>th</sup> percentile) at ages 1 to 5 years was associated with higher adult BMI, while 1 PCS reported no association (quantitative findings NR). Of the 10 PCS that reported an association, 4 reported that a higher BMI (or a BMI above the 75<sup>th</sup> or 85<sup>th</sup> percentile) in children aged 1 to 5 years was associated with a higher risk of adult overweight or obesity, with estimates ranging from a RR of 1.8 to 2.72 (95% CI NR; p<0.05 reported for 1 PCS).
- 8.76 Two of the 10 PCS were in male only cohorts and one was in a female only cohort. In 1 PCS, there was an association in girls but not in boys. Of the 11 PCS, the SR reported quantitative findings for 5. Of these, 1 PCS adjusted for parental weight status and the other 4 were unadjusted.
- 8.77 It should be emphasised that while these findings may highlight the natural history of becoming overweight and obesity in adulthood, they do not provide mechanistic insights to allow causal inferences to be made.

### **Summary: child BMI or weight status and adult BMI or weight status**

- 8.78 The evidence identified from SRs on any relationship between child BMI or weight status and adult BMI or weight status is summarised in Table 8.3.

**Table 8.3. Summary of the evidence on the relationship between body mass index (BMI) or weight status in children aged 1 to 5 years and adult BMI or risk of adult overweight or obesity**

Exposure	Outcome	Direction of association <sup>1</sup>	Certainty of evidence
Child BMI or weight status	Adult Body Mass Index (BMI) or risk of adult overweight or obesity	↑	Adequate

<sup>1</sup> Direction of association for reported outcomes: ↑ increase

- 8.79 The available evidence from SRs examining the relationship between BMI or weight status in children aged 1 to 5 years and adult BMI or weight status is from 1 SR without MA given a critically low confidence rating using the AMSTAR 2 tool.
- 8.80 Evidence from 10 PCS included in the SR by Brisbois et al (2012) suggests that higher BMI or weight status in children aged 1 to 5 years is associated with higher adult BMI or risk of adult overweight or obesity. Due to the large number of studies (including several large PCS), and consistency in the direction of the results across the studies, the evidence was graded 'adequate'. However, as these studies do not provide mechanistic insights into the relationship between child BMI or weight status and adult BMI or weight status, the association can only be considered predictive rather than causal. In addition, as a MA was not conducted, it is not possible to estimate the strength of this association.

## **Systematic review evidence identified on child BMI and other health outcomes in adulthood**

- 8.81 One SR with MA (Llewellyn et al, 2016) was identified that examined the relationship between child BMI and type 2 diabetes (T2D), coronary heart disease (CHD), stroke and breast cancer in adulthood.
- 8.82 The SR did not state participant numbers included in its MAs. The SR also did not list key confounding factors but stated that, where possible, results from models adjusted for confounding were used in the MA. Models adjusted for adult BMI were not considered for inclusion in the MA because the focus of the SR was to examine the relationship between childhood obesity and morbidities without knowledge of later adult obesity.
- 8.83 All results from primary studies were converted into odds ratios (ORs) per standard deviation (SD) of BMI (with 95% CI) to allow calculation of pooled ORs for the MAs. This required some assumptions about the distributions of obesity in

the childhood population, such as that BMI follows a normal distribution. The SR acknowledged that this assumption may be invalid as the distribution of BMI may be positively skewed.

8.84 Limitations of evidence provided by Llewellyn et al (2016) included the following:

- many of the included PCS had low participant retention rates (<80%) by the final study measurement
- many of the cohorts commenced in the 1920s and 1950s. As social conditions for children have changed considerably since that time, it is unclear whether the evidence on any relationship between childhood BMI and adult morbidity from such cohorts accurately reflects present day conditions. On the other hand, some cohorts may not have had a sufficiently long follow up duration to fully capture adult morbidity-related events.

8.85 Details of the SR can be found in Annex 5 (Table A5.6). Quality assessment of the SRs using the AMSTAR 2 tool can be found in Annex 8 (Table A8.9). Additional data extracted on the primary studies can be found in Annex 9 (Table A9.39). The criteria used to grade the evidence are provided in chapter 2 (Table 2.4, paragraphs 2.54 to 2.59). Summary tables of the evidence grading process for this section are provided in Annex 10 (Tables A10.27 and A10.28, and Table A10.36).

## Child BMI and adult type 2 diabetes (T2D)

8.86 Llewellyn et al (2016) (AMSTAR 2 confidence rating: critically low) included 1 PCS (number of participants NR) that reported that child BMI at age 6 years and under was associated with incidence of T2D in adulthood (OR per SD of BMI 1.23; 95% CI 1.10 to 1.37).

## Summary: child BMI and adult T2D

8.87 The evidence identified from SRs on childhood BMI and T2D is summarised in Table 8.4.

**Table 8.4. Summary of the evidence on the relationship between child body mass index (BMI) and adult type 2 diabetes**

Exposure	Outcome	Direction of association	Certainty of evidence
Child Body Mass Index	Adult type 2 diabetes	Not applicable	Insufficient

8.88 The available evidence from SRs on the relationship between childhood BMI and adult T2D is from 1 SR with MA given a critically low confidence rating using the AMSTAR 2 tool. As the MA (Llewellyn et al, 2016) included only 1 PCS that examined this relationship in children aged 1 to 5 years, the evidence from this MA was graded 'insufficient'.

## Child BMI and adult coronary heart disease (CHD)

- 8.89 Llewellyn et al (2016) included a subgroup MA that reported no association between child BMI at age 6 years and under with incidence of CHD in adulthood (OR per SD of BMI 0.97; 95% CI 0.85 to 1.10;  $I^2=52%$ ; random-effects model; 3 PCS, number of participants NR). However, it is notable that an association was reported between higher child BMI at older ages (age 7 to 11 years and 12 to 18 years) and incidence of CHD.

### Summary: child BMI and adult CHD

- 8.90 The evidence identified from SRs on any relationship between child BMI and adult CHD is summarised in Table 8.5.

**Table 8.5. Summary of the evidence on the relationship between child body mass index (BMI) and adult coronary heart disease**

Exposure	Outcome	Direction of association	Certainty of evidence
Child Body Mass Index	Adult coronary heart disease	No association	Moderate

- 8.91 The available evidence from SRs on the relationship between child BMI and adult CHD is from 1 SR with MA, given a critically low confidence rating using the AMSTAR 2 tool.
- 8.92 Evidence from the subgroup MA conducted by Llewellyn et al (2016) reported no association between child BMI at age 6 years and under and incidence of CHD in adulthood. It is unclear whether estimates included in the MA were adjusted for potential key confounding factors. The evidence was graded 'moderate' given the number of PCS included in the MA.

## Child BMI and adult stroke

- 8.93 Llewellyn et al (2016) included a subgroup MA that reported no association between BMI in children aged 6 years and under and incidence of stroke in adulthood (OR per SD of BMI 0.94; 95% CI 0.75 to 1.19;  $I^2=58%$ ; random-effects model; number of participants NR). However, it is notable that an association was reported between higher child BMI in older age groups that were examined (age 7 to 11 years and 12 to 18 years) and incidence of stroke.

### Summary: child BMI and adult stroke

- 8.94 The evidence identified from SRs on any relationship between child BMI and adult stroke is summarised in Table 8.6.

**Table 8.6. Summary of the evidence on the relationship between child body mass index (BMI) and adult stroke**

Exposure	Outcome	Direction of association	Certainty of evidence
Child Body Mass Index	Adult stroke	No association	Moderate

- 8.95 The available evidence from SRs on the relationship between child BMI and adult stroke is from 1 SR with MA, given a critically low confidence rating using the AMSTAR 2 tool.
- 8.96 Evidence from the MA conducted by Llewellyn et al (2016) suggests that there is no association between BMI at age 6 years and under and adult stroke. It is unclear whether estimates included in the MA were adjusted for potential confounding factors. The evidence was graded 'moderate' and not downgraded due to medium statistical heterogeneity.

### **Child BMI and adult breast cancer**

- 8.97 Llewellyn et al (2016) included 1 PCS that examined the relationship between BMI in children aged 1 to 5 years and incidence of breast cancer in adulthood. The PCS reported no association between child BMI at age 6 years and under and incidence of breast cancer (OR per SD of BMI 0.88; 95% CI 0.67 to 1.16; number of participants NR). (There was also no association reported in older children).

### **Summary: child BMI and adult breast cancer**

- 8.98 The evidence identified from SRs on any relationship between child BMI and adult breast cancer is summarised in Table 8.7.

**Table 8.7. Summary of the evidence on the relationship between child body mass index (BMI) and adult breast cancer**

Exposure	Outcome	Direction of association	Certainty of evidence
Child Body Mass Index (BMI)	Adult breast cancer	Not applicable	Insufficient

- 8.99 The available evidence from SRs on the relationship between child BMI and adult breast cancer is from 1 SR with MA, given a critically low confidence rating using the AMSTAR 2 tool. As the MA (Llewellyn et al, 2016) included only 1 PCS that examined this relationship in children aged 1 to 5 years, the evidence from this MA was graded 'insufficient'.

# Summary of the systematic review evidence identified on excess weight and obesity

- 8.100 This section draws together the evidence relating to excess weight and obesity from throughout this report, including the current chapter.
- 8.101 Overall, there was a paucity of SR evidence on the majority of dietary exposures and their relationship with excess weight or obesity in children aged 1 to 5 years. Much of the evidence identified from SRs was graded 'insufficient' (see Annex 10, Table 10.36).
- 8.102 Table 8.8 lists the exposure-outcome relationships for which SR evidence was graded 'adequate', 'moderate' or 'limited'.
- 8.103 The strongest evidence identified relates to the health impact of consuming sugar-sweetened beverages (SSBs). There was 'adequate' evidence that higher consumption of SSBs in children aged 1 to 5 years is associated with a greater odds of overweight or obesity in childhood, and 'moderate' evidence that higher SSB consumption in children aged 1 to 5 years is associated with a greater increase in BMI (or BMI z-score or weight-for-height z-score) in childhood and adolescence, compared with lower SSB consumption. These findings strengthen those reported in the SACN report 'Carbohydrates and Health' that consumption of SSBs, compared with non-calorically sweetened beverages, results in greater weight gain and increases in BMI in children aged 5 years and older (SACN, 2015).
- 8.104 There was 'moderate' evidence that higher total protein intake in children aged 1 to 5 years is associated with higher BMI in childhood. This finding supports the conclusion from the SACN report ['Feeding in the First Year of Life'](#) that higher protein intake during infancy (for example, through infant formula feeding) promotes rapid weight gain and later risk of obesity (SACN, 2018).
- 8.105 There was 'adequate' evidence that higher child BMI or weight status at ages 1 to 5 years is associated with higher adult BMI or risk of overweight or obesity. This is a concern given the high prevalence of overweight and obesity in young children in the UK, particularly in lower socioeconomic groups and in some ethnic groups (see [Excess weight and obesity in young children in the UK](#)).
- 8.106 This report also identified:
- 'moderate' evidence that larger portion sizes of snack or lunch foods (in grams or energy intake) in preschool settings are associated with higher food and energy intake in the short term (less than 6 months). However, no evidence

was identified on whether varying portion sizes directly impacts children's body weight

- 'moderate' evidence that there is no association between child BMI at age 6 years and under and incidence of coronary heart disease in adulthood
- 'moderate' evidence that there is no association between child BMI at age 6 years and under and incidence of stroke in adulthood.

**Table 8.8. Summary of the evidence identified with obesity related outcomes**

Chapter	Exposure (in children aged 1 to 5 years)	Outcome	Direction of effect or association	Certainty of evidence
Energy and macronutrients (chapter 3)	Larger portion sizes of snacks and meals provided in preschool settings	Food and energy intake (short term, less than 6 months)	↑	Moderate
Energy and macronutrients (chapter 3)	Total fat intake	Body Mass Index (BMI) or body weight (shorter term)	No association	Limited
Energy and macronutrients (chapter 3)	Higher total protein intake	BMI	↑	Moderate
Foods, dietary patterns and dietary components (chapter 5)	Consuming 'unhealthy' dietary patterns (defined in paragraph 5.140)	Body fat	↑	Limited
Drinks (chapter 6)	Higher fruit juice consumption	Change in BMI	↑ (non-TDEI adjusted) No association (TDEI-adjusted)	Limited
Drinks (chapter 6)	Higher sugar-sweetened beverage (SSB) consumption	Odds of overweight and obesity	↑	Adequate
Drinks (chapter 6)	Higher SSB consumption	Change in BMI	↑	Moderate
Excess weight and obesity (chapter 8)	Rapid early weight gain or growth	Adult BMI	↑	Limited
Excess weight and obesity (chapter 8)	Early adiposity rebound (occurring before age 5 years)	Adult BMI or risk of adult obesity	↑	Limited
Excess weight and obesity (chapter 8)	Higher child BMI or weight status	Adult BMI or risk of overweight or obesity	↑	Adequate
Excess weight and obesity (chapter 8)	Child BMI	Incident adult coronary heart disease	No association	Moderate
Excess weight and obesity (chapter 8)	Child BMI	Incident adult stroke	No association	Moderate

<sup>1</sup> Direction of effect or association for reported outcomes: ↑increase; ↓decrease or inverse

# 9 Oral Health

## Background

### Oral health of children in the UK

- 9.1 Oral health is integral to good general health and well-being. Despite this, it is estimated that oral diseases affect 3.5 billion people worldwide, with untreated dental caries being among the most prevalent noncommunicable diseases (Institute for Health Metrics and evaluation, 2018).
- 9.2 Dietary sugars are the direct cause of dental caries, a biofilm-mediated disease that results in the phased demineralisation and remineralisation of dental hard tissues (Pitts et al, 2017; Sheiham & James, 2015). Destruction of susceptible dental hard tissues is caused by acidic by-products from the bacterial fermentation of dietary sugars by oral bacteria (Marsh & Martin, 1999). These acidic products (mainly lactic acid) cause a drop in pH levels which makes the tooth susceptible to demineralisation. Grooves called pits and fissures, particularly on the biting surfaces of teeth, easily collect dental biofilm and are the first sites to be affected (Pitts et al, 2017). In the early stages of the process, this demineralisation is reversible, and the early carious lesion can remineralise. Fluoride acts as a catalyst to this remineralisation process (ten Cate, 2013). It is important to note that this is a very different process to the condition of dental erosion which does not require sugars or bacteria to be present but is caused by a range of acids which thin the surface enamel; they may be external (for example, acidic food and drinks) or intrinsic acids (for example, gastric reflux) (DHSC, 2021b).
- 9.3 Tooth decay in early childhood is known as early childhood caries (ECC) and is defined as “the presence of one or more decayed (non-cavitated or cavitated lesions), missing (due to caries), or filled tooth surfaces in any primary tooth” in a child under the age of 6 years (AADP, 2021). Severe ECC (S-ECC) is defined as “1) any sign of smooth-surface caries in a child younger than 3 years of age, 2) from ages 3 to 5, one or more cavitated, missing (due to caries), or filled smooth surfaces in primary maxillary anterior teeth, or 3) a decayed, missing, or filled score of greater than or equal to 4 (age 3 years), greater than or equal to 5 (age 4 years), or greater than or equal to 6 (age 5 years)” (AADP, 2021).
- 9.4 Dental caries in primary teeth is a risk indicator for caries in the permanent dentition. Longitudinal studies have shown that children who have developed dental caries in their primary teeth (by age 7 to 9 years) go on to have high levels of disease in their permanent dentition (Broadbent et al, 2008; Hall-Scullin et al, 2017; Li & Wang, 2002; Skeie et al, 2006). Dental caries is a cumulative

progressive disease that impacts across the life course and increases the risk of tooth loss (Elderton, 2003).

## **Prevalence of oral health problems in children aged 1 to 5 years in the UK**

- 9.5 There have been substantial reductions in dental caries levels since the 1970s but despite being largely preventable, dental caries in children remains a major public health problem. National surveys have shown the scale of the problem. In 2013, 40% of children aged 5 years in Northern Ireland had obvious tooth decay (HSCIC, 2015), 34.2% in Wales in 2015 to 2016 (Cardiff University, 2017), 26.5% in Scotland in 2020 (Public Health Scotland, 2020), and 23.4% in England in 2019 (PHE, 2020b). For those children at risk, tooth decay starts early. In 2020, a survey of children aged 3 years in England found that 10.7% had visible tooth decay, with an average 3 teeth affected (PHE, 2021c). Almost 9 out of 10 hospital tooth extractions among children aged 0 to 5 years are due to preventable tooth decay and tooth extraction is still the most common hospital procedure in children aged 6 to 10 years (PHE, 2020b; PHE, 2021b). Just under 50,000 children aged 0 to 19 years were admitted to hospital to have teeth removed under general anaesthesia in 2019 to 2020 (PHE, 2021b). This pattern is similar or worse in Scotland, Wales and Northern Ireland (SACN, 2018).
- 9.6 Children from poor and disadvantaged backgrounds experience much higher levels of dental caries than their more advantaged peers (Watt et al, 2015) and are more severely affected (Holmes et al, 2015; Pitts et al, 2015). For example, in 2020, the prevalence among the most deprived children at age 5 years in England was 34% compared with 14% for the least deprived (PHE, 2021c), with 38% of the variation in the prevalence of tooth decay explained by deprivation.

## **Impact of oral health problems on children and families**

- 9.7 Dental caries has a significant impact on the quality of life of children and families. For children, this can result in pain, infection, difficulties with eating contributing to risk of undernutrition (Tanner et al, 2022), sleeping, speaking, socialising and absence from school (Heilmann et al, 2015; Nuttall et al, 2006; OHID, 2022b).

## UK guidance for oral health improvement

- 9.8 Existing UK government guidance on dental caries prevention in young children (DHSC, 2021a) include the following recommendations on feeding practices, dietary intake and oral hygiene:
- infants should be introduced to drinking from a free-flow cup from the age of 6 months while feeding from a bottle should be discouraged from the age of 1 year
  - sugars should not be added to foods or drinks
  - minimise the amount and frequency of consumption of sugar-containing foods and drinks
  - avoid sugar-containing foods and drinks at bedtime when saliva flow is reduced and buffering capacity is lost
  - parents or carers should brush their children's teeth up to the age of 3 years, and brush or supervise tooth brushing from ages 3 to 6 years
  - start brushing as soon as the first tooth appears (usually at about 6 months of age), at least twice a day with fluoride toothpaste last thing at night and on at least one other occasion
  - see a dentist as soon as the first tooth appears and no later than the first birthday (BSPD, 2016)
  - use fluoridated toothpaste containing at least 1,000 ppm fluoride
  - use only a smear of fluoride toothpaste up to the age of 3 years, and from ages 3 to 6 years, a pea-sized amount of toothpaste.
- 9.9 For all guidance on maintaining good oral health, see [Delivering Better Oral Health](#) (DHSC, 2021a). The guidance seeks to ensure a consistent UK wide approach to prevention of oral diseases. In Scotland, the guidance is used to inform its oral health improvement policies.
- 9.10 Water fluoridation is one of a range of interventions available to improve oral health. Fluoride occurs naturally and can be present in water and some foods in varying concentrations. In some areas with low natural fluoride levels, fluoride is added to public drinking water (in line with safe limits) to improve dental health. In the UK, policy on water fluoridation varies by region and country. For example, around 1 in 10 people in England have fluoride added to their drinking water supplies (OHID, 2022d); while in Scotland, Wales and Northern Ireland, water supplies are currently not fluoridated. In England, the Office for Health Improvement and Disparities (OHID), on behalf of the Secretary of State for Health and Social Care, has a legal duty to monitor the effects of water fluoridation schemes on health and report on it every 4 years. The 2022 monitoring report found that children aged 5 years living in areas in England with higher fluoride

concentrations were less likely to experience dental caries, and less likely to experience severe dental caries, than in areas with low fluoride concentrations (OHID, 2022d).

## Breastfeeding and bottle feeding and oral health

- 9.11 The World Health Organization (WHO) recommends that breastfeeding continues for up to 2 years of age or beyond; while in the UK, continued breastfeeding is recommended for at least the first year of life. In its report [‘Feeding in the first year of life’](#), SACN concluded that breastfeeding up to 12 months of age is associated with a decreased risk of dental caries and may offer some protection when compared with feeding infant formula (SACN, 2018).
- 9.12 Human milk, cows’ milk, infant formula and formula milks marketed specifically for children aged 12 months and older (see Glossary) all contain sugars. Cows’ milk contains approximately 4% lactose, which may be the least cariogenic of the sugars, while containing high levels of calcium, phosphate and proteins that have a protective effect against dental caries (Grenby et al, 2001; WHO, 2003; WHO, 2007a). Human milk and infant formula contain approximately 7% sugars, primarily lactose, but significantly lower levels of calcium and phosphate compared with cows’ milk (PHE, 2021a). Lactose-free formula and soya-based infant formula often contain free sugars such as glucose, as well as maltodextrins, which are hydrolysed by salivary amylase into free sugars, as a replacement for lactose (FSNT, 2021; NHS, 2022). Formula milks which are marketed specifically for children aged 1 year and older (‘growing up’ and ‘toddler’ milks) also contain free sugars (see [Types of formula milks](#) for details). Therefore, it is possible that exposure to breast milk and formula milks both carry risks of dental caries. However, data show that breastfeeding up to age 12 months may be protective against dental caries compared with formula feeding (SACN, 2018).
- 9.13 There are also few data on the impact of infant feeding mode and duration on the maturation and dysbiosis of the oral microbiota in infants and children, and subsequent development of ECC. Preliminary data show that breastfeeding strongly influences the development of the oral microbiome (Dzidic et al, 2018). It is also difficult to separate out the effects of various factors that could influence ECC risk: the mode and frequency of feeding; the effects of sugars from complementary feeding and factors related to socioeconomic status (SES).
- 9.14 Factors that have been explored include the sugars content of breast milk or infant formula, although in the case of the latter, much of the experimental research has been conducted in adults (Tan et al, 2016). Investigations have also sought to determine the impact of length of contact with breast milk or infant formula on the erupted dentition (that is, the frequency of feeding and feeding practices which

result in pooling of breast milk or infant formula around the surfaces of the teeth), and the influence of age of colonisation and levels of cariogenic bacteria (for example, *Streptococcus mutans*) in a child's mouth. The growth and adhesion of cariogenic bacteria, particularly oral Streptococci, are inhibited by breast-specific Lactobacilli and substances including human casein and secretory IgA in breast milk, which are not found in infant formula (Danielsson Niemi et al, 2009; Holgerson et al, 2013). The risk of dental caries also rises with increasing number of teeth as the primary teeth erupt over time up until around 2 years.

## **Systematic review evidence identified on oral health**

- 9.15 Eight systematic reviews (SRs) were identified that examined the relationship between feeding practices, food and drink consumption, and oral health in children (Baghlaf et al, 2018; Dror & Allen, 2014; Hermont et al, 2015; Hooley et al, 2012a; Hooley et al, 2012b; Moynihan & Kelly, 2014; Tham et al, 2015; Thomaz et al, 2018).
- 9.16 An additional 3 SRs were identified for consideration after the public consultation on the draft report. Of these, 2 SRs (Moores et al, 2022; Moynihan et al, 2019) are described in the main report because they provide evidence that added to the evidence base. Details of Cascaes et al (2022) can be found in Annex 6 (Table A6.3) and Annex 10 (Table A10.34).
- 9.17 Key exposures were (presented in order of certainty of evidence):
- (free) sugars intake
  - sugar-sweetened beverage consumption
  - breastfeeding beyond 12 months
  - use of infant feeding bottles for milk feeds beyond 12 months
  - night time use of infant feeding bottles for milk feeds
  - use of infant feeding bottles to consume liquids containing free sugars
  - consumption of foods containing free sugars
  - consumption of milk and dairy products
  - baseline body weight.
- 9.18 Key outcomes were the development of ECC, severe-ECC and malocclusion.
- 9.19 Details of the 10 SRs can be found in Annex 5 (Table A5.7) and Annex 6 (Table A6.3). Quality assessment of the SRs using the AMSTAR 2 tool can be found in Annex 8 (Table A8.10). Additional data extracted on the primary studies can be

found in Annex 9 (Table A9.40 to A9.50). The criteria used to grade the evidence are provided in chapter 2 (Table 2.4, paragraphs 2.54 to 2.59). Summary tables of the evidence grading process for this section are provided in Annex 10 (Tables A10.29 to A10.36).

## **Limitations of the systematic review evidence identified on oral health**

- 9.20 None of the primary studies included in SRs that examined the potential impact of breastfeeding on development of ECC directly compared breastfeeding beyond 12 months with either children fed cows' milk or formula milks beyond 12 months.
- 9.21 Most of the evidence from SRs that examined the relationship between breastfeeding or use of infant bottles for feeding and development of ECC was derived from studies conducted in upper middle income countries (UMICs). This may limit the generalisability of the findings to children living in the UK.
- 9.22 Primary studies included in the SRs varied considerably in their exposures (for example, different measures foods containing free sugars consumed at different times of day) and outcome measures (for example, caries incidence or prevalence, caries increment, early and severe-ECC measured using different indices), making comparisons between studies difficult.
- 9.23 Primary studies included in the SRs seldom adequately measured or accounted for potential modifying and confounding factors. Potential confounding factors include the consumption of free sugars from foods and drinks (especially in breast- and bottle feeding studies), night time feeding, and household socioeconomic status (SES). Important modifying factors include poor oral hygiene practices (for example, the infrequent or delayed introduction of tooth brushing and not using fluoride-containing toothpaste) and exposure to fluoride in water (Ha et al, 2019).
- 9.24 These confounding and modifying factors may also be associated with one another. For example, parents or carers who adopt good oral hygiene practices may be less likely to offer their children cariogenic foods and drinks; both these factors may in turn be associated with household SES.
- 9.25 SRs seldom reported sources of funding of included studies. Caution should be applied when interpreting findings particularly from studies funded by companies that sell or promote the use of formula milks.

## Free sugars intake and development of dental caries

- 9.26 SACN performed a SR on sugars intake and oral health, which was published as an annex to its report [‘Carbohydrates and health’](#) (SACN, 2015). A summary of the findings from the SR that are relevant to children aged 1 to 5 years is presented below.
- 9.27 Two PCS reported frequency of sugar consumption in relation to development of dental caries in primary dentition but provided little evidence of an association. It was unclear what was precisely meant by the exposure term ‘sugar’, as further details were not reported.
- 9.28 The PCS mostly reported that higher frequencies of consumption of sugar-containing drinks (including non-carbonated fruit drinks and fruit juice) increased development of caries in primary dentition (5 out of 6 studies).
- 9.29 Reported associations between frequent consumption of sweets (including confectionery and candy) and development of dental caries in primary dentition were less consistent. Half the studies (2 out of 4) reported an association between higher frequency of consumption and increased development of dental caries while the other half reported no relationship. The exposure term ‘sweets’ (including confectionery and candy) was unclear, as details were not reported.
- 9.30 For this report, 3 SRs without MAs (Baghlaf et al, 2018; Hooley et al, 2012b; Moores et al, 2022) and 1 SR with MA (Moynihan & Kelly, 2014) were identified that included studies examining the relationship between free sugars intake in children aged 1 to 5 years and the development of dental caries. It should be noted that Moores et al (2022) is an update of Moynihan & Kelly (2014) and includes evidence identified between 2011 and 2020. It should also be noted that although Moynihan & Kelly (2014) performed MAs, findings from PCS in children aged 1 to 5 years were not pooled into a single MA. Therefore, for this report, study findings from this SR were considered individually.
- 9.31 Moynihan & Kelly (2014) (AMSTAR 2 confidence rating: high) included 4 PCS that examined the relationship between free sugars intake in children aged 1 to 5 years and development of dental caries. Moores et al (2022)(AMSTAR 2 confidence rating: high) included a follow-up study (in adolescents) of 1 PCS that was included in Moynihan & Kelly (2014), as well as an additional PCS. Hooley et al (2012b) (AMSTAR 2 confidence rating: critically low) also included an additional PCS.
- 9.32 The 6 PCS included in the 3 SRs were conducted in children aged 1 to 4 years at baseline and followed up for 1 to 4 years (in 5 PCS) and 16 years (in 1 PCS). Exposures were intakes of sucrose, free sugars or added sugars. Outcome

measures were a measure of caries increment over time (mostly using the WHO diagnostic criteria) or caries incidence or prevalence.

- 9.33 Of the 6 PCS, 5 PCS (in a total of 2938 participants in HIC and UMIC) reported an association between higher free sugars intake at ages 1 to 4 years and increased development of dental caries in childhood and adolescence compared with lower free sugars intake. Four of the five PCS adjusted for SES or oral hygiene practices (tooth brushing or use of a fluoride agent).
- 9.34 The sixth PCS reported no association (unadjusted).
- 9.35 Of the 5 PCS that reported an association, 1 PCS (in 2181 participants, in HIC) reported that restricting intake of free sugars to less than 5% total dietary energy intake (TDEI) protected against dental caries. There was a higher prevalence of ECC at ages 2 to 3 years in children who consumed more than 10% TDEI as free sugars at ages 1 to 2 years compared with children who restricted their intake of free sugars to less than 5% TDEI at the same age (Prevalence Ratio [PR] 1.97; 95% CI 1.13 to 3.34; p-value not reported). The analysis was adjusted for maternal education and SES.
- 9.36 Another PCS (in 510 participants, in UMIC) reported that restricting intake of free sugars to less than 10% TDEI at age 3 years was associated with reduced odds of developing ECC 1 year later compared with children with intake of free sugars greater than 10% TDEI (Odds Ratio [OR] 2.99; 95% CI 1.82 to 4.91; p<0.001). The analysis was adjusted for SES and oral hygiene practices (tooth brushing and use of fluoride gel).

## Summary: free sugars intake and development of dental caries

- 9.37 The evidence identified from SRs on free sugars intake and development of dental caries is summarised in Table 9.1.

**Table 9.1. Summary of the evidence on sugars intake and development of dental caries**

Exposure	Outcome	Direction of association <sup>1</sup>	Certainty of evidence
Intake of free sugars	Dental caries (increment, incidence or prevalence)	↑	Adequate

<sup>1</sup> Direction of association for reported outcomes: ↑ increase

- 9.38 The available evidence from SRs on the relationship between free sugars intake and development of dental caries in children aged 1 to 5 years is from 3 SRs, 2

given a high confidence rating using the AMSTAR 2 tool, and 1 given a critically low confidence rating.

- 9.39 Evidence from 5 PCS included in Moores et al (2022), Moynihan & Kelly (2014) and Hooley et al (2012b) suggests that higher free sugars intake in children aged 1 to 5 years is associated with increased dental caries (increment, incidence or prevalence) in childhood and adolescence compared with lower free sugars intake. Of the 5 PCS, 1 PCS reported that restricting intake of free sugars to less than 5% TDEI was protective against development of dental caries.
- 9.40 The evidence from the 5 PCS was graded 'adequate' given the consistent findings across the PCS, including large effect sizes reported in some, and adequate accounting for key confounding factors in most PCS.
- 9.41 These findings strengthen those from the SACN report '[Carbohydrates and health](#)'. The evidence is also consistent with current UK recommendations that intake of free sugars should not exceed the population average of 5% TDEI.

## **Sugar-sweetened beverages and development of dental caries**

- 9.42 One SR with MA (Moynihan et al, 2019) was identified that examined the relationship between sugar-sweetened beverage (SSB) consumption in children aged 1 to 5 years and development of dental caries. For evidence relating SSB consumption and other health outcomes, see [Sugar-sweetened beverages](#) in chapter 6. It should be noted that studies included in the SR used the terminology 'sugar-sweetened beverage', 'sugar-containing liquids', and 'sugary drinks'. For consistency with the rest of this report, SSB is used to describe the evidence in this section.
- 9.43 Moynihan et al (2019) (AMSTAR 2 confidence rating: moderate) included 4 PCS on SSB consumption in children aged 1 to 5 years and development of dental caries. All 4 PCS were conducted in HIC.
- 9.44 All 4 PCS (in a total of 32,982 participants) reported an association between consumption of SSBs at ages 1 to 1.5 years and development of ECC up to 3.5 years later compared with not consuming SSBs.
- 9.45 One PCS (in 125 participants) reported a caries prevalence OR 3.04 (95% CI 1.07 to 8.64) at age 18 months for children who consumed SSBs at ages 6 to 18 months, adjusted for age only.
- 9.46 One PCS (in 31,202 participants) reported a caries incidence OR 1.56 (95% CI 1.46 to 1.65) at age 3 years for children who consumed SSBs at age 1.5 years, adjusted for SES, tooth brushing frequency and use of a fluoride agent, falling asleep with a bottle.

- 9.47 One PCS (in 289 participants) reported an ECC experience OR 2.2 (95% CI 1.1 to 4.5) at ages 2 and 3 years for nightly consumption of SSBs at age 1 year; and an ECC experience OR 1.5 (95% CI 0.8 to 2.8) at ages 2 and 3 years for sometimes consuming SSBs at night at age 1 year. The analyses were adjusted for SES and tooth brushing frequency.
- 9.48 None of the 3 PCS adjusted for intake of dietary sugars from the rest of the diet.
- 9.49 Conversely, the fourth PCS (in 1366 participants) reported that not consuming SSBs at age 1 was associated with an OR 2.26 (95% CI 1.07 to 4.77) for being caries free at age 5 years. It is unclear whether the analysis was adjusted.

## Summary: sugar-sweetened beverage consumption and development of dental caries

- 9.50 The evidence identified from SRs on SSB consumption and development of dental caries in children aged 1 to 5 years is summarised in Table 9.2.

**Table 9.2. Summary of the evidence on sugar-sweetened beverage consumption and development of dental caries**

Exposure	Outcome	Direction of association <sup>1</sup>	Certainty of evidence
Sugar-sweetened beverage consumption	ECC (incidence, prevalence or experience)	↑	Limited

<sup>1</sup> Direction of association for reported outcomes: ↑ increase

- 9.51 The available evidence from SRs on the relationship between SSB consumption and development of dental caries in children aged 1 to 5 years is from 1 SR given a moderate confidence rating using the AMSTAR 2 tool.
- 9.52 Evidence from 4 PCS included in Moynihan et al (2019) suggests that higher SSB consumption in children aged 1 to 5 years is associated with increased ECC (incidence, prevalence or experience) compared with not consuming SSBs. The evidence was graded 'limited' due to the small number of studies and limited adjustment for key confounding factors (for example, free sugars in the rest of the diet).
- 9.53 The evidence supports current advice that the amount and frequency of consumption of food and drinks containing sugars should be minimised to prevent caries development in children aged 0 to 6 years old (DHSC, 2021a).

## Breastfeeding beyond the first year of life and development of dental caries

- 9.54 Infant feeding and oral health up to the age of 1 year was considered in SACN's report '[Feeding in the first year of life](#)' (2018). For this report, consideration was given to SR evidence of any effect, protective or otherwise, of breastfeeding into the second year of life and beyond on the development of dental caries between the ages of 1 and 5 years.
- 9.55 Two SRs with MAs were identified that examined the relationship between breastfeeding for 12 months and beyond and development of dental caries (Moynihan et al, 2019; Tham et al, 2015). While Moynihan et al (2019) included MAs on other exposures, the review authors considered data on breastfeeding (and other modes of feeding) unsuitable for pooling in its evidence synthesis. Tham et al (2015) pooled estimates from PCS together with those from cross-sectional and case-control studies. Therefore, for this report, study findings from both SRs were considered individually. A third SR without MA (Hooley et al, 2012b) which did not specifically search for infant and young child feeding but included 1 additional PCS that examined the relationship between breastfeeding beyond 12 months and development of dental caries. Therefore, this additional PCS was also considered.
- 9.56 Moynihan et al (2019)(AMSTAR 2 confidence rating: moderate) included 1 PCS; Tham et al (2015) (AMSTAR 2 confidence rating: low) included 2 PCS; and Hooley et al (2012b)(AMSTAR 2 confidence rating: critically low) included 1 additional PCS that examined the relationship between breastfeeding for 12 months and beyond and development of ECC. Primary studies included in the SRs were mainly conducted in UMICs.
- 9.57 All 4 PCS (in a total of 1778 participants) reported no association between breastfeeding for 12 months or longer and development of ECC or severe-ECC (S-ECC) compared with breastfeeding for less than 12 months. Quantitative findings were reported for 3 PCS.
- 9.58 One PCS (in 870 participants) reported a mean ratio of decayed, missing, filled surfaces in primary dentition of 0.9 (95% CI 0.6 to 1.3) at age 5 years; and a RR (relative risk) for S-ECC at age 5 years of 1.0 (95% CI 0.6 to 1.6).
- 9.59 The second PCS (in 315 participants) reported an adjusted OR for ECC at age 41 to 50 months of 1.09 (95% CI 0.45 to 2.71); while the third PCS (in 715 participants) reported an adjusted PR for S-ECC at age 38 months of 1.39 (95% CI 0.73 to 2.64).
- 9.60 All PCS adjusted for SES and proxy measures of free sugars in the diet (for example, added sugars to feeding bottles, introduction to sweets before age 6 months, consumption of SSBs in the complementary diet). One PCS also reported

adjusting for oral hygiene practices (tooth brushing and use of fluoride toothpaste or gel).

- 9.61 Two PCS also compared breastfeeding for 24 months and beyond with breastfeeding for less than 24 months. One PCS (in 1303 participants) reported an association between breastfeeding for 24 months and beyond and higher risk of S-ECC at age 5 years compared with breastfeeding for less than 24 months (RR 2.4; 95% CI 1.7 to 3.3), adjusted for SES and free sugars intake. The other PCS (in 537 participants) reported no association between breastfeeding for 24 months and beyond and S-ECC prevalence at age 38 months (PR 1.17; 95% CI 0.85 to 1.78), although with a wide confidence interval. The analysis was adjusted for SES and a measure of free sugars intake (added sugar in bottle).

## Summary: breastfeeding beyond the first year of life and development of dental caries

- 9.62 The evidence identified from SRs on breastfeeding beyond the first year of life and development of dental caries is summarised in Table 9.3.

**Table 9.3. Summary of the evidence on breastfeeding beyond the first year of life and development of dental caries**

Exposure	Outcome	Direction of association	Certainty of evidence
Breastfeeding ≥12 months compared with <12 months	Early childhood caries (ECC) or severe ECC	No association	Limited
Breastfeeding ≥24 months compared with <24 months	Early childhood caries (ECC) or severe ECC	Not applicable	Insufficient

- 9.63 The available evidence from SRs on any relationship between continued breastfeeding and development of dental caries in children is from 3 SRs, 1 given a moderate confidence rating using the AMSTAR 2 tool, the others given low and critically low confidence ratings.
- 9.64 Evidence from 4 PCS from the 3 SRs by Hooley et al (2012b); Moynihan et al (2019); Tham et al (2015) suggests that there is no association between breastfeeding for 12 months and longer and development of ECC or S-ECC compared with breastfeeding for less than 12 months. The evidence was graded 'limited' due to the small number of studies (quantitative findings were reported only for 3 studies), lack of consideration of study power, lack of adjustment for oral hygiene practices, and unclear generalisability of the findings to the UK because most of the PCS were conducted in UMIC.

- 9.65 There was insufficient evidence from SRs to enable conclusions to be drawn on any relationship between breastfeeding for 24 months and beyond and development of ECC or S-ECC as fewer than 3 primary studies included in the SR examined this relationship.

## **Use of infant feeding bottles for milk feeds beyond 12 months and development of dental caries**

- 9.66 Current UK advice states that when using a bottle for feeding, the bottle should not contain anything other than breast milk, formula milk or water, and that sugar should not be added (NHS, 2022). At the same time, young children aged 1 year and over should be discouraged from drinking from a bottle (DHSC, 2021a).
- 9.67 One SR without MA (Hooley et al, 2012b) was identified that included PCS that examined the relationship between use of infant feeding bottles for milk feeds beyond 12 months and development of dental caries. However, the research question and search strategy of this SR encompassed any parental or caregiver practices that might relate to dental caries development and was not particular to modes of infant and young child feeding. Therefore, the literature search conducted by Hooley et al (2012b) cannot be said to be comprehensive for identifying studies on the use of infant bottles for milk feeds and the development of dental caries.
- 9.68 Hooley et al (2012b) (AMSTAR 2 confidence rating: critically low) included 1 PCS (in 592 participants) that reported no association between use of infant feeding bottles for milk feeds beyond 12 months and development of ECC at age 18 to 36 months (quantitative findings NR). The study did not adjust for key confounding factors. In addition, the contents of the bottles used in the study was not stated. However, it can be assumed that this was either milk or formula milk given that the study compared “being bottle fed” with “being breastfed”.

## **Summary: use of infant feeding bottles for milk feeds and development of dental caries**

- 9.69 The evidence identified from SRs on use of infant feeding bottles for milk feeds beyond 12 months and development of dental caries is summarised in Table 9.4.

**Table 9.4. Summary of the evidence on use of infant bottles for milk feeds and development of dental caries**

Exposure	Outcome	Direction of association	Certainty of evidence
Use of infant bottles for milk feeds	Early childhood caries (ECC)	Not applicable	Insufficient

- 9.70 The available evidence from SRs on the relationship between use of infant bottles for milk feeds beyond 12 months and development of dental caries is from 1 SR without MA given a critically low confidence rating using the AMSTAR 2 tool.
- 9.71 There was ‘insufficient’ evidence from SRs to enable conclusions to be drawn on the relationship between use of infant bottles for milk feeds beyond 12 months and development of dental caries as fewer than 3 primary studies included in the SR examined this relationship. However, as drinks containing free sugars may be given by bottle, the current advice (DHSC, 2021a) that young children aged 1 year and over should be discouraged from drinking from a bottle remains valid for helping prevent dental caries and supporting broader young child development.

## Night time bottle milk feeds and development of dental caries

- 9.72 Salivary flow, and therefore the ability of salivary bicarbonate to neutralise plaque acids, is reduced at night time and when in a supine position. Therefore, sugars in milk feeds consumed at night time are potentially more cariogenic. Night feeding in this context is a situation where a child is left with a bottle overnight. The bottle may contain cows’ milk or formula milk. This section does not include other foods and drinks consumed around bedtime.
- 9.73 One SR without MA (Hooley et al, 2012b) (AMSTAR 2 confidence rating: critically low) included 2 PCS that examined the relationship between bottle milk feeds at night time in children beyond 12 months and development of dental caries. As the research question and search strategy of this SR encompassed any parental or caregiver practices that might relate to dental caries development and was not specific to modes of infant and young child feeding, the literature search conducted by Hooley et al (2012b) cannot be said to be comprehensive for identifying studies on night time bottle feeding and the development of dental caries.
- 9.74 The 2 PCS (in a total of 1764 participants) reported that being put to bed with a bottle of milk (type of milk not specified) after the age of 1 year was associated with increased ECC at ages 3 to 7 years (quantitative findings NR). Both PCS adjusted for tooth brushing; 1 PCS also adjusted for a crude measure of free sugar

intake (frequency of consumption of between-meal sweet foods and drink). Neither PCS included breastfeeding during the night as a comparator nor was it clear whether this mode of feeding was examined.

## Summary: night time bottle milk feeds and development of dental caries

- 9.75 The evidence identified from SRs on night time bottle milk feeds beyond 12 months and development of dental caries is summarised in Table 9.5.

**Table 9.5. Summary of the evidence on night time bottle milk feeds beyond 12 months and development of dental caries**

Exposure	Outcome	Direction of association	Certainty of evidence
Night time bottle milk feeds beyond 12 months	Early childhood caries (ECC)	Not applicable	Insufficient

- 9.76 The available evidence from SRs on the relationship between night time bottle milk feeds beyond 12 months and development of dental caries is from 1 SR without MA given a critically low confidence rating using the AMSTAR 2 tool.
- 9.77 There was ‘insufficient’ evidence from SRs to enable conclusions to be drawn on any relationship between night time bottle milk feeds beyond 12 months and development of dental caries in young children as fewer than 3 primary studies included in the SR examined this relationship. However, as drinks containing free sugars may be given by bottle, the current advice (DHSC, 2021a) that young children aged 1 year and over should be discouraged from drinking from a bottle remains valid for helping prevent caries development and supporting broader young child development.

## Use of infant feeding bottles to consume liquids containing free sugars and development of dental caries

- 9.78 Current UK advice states that when using a bottle for feeding, the bottle should not contain anything other than breast milk, formula milk or water, and that sugar should not be added (NHS, 2022). At the same time, young children aged 1 year and over should be discouraged from drinking from a bottle (DHSC, 2021a).
- 9.79 One SR (Moynihan et al, 2019) (AMSTAR 2 confidence rating: moderate) was identified that examined the relationship between use of infant feeding bottles to

consume liquids containing free sugars in children aged 1 to 5 years and development of dental caries. An additional SR (Hooley et al, 2012b) (AMSTAR 2 confidence rating: critically low) also included PCS that examined this relationship. However, the research question and search strategy of the SR encompassed any parental or caregiver practices that might relate to dental caries development and was not specific to modes of infant or young child feeding. Therefore, the literature search conducted by Hooley et al (2012b) cannot be said to be comprehensive for identifying studies on the use of infant feeding bottles to consume liquids containing free sugars and development of dental caries.

- 9.80 Moynihan et al (2019) included 3 PCS that examined the relationship between use of infant feeding bottles to consume liquids containing free sugars in children aged 1 to 5 years and development of dental caries. The PCS were mostly conducted in HIC.
- 9.81 All 3 PCS (in a total of 938 participants) reported an association between consumption of liquids containing free sugars (for example, fruit juices, 'soft drinks' or sweetened milk) at ages 12 to 39 months and development of ECC or S-ECC up to 3 years later compared with not consuming liquids containing free sugars.
- 9.82 Quantitative findings were reported for 2 PCS. One PCS (in 315 participants) reported that use of infant feeding bottles for consuming sweetened liquids other than milk at ages 29 to 39 months was associated with OR of ECC 2.47 (95% CI 1.23 to 5.05) at age 41 to 50 months. One PCS (in 334 participants) reported that use of infant feeding bottles for consuming fruit juices or soft drinks at age 12 months was associated with a RR of S-ECC 1.41 (95% CI 1.08 to 1.86) at age 4 years. Both PCS adjusted for SES but neither adjusted for intake of dietary sugars from the rest of the diet. 1 PCS also adjusted for oral hygiene practices (tooth brushing frequency and use of fluoride toothpaste or gel).
- 9.83 The third PCS did not report data for the comparator group (for example, children who received milk or water from an infant feeding bottle) and did not adjust for any confounders.
- 9.84 Hooley et al (2012b) included 1 additional PCS that reported no association between use of infant feeding bottles for consuming sweetened milk beyond age 12 months and later development of dental caries. Quantitative details were not reported, and the analysis was unadjusted.

## Summary: use of infant feeding bottles to consume liquids containing free sugars and development of dental caries

- 9.85 The evidence identified from SRs on use of infant feeding bottles to consume liquids containing free sugars and development of dental caries in children aged 1 to 5 years is summarised in Table 9.6.

**Table 9.6. Summary of the evidence on use of infant feeding bottles to consume liquids containing free sugars and development of dental caries**

Exposure	Outcome	Direction of association	Certainty of evidence
Use of infant feeding bottles to consume liquids containing free sugars	Early childhood caries (ECC) or severe ECC	Not applicable	Insufficient

- 9.86 The available evidence from SRs on the relationship between use of infant feeding bottles to consume liquids containing free sugars and development of dental caries in children aged 1 to 5 years is from 2 SRs, 1 given a moderate confidence rating using the AMSTAR 2 tool, the other given a critically low rating.
- 9.87 Although evidence from 3 PCS included in Moynihan et al (2019) and Hooley et al (2012b) suggested that there is a relationship between use of infant feeding bottles to consume liquids containing free sugars and development of ECC or S-ECC, the evidence was graded 'insufficient'. This was due to the small number of studies that reported an association (3 PCS), including 1 PCS for which data for the control group was not reported, and lack of adjustment for free sugars in the rest of the diet and oral hygiene practices.
- 9.88 However, the current advice that young children aged 1 year and over should be discouraged from drinking from a bottle (DHSC, 2021a) remains valid for helping prevent dental caries development and supporting broader young child development.

## Foods containing free sugars and development of dental caries

- 9.89 Current advice states that the amount and frequency of consumption of food and drinks containing sugars should be minimised to prevent caries development in children aged 0 to 6 years old (DHSC, 2021a).

- 9.90 For this report, 1 SR with MA (Moynihan et al, 2019) (AMSTAR 2 confidence rating: moderate) and 1 SR without MA (Baghlaf et al, 2018) (AMSTAR 2 confidence rating: high) was identified that examined the relationship between consumption of foods containing free sugars in children aged 1 to 5 years and development of dental caries. A third SR without MA (Hooley et al, 2012b) (AMSTAR 2 confidence rating: critically low), which did not specifically search for studies in this topic area, also included PCS on this relationship and was therefore also considered. The 3 SRs identified 4 PCS in children aged 1 to 5 years. The PCS were mostly conducted in HIC.
- 9.91 All 4 PCS (in a total of 2427 participants) reported that frequent consumption of foods containing free sugars was associated with increased development of dental caries in children aged 1 to 5 years. However, the exposures (both in terms of type of food, frequency of consumption, and time of day when these were consumed) across the studies were heterogeneous, making direct comparison between the studies difficult. Quantitative details were reported for 2 PCS.
- 9.92 One PCS (in 1576 participants) reported that consumption of sweets at bedtime without brushing teeth at the ages of 3 to 6 years was associated with an OR of 1.33 (95% CI 1.01 to 1.68; p-value not reported) of experiencing a caries increment when examined 1 year later. The study adjusted for frequency of between-meal intake of sweets, tooth brushing, and fluoride exposure (although not specifically bedtime tooth brushing).
- 9.93 One PCS (in 334 participants) reported that consumption of foods with a high density of added sugars (50% simple carbohydrates per 100g food) at age 12 months was associated with a RR of S-ECC of 1.43 (95% CI 1.08 to 1.89) at age 4 years compared with not consuming such foods, adjusted for SES and bottle use for fruit juices or soft drinks at baseline.

## Summary: foods containing free sugars and development of dental caries

- 9.94 The evidence identified from SRs on consumption of foods containing free sugars and development of dental caries in children aged 1 to 5 years is summarised in Table 9.7.

**Table 9.7. Summary of the evidence on foods containing free sugars and development of dental caries**

Exposure	Outcome	Direction of association <sup>1</sup>	Certainty of evidence
Foods containing free sugars and development of dental caries	Early childhood caries (ECC) or severe ECC (caries incidence, prevalence, experience)	Not applicable	Insufficient

- 9.95 The available evidence from SRs on the relationship between consumption of foods containing free sugars and development of dental caries in children aged 1 to 5 years is from 3 SRs, 1 given a high confidence rating using the AMSTAR 2 tool and the others given moderate and critically low confidence ratings.
- 9.96 Although evidence from 4 PCS included in Baghlaf et al (2018); Hooley et al (2012b); Moynihan et al (2019) suggests that consumption of foods containing free sugars in children aged 1 to 5 years is associated with increased development of ECC or S-ECC, the evidence was graded 'insufficient'. This was due to heterogeneity of exposures, lack of quantitative data for 2 of the 4 PCS to judge effect sizes, and limited adjustment for key confounding factors (for example, SES and intake of dietary sugars from rest of the diet).
- 9.97 However, based on the strong SR evidence identified on free sugars intake and development of dental caries (see paragraphs 9.39 and 9.40), current advice that the amount and frequency of consumption of food and drinks containing sugars should be minimised to prevent caries development in children aged 0 to 6 years remains valid (DHSC, 2021a).

## **Milk and dairy consumption and development of dental caries**

- 9.98 One SR without MA (Dror & Allen, 2014) (AMSTAR 2 confidence rating: critically low) was identified that included 1 PCS that examined the relationship between the consumption of milk and other dairy products and dental caries in children aged 1 to 5 years. For evidence related to milk and dairy consumption and other health outcomes, see [Dairy foods](#) (chapter 5) and [Milk](#) (chapter 6).
- 9.99 The PCS (in 642 participants) reported that the median milk consumption at ages 2 and 3 years was lower in children with surface and tooth level dental caries at ages 4 to 7 years (estimate of association NR; 95% CI NR;  $p < 0.05$ ). The PCS also reported that low cumulative (below the median) dairy consumption (excluding milk) at ages 1 to 5 years was associated with fewer surface caries at ages 4 to 7 years (estimate of association NR; 95% CI NR;  $p < 0.01$ ) compared with higher cumulative (above the median) dairy consumption (excluding milk), adjusted for sex, age at dental examination, fluoride exposure and SSB consumption.

## **Summary: milk and dairy consumption and oral health**

- 9.100 The evidence identified from SRs on milk and dairy consumption and oral health in children aged 1 to 5 years is summarised in Table 9.8.

**Table 9.8 Summary of the evidence on dairy consumption and oral health**

Exposure	Outcome	Direction of association	Certainty of evidence
Milk consumption	Development of dental caries	Not applicable	Insufficient
Non-milk dairy product consumption	Development of dental caries	Not applicable	Insufficient

- 9.101 The available evidence on the relationship between milk and dairy consumption and oral health in children aged 1 to 5 years is from 1 SR given a critically low confidence rating using the AMSTAR 2 tool.
- 9.102 There was 'insufficient' evidence from SRs to enable conclusions to be drawn on any relationship between dairy consumption and dental health in children 1 to 5 years as fewer than 3 primary studies included in the SR examined this relationship.

## **Continued breastfeeding or use of bottles for feeding and malocclusion**

- 9.103 Malocclusion describes the alignment of teeth which are considered not to be in a normal position in relation to adjacent teeth (that is, the teeth are not correctly aligned) (Nelson, 2019). The term covers a range of disorders relating to development which stem from a variety of causes.
- 9.104 Malocclusion has been suggested to vary between breast and bottle fed children. The proposed biological mechanism is that children who are breastfed have more facial muscle activity compared with bottle fed children and this promotes craniofacial growth and jaw bone development. The growth of the face is affected by the infant's use of their facial muscles during feeding and suckling.
- 9.105 In its report '[Feeding in the first year of life](#)', SACN concluded that infants who were breastfed had a lower risk of malocclusion development than children who were not breastfed (SACN, 2018).
- 9.106 For this report, 1 SR with MA (Thomaz et al, 2018) and 1 SR without MA (Hermont et al, 2015) were identified which examined the relationship between breastfeeding beyond 12 months and malocclusion. All the studies identified by Hermont et al (2015) on this relationship were also included in Thomaz et al (2018). As Thomaz et al (2018) is the more recent and comprehensive of the 2 SRs, findings relating to breastfeeding are reported from this SR only. Relevant studies included in the SR were conducted in HIC and UMIC.
- 9.107 Thomaz et al (2018) (AMSTAR 2 confidence rating: moderate) reported that breastfeeding beyond 12 months was associated with a decreased odds of

malocclusion compared with breastfeeding for less than 12 months (OR 0.38; 95% CI 0.24 to 0.60;  $p < 0.0001$ ;  $I^2 = 0$ ; random-effects model; 3 PCS, 419 participants). Of the 3 PCS included in the subgroup MA, 1 adjusted for non-nutritive sucking habits, which the SR authors considered an important confounding factor. In terms of types of malocclusion, breastfeeding for 12 months and beyond was associated with a decreased odds of overjet compared with breastfeeding for less than 12 months (OR 0.30; 95% CI 0.16 to 0.57;  $p = 0.0003$ ;  $I^2 = 0$ ; random-effects model; 2 PCS, 272 participants). A lack of evidence from PCS prevented the estimation of summary measures of associations with other types of malocclusion (Thomaz et al, 2018).

- 9.108 1 SR without MA (Hermont et al, 2015) (AMSTAR 2 confidence rating: moderate) was identified that included 1 PCS that examined the relationship between use of bottles for feeding beyond 12 months and development of malocclusion. The PCS (in 80 participants) reported an association between use of bottles for feeding at age 12 and 30 months and posterior crossbite at age 30 months (estimates of association NR; 95% CI NR;  $p = 0.02$  and  $p = 0.04$ , respectively). The study did not control for any potential confounding factors. The clinical importance of these findings is also unclear as there is some evidence that some malocclusions can be self-corrected during the transition from primary to permanent dentition (Thomaz et al, 2018).

## Summary: continued breastfeeding or use of bottles for feeding and malocclusion

- 9.109 The evidence identified from SRs on breastfeeding or use of bottles for feeding beyond 12 months and development of malocclusion is summarised in Table 9.9.

**Table 9.9. Summary of the evidence on breastfeeding or use of bottles for feeding beyond 12 months and malocclusion**

Exposure	Outcome	Direction of association <sup>1</sup>	Certainty of evidence
Breastfeeding beyond 12 months	Development of malocclusion	↓ (protective)	Moderate
Use of bottles for feeding beyond 12 months	Development of malocclusion	Not applicable	Insufficient

<sup>1</sup> Direction of association for reported outcomes: ↓ inverse association

- 9.110 The available evidence from SRs on the relationship between breastfeeding or use of bottles during feeding beyond 12 months, and the development of malocclusion is from 2 SRs (1 with MA), 1 given a moderate confidence rating using the AMSTAR 2 tool, the other given a low confidence rating.

- 9.111 Evidence from a MA of 3 PCS conducted by Thomaz et al (2018) suggests that breastfeeding beyond 12 months protects against the development of malocclusion. The evidence was graded 'moderate' due to the large effect size and lack of statistical heterogeneity. This evidence is consistent with the SACN report ['Feeding in the first year of life'](#) which found that 'ever breastfed' children may be less likely to develop malocclusions compared with 'never breastfed' children (SACN, 2018).
- 9.112 There was 'insufficient' evidence from SRs to enable conclusions to be drawn on the relationship between use of bottles for feeding beyond 12 months and development of malocclusion as fewer than 3 primary studies included in the SR by Hermont et al (2015) examined this relationship.

## **Body weight and development of dental caries**

- 9.113 Concern over high levels of childhood obesity and the likelihood of excess free sugars consumption being a shared risk factor for both obesity and dental caries led to a population study which matched data on individuals' height, body weight and dental caries experience among over 67,000 children at age 5 years. In comparison with children of a healthy weight, dental caries was significantly more likely among those with overweight or very overweight, once confounding factors were accounted for (deprivation, ethnicity and water fluoridation status) (PHE, 2019b).
- 9.114 To determine whether there is a relationship between weight status and development of dental caries, studies need to test against a full range of body mass index (BMI) or weight categories. This is because there is some evidence that the association between BMI or body weight and development of dental caries in primary dentition is U-shaped and that children with overweight or underweight have an increased risk of developing dental caries (Hooley et al, 2012a), especially for primary dentition (Tanner et al, 2022).
- 9.115 One SR without MA (Hooley et al, 2012a) (AMSTAR 2 confidence rating: low) was identified that examined the relationship between BMI or body weight and development of dental caries in children and adolescents and included 1 PCS that examined this relationship in children aged 1 to 5 years. The PCS (in 788 participants) was conducted in low-income households living in a HIC and reported that children in the highest quartile for weight-for-age (as a percentile according to the US Centers for Disease Control and Prevention growth charts) at a mean age 2.6 years was associated with a greater risk of developing dental caries 2 years later. The study adjusted for age, SSB consumption, tooth brushing, baseline caries and SES. However, the SR noted that this study assumed a linear

relationship between body weight and risk of dental caries, even though almost 25% of the children in the sample had a low weight-for-age percentile.

## Summary: body weight and development of dental caries

- 9.116 The evidence identified from SRs on the relationship between body weight and development of dental caries in children aged 1 to 5 is summarised in Table 9.10.

**Table 9.10. Summary of the evidence on body weight and development of dental caries**

Exposure	Outcome	Direction of association	Certainty of evidence
Body weight	Development of dental caries	Not applicable	Insufficient

- 9.117 The available evidence from SRs on any relationship between body weight and development of dental caries in children aged 1 to 5 years is from 1 SR without MA given a low confidence rating using the AMSTAR 2 tool. There was 'insufficient' evidence from SRs to draw conclusions on the relationship between BMI and caries risk in children as there were fewer than 3 primary studies included in the SR that examined this relationship. More quality research that considers the full spectrum of weight status against dental caries risk is needed.

## Vitamin D status and oral health

- 9.118 The impact of clinical vitamin D deficiency on tooth development has been recognised for many years and has been described in both vitamin D dependent rickets (Kikuchi et al, 1988; Zambrano et al, 2003) and in hypophosphataemic vitamin D resistant rickets (Goodman et al, 1998; Murayama et al, 2000; Nishino et al, 1990; Seow et al, 1995). Teeth are relatively protected during the mineralisation phase so effects on teeth are fewer than those seen skeletally (SACN, 2016). However, there can be disturbances of both enamel and dentine formation. The enamel that develops is hypoplastic, pitted and relatively thin, with reduced mineralisation making the teeth more susceptible to caries. The dentine is abnormal in macroscopic structure and has lower than normal levels of mineralisation. Individuals with rickets, a condition usually caused by vitamin D or calcium deficiency, can develop high levels of dental caries and tooth wear that spread rapidly through the enamel and underlying thinned dentine to expose the dental pulp, which results in early pulp death. These changes in the structure of enamel and dentine occur during tooth development from intra-uterine development up to around age 18 years (SACN, 2016).

- 9.119 A number of mechanisms have been proposed to explain how low vitamin D status can contribute to increased risk of dental caries in children. Low vitamin D status may lower the concentrations of salivary antimicrobial peptides (AMP) that target cariogenic bacteria, increasing the risk of dental caries (Seminario & Velan, 2016). However, data on any relationship between low vitamin D status and salivary AMP concentrations in young children are lacking; while data pertaining to any relationship between salivary AMP and risk of dental caries in children aged under 5 years have been cross-sectional and therefore inconclusive (Colombo et al, 2016; Malcolm et al, 2014).
- 9.120 Animal studies have shown that vitamin D deficiency could increase the risk of dental caries by lowering the flow rate and calcium concentration of parotid saliva (Glijer et al, 1985; Peterfy et al, 1988). However, data in young children are lacking.
- 9.121 This report found a paucity of evidence on the relationship between vitamin D status and oral health in children aged 1 to 5 years. Only 1 SR with MA (Hujoel, 2013) was identified since SACN (2016) that examined the effect of vitamin D supplementation on risk of dental caries in children. The SR included only 2 controlled trials (out of 24 trials in total) in children aged 1 to 5 years, and its findings could not be disaggregated from the findings in older children as these were pooled into a MA. In addition, the majority of the studies included in the SR (including the 2 studies in children aged 1 to 5 years) were conducted between or during the 2 world wars when nutritional and lifestyle exposures (such as fluoride exposure) as well as public health concerns (such as a greater prevalence of rickets) were different from the present day. Research into vitamin D that reflects contemporary lifestyles is needed.
- 9.122 For details on vitamin D and its effect on health outcomes other than oral health, see [chapter 4](#).

# 10 Risks of chemical toxicity

- 10.1 To complement SACN's review of the scientific evidence underpinning current dietary recommendations for infants and young children in the UK, the Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT) was asked to examine the risks of toxicity from chemicals in the diet of infants and young children and to consider whether current advice to government should be revised.
- 10.2 In 2015, COT identified a number of dietary chemicals that might pose a risk to infants and young children on the basis of their known or suspected adverse effects and for which advice might be needed.
- 10.3 Subsequently, COT published an [overarching statement on the potential risks from extraneous chemicals in the diet of infants aged 0 to 12 months and young children aged 1 to 5 years](#) (COT, 2019). In 2020, COT published an [addendum to the overarching statement](#) on the potential risk of the remaining chemicals (COT, 2020). Table 10.1 provides an overview of the conclusions for all chemicals for children aged 1 to 5 years. A summary of COT's evaluations on potential chemical risks from the infant diet is provided in the SACN report '[Feeding in the first year of life](#)' (2018).
- 10.4 A number of chemicals identified for review were not included in the overarching statement or the addendum. Some of these have been subject to a full review, while others were considered to be either outside COT's remit or for it to be unnecessary to change COT's existing advice to government in the absence of any new data. A full list of all chemicals identified by COT, with the respective links to the discussion papers or statements where applicable, can be found in Table 10.2.

**Table 10.1. Summary of the substance evaluations included in the 2019 COT overarching statement and the 2020 COT addendum to the overarching statement on potential chemical risks from the diet of young children (1 to 5 years)**

Substance category	Chemical considered	Summary of COT conclusions
Contaminants and process contaminants	Chlorate (COT, 2019)	<p>The data collected by the Food Standards Agency (FSA) on chlorate has been submitted to, and forms part of, the evaluation performed by the European Food Safety Authority (EFSA). While further data collection has been undertaken, the data are unlikely to change the (UK) exposure assessment undertaken by EFSA or conclusions drawn therefrom. COT therefore did not consider it necessary to undertake a full risk assessment itself.</p> <p>COT agrees with the overall conclusion by EFSA. <b>Chronic dietary exposure to chlorate is of potential concern for high consumers, particularly to individuals with mild to moderate iodine deficiency.</b> Drinking water was the major contributor, at up to 40 to 60%. Single acute exposures to chlorate at levels found in food and drinking water, however, are unlikely to cause adverse effects, including in vulnerable individuals.</p>
Contaminants and process contaminants	Furan and methylfurans (COT, 2019)	<p>Non-neoplastic effects of furan are not of toxicological concern, the combined exposures of furan and methylfurans however are of potential toxicological concern. Neoplastic effects of furan for young children<sup>1</sup> for ready-to-eat-meals and total exposure are of <b>potential toxicological concern</b>. However, there is a level of uncertainty concerning the carcinogenic mode of action (MoA) of furan and whether it is directly genotoxic and COT acknowledges that its assessment is based on worst case assumptions.</p> <p>The lack of occurrence data for methylfurans add to the uncertainties for the summed exposure and could therefore lead to an over as well as underestimation of risk.</p>
Contaminants and process contaminants	Hexachloro-cyclohexanes (COT, 2020)	COT concluded that the exposures in the diet of young children aged 1 to 5 years are <b>not of toxicological concern</b> .

Substance category	Chemical considered	Summary of COT conclusions
Contaminants and process contaminants	Legacy chemicals <sup>2</sup> (COT, 2019)	Although these chemicals are persistent in the environment, their levels have decreased since their use was banned. <b>As the levels for legacy chemicals are expected to further decline</b> , COT confirmed the conclusions of its previous assessments, that <b>there is no indication of concern</b> for health from the presence of these chemicals in the diet of young children.
Contaminants and process contaminants	Monochloro-propanediol (MCPD), its fatty acid and glycidol (COT, 2020)	Given the limited UK-specific occurrence data, COT assessed 3-MCPD, its fatty acid esters and glycidol, based on the latest EFSA evaluation. Overall, COT agreed that some of EFSA's margin of exposure (MOE) values for glycidol and exceedances of the tolerable daily intake (TDI) for 3-MPCD are of <b>potential concern</b> for young children aged 1 to 5 years. However, as concluded by EFSA, there are a number of uncertainties in these risk assessments such as uncertainty in the reference point used as a basis for the calculation of the MOE values for glycidol, and the long-term effects of 3-MCPD on the male reproductive system, as well as in the occurrence data.
Contaminants and process contaminants	Perchlorate (COT, 2019)	The data collected by the FSA on perchlorate have been submitted to, and are part of, the evaluation performed by EFSA. The COT therefore did not consider it necessary to undertake a full risk assessment itself. In agreement with EFSA, the COT concluded that while there are considerable uncertainties in the assessment, the chronic and short term estimated exposures for young children are of <b>potential concern</b> , particularly in the case of a mild to moderate iodine deficiency.
Contaminants and process contaminants	Polycyclic aromatic hydrocarbons (PAHs) (COT, 2020)	COT concluded that the intakes of PAHs Benzo[a]pyrene (BaP) and PAH4 (the sum of benzo[a]anthracene, chrysene, benzo[b]fluoranthene, and benzo[a]pyrene) from human breast milk and food are of <b>low concern</b> for health for children aged 1 to 5 years. Intakes from soil and dust are not expected to contribute markedly to lifetime exposure.

Substance category	Chemical considered	Summary of COT conclusions
Contaminants and process contaminants	Tetrabromo-bisphenol (COT, 2020)	Given the absence of genotoxicity, tumours only at high doses, large MOEs, and conservatism of exposure estimates based on non-detects, an MOE of 100 was considered to be sufficiently protective for human health. Thus, the calculated MOEs for UK chronic dietary exposures were considered <b>not to be cause for concern</b> for children aged 1 to 5 years.
Food Additives	Food additives (COT, 2019)	The additives regulation applies to all foods produced, including foods specifically for young children. Therefore, COT deemed it not necessary to assess food additives again in these age groups.
Food Additives	Sweeteners (COT, 2020)	COT concluded that the exposures in the diet of children aged 1 to 5 years of the most commonly used sweeteners in the UK (aspartame, acesulfame K, saccharine, sorbitol and xylitol, stevia and sucralose) were <b>not of toxicological concern</b> .
Natural Toxins - Mycotoxins	Aflatoxin (COT, 2020)	Aflatoxin levels in all samples in the Total Diet Survey (TDS) were below their respective limit of quantification (LOQ). However, <b>given that aflatoxins are genotoxic and carcinogenic</b> their presence in food is always undesirable and when exposure was estimated based on their LOQs, it was <b>not possible to exclude a safety concern</b> .
Natural Toxins - Mycotoxins	Citrinin (COT, 2020)	COT concluded that exposures to citrinin are <b>not of toxicological concern for nephrotoxicity</b> . However, it was noted that due to lack and limitations of the available data, <b>a concern for genotoxicity and carcinogenicity cannot be excluded</b> .
Natural Toxins - Mycotoxins	Cyclopiazonic acid (COT, 2020)	COT concluded that the exposures in the diet of children aged 1 to 5 years are <b>not of toxicological concern</b> .

Substance category	Chemical considered	Summary of COT conclusions
Natural Toxins - Mycotoxins	Diacetoxyscirpenol (COT, 2020)	COT concluded that the exposures in the diet of children aged 1 to 5 years are <b>not of toxicological concern</b> .
Natural Toxins - Mycotoxins	Deoxynivalenol (DON) and its acetylated/modified forms (COT, 2020)	COT concluded that exposures to DON, 15-Ac-DON, 3-Ac-DON, and the sum of all 3 forms in the diets of children aged 1 to 5 years are <b>unlikely to be of toxicological concern</b> . However, COT noted that the sum of all forms is not based on individual measured values but on summing the respective averages of the concentrations provided. Therefore, the estimated exposures could be an overestimation of the actual values.
Natural Toxins - Mycotoxins	Ergot alkaloids (COT, 2020)	COT concluded that the exposures in the diet of children aged 1 to 5 years are <b>not of toxicological concern</b> .
Natural Toxins - Mycotoxins	Fumonisin (COT, 2020)	COT concluded that the exposures in the diet of children aged 1 to 5 years are <b>not of toxicological concern</b> .
Natural Toxins - Mycotoxins	Fusarenon-X (COT, 2020)	COT concluded that exposures to fusarenon-X in the diets of young children aged 1 to 5 years are <b>not of toxicological concern</b> . However, COT noted that there were some uncertainties involved in the extrapolation of the data. The committee agreed that the likelihood of co-occurrence of fusarenon-x with other type B trichothecenes, deoxynivalenol and nivalenol, at the reported levels is low and that acute co-exposure was unlikely to result in adverse toxicological effects.
Natural Toxins - Mycotoxins	Moniliformin (COT, 2020)	COT concluded that the exposures in the diet of young children aged 1 to 5 years are <b>not of toxicological concern</b> .
Natural Toxins - Mycotoxins	Nivalenol (COT, 2020)	COT concluded that the exposures in the diet of young children aged 1 to 5 years are <b>not of toxicological concern</b> .

Substance category	Chemical considered	Summary of COT conclusions
Natural Toxins - Mycotoxins	Patulin (PAT) (COT, 2020)	COT concluded that exposures to PAT in the diets of young children aged 1 to 5 years are <b>not of toxicological concern</b> , but this is <b>contingent on resolution of the genotoxic potential of PAT</b> .
Natural Toxins - Mycotoxins	Sterigmatocystin (COT, 2020)	COT concluded that the exposures in the diet of young children aged 1 to 5 years are <b>not of toxicological concern</b> .
Natural Toxins - Mycotoxins	Zearalenone (COT, 2020)	COT concluded that the exposures in the diet of young children aged 1 to 5 years are <b>not of toxicological concern</b> .
Natural Toxins – other than mycotoxins	Alcohol (COT, 2019)	As children aged 1 to 5 years would not be consuming alcohol directly, any further assessment of alcohol in this age group is not required.
Natural Toxins – other than mycotoxins	Caffeine (COT, 2019)	As children aged 1 to 5 years would not be expected to be consuming high-caffeine beverages, COT concluded that no further assessment of caffeine for this age group is required.
Natural Toxins – other than mycotoxins	Soya <sup>3</sup> phytoestrogens (COT, 2019)	In 2019, COT confirmed their 2013 conclusion that there was no scientific basis for a change in the current advice for children aged 0 to 12 months and that soy formula should be used only in exceptional circumstances. There are also potential concerns for children up to 5 years of age consuming soy drinks. COT considered new data on soya phytoestrogens during their evaluation of plant-based drinks (PBD) and again confirmed their previous conclusions.

Substance category	Chemical considered	Summary of COT conclusions
Natural Toxins – other than mycotoxins	Tropane alkaloids (TAs) (COT, 2020)	<p>Overall, all estimated acute exposures of young children aged 1 to 5 years to (-)-hyoscyamine and (-)-scopolamine or the sum of <b>(-)-hyoscyamine and (-)-scopolamine are unlikely to be of toxicological concern.</b></p> <p>However, COT noted that a number of other TAs of unknown potency were present at higher concentrations than (-)-hyoscyamine and (-)-scopolamine, with some of these reported at detectable levels in up to 26% of the cereal-based samples. In the absence of any toxicological data and health based guidance values (HBGVs) on these TAs there is a <b>high degree of uncertainty to the risks associated with total TAs</b> in the diet.</p>
Nutrients	Chromium (COT, 2019)	<p>Chromium is present in food and the environment largely as Cr(III). EFSA has established a TDI for Cr(III) of 300 µg/kg body weight. Estimated dietary exposures for young children aged 1 to 5 years indicate chromium intake well below the TDI and is therefore considered <b>not to be of toxicological concern.</b> Environmental exposure to Cr(III) from dust, soil and air was calculated to be at most 0.038, 0.15 and 0.036% of the EFSA TDI, respectively and is therefore considered not to be of toxicological concern.</p>
Nutrients	Selenium (COT, 2019)	<p>Overall, COT concluded that estimated dietary exposures to selenium for young children aged 1 to 5 years were below the upper level/limit (UL) and are therefore <b>unlikely to be of toxicological concern.</b></p>
Nutrients	Vitamin A (COT, 2019)	<p>Following its update in 2017, COT concluded that the <b>possibility of adverse effects cannot be excluded in high consumers, primarily those who regularly eat liver.</b> However, if effects did occur it would be in a small proportion of consumers.</p> <p>COT found no scientific basis for a change in current government advice. An assessment of vitamin A intakes in children aged 1 to 5 in the UK is provided in chapter 4.</p>

Substance category	Chemical considered	Summary of COT conclusions
Nutrients	Zinc (COT, 2019)	COT concluded, that overall, estimated dietary exposures do not indicate excessive zinc intakes and are therefore <b>unlikely to be of toxicological concern</b> . However, COT did note that all HBGVs and UL are derived from adults and it is therefore difficult to identify a HBGV or UL that is applicable to young children.

<sup>1</sup> Following EFSA's approach the exposure estimates were calculated using age groups of 4 to 18 months and 18 to 60 months for furan and methylfurans. The latter have been used to cover the conclusions for this report.

<sup>2</sup> (including aldrin, dieldrin, endrin, chlordane, heptachlor, hexachlorobenzene, mirex, toxaphene, DDT, endosulfan, pentachlorobenzene, chlordane).

<sup>3</sup> Update since the overarching statements publication: Soya phytoestrogens are currently undergoing a separate review, with emphasis on soya drink consumption in children aged 6 months to 5 years.

**Table 10.2. Summary of evaluations for chemicals that underwent a separate full COT review**

Chemical considered	Summary of COT conclusions	Web link
Acrylamide	For exposure of young children to acrylamide from infant formula and food, COT concluded that the MOEs <b>did not suggest any concern regarding neurotoxicity</b> . Although human studies do not prove that acrylamide causes cancer, there is a <b>potential concern regarding carcinogenicity</b> relating to exposures in this age group based on extrapolations from experimental studies.	<a href="#">Potential risks from acrylamide in the diet of infants and young children (COT, 2016)</a>
Aluminium	Whilst there are some uncertainties in the overall risk assessment surrounding the potential aggregated exposure, including exposures from soil and dust, COT concluded that estimated exposures of young children to aluminium from the diet, including soya-based infant formula, <b>do not indicate toxicological concerns</b> or a need for any modification in advice to Government.	<a href="#">Potential risks from aluminium in the infant diet (COT, 2013) and addendum (COT, 2016)</a>
Arsenic	COT concluded that the total exposure to inorganic arsenic, from dietary (commercial infant foods and other foods) and non-dietary (soil and dust) sources, in young children aged 1 to 5 years was of <b>potential concern</b> to health. Dietary sources generally contribute more significantly to exposure in these age groups than non-dietary sources such as soil and dust. In general, the food groups making the highest contribution were miscellaneous cereals (including rice and commercial rice products for this age group) and potatoes. Consumption of infant or “adult” rice cakes did not indicate an increased risk, while COT concluded	<a href="#">Statement on arsenic in infants and young children (COT, 2016)</a>  <a href="#">Editorial update</a>

Chemical considered	Summary of COT conclusions	Web link
	that the current government dietary advice not to use rice drink as a substitute for breast milk, infant formula or cows' milk should remain in place. COT reiterated that efforts to reduce the levels of inorganic arsenic in food and water should continue.	
Bisphenol A	EFSA has published a review of BPA recommending a TDI of 0.2 ng/kg bw/day. This is being reviewed by COT.	<a href="#">2023 EFSA opinion on Bisphenol A</a>
Cadmium (Cd)	Although the EFSA tolerable weekly intake (TWI) of Cd was exceeded in some cases, these exceedances were small in magnitude and would not be expected to remain at this level over the decades of bioaccumulative exposure considered by EFSA in setting the HBGV. COT concluded that this was therefore <b>not a major cause for concern</b> .	<a href="#">The potential risks from cadmium in the infant diet (COT, 2018)</a>
Copper	COT concluded that intake of copper by young children aged 1 to 5 years through consumption of breast milk, infant formula, food and drinking water was below the safe upper level derived by the Expert Group on Vitamins and Minerals (EVM) and thus that there was <b>no toxicological concern</b> to the health of infants and young children with normal copper homeostasis.	<a href="#">Potential risks from copper in the diets of infants and young children (COT, 2018)</a>
Dioxins and dioxin-like compounds	In 2018, EFSA established a new TWI for dioxin. Due to the uncertainties in EFSA's assessment, COT did not agree with the newly established TWI and the 7-fold reduction in the TWI was considered too conservative for the database overall. The European Commission (EC) has not yet adopted EFSA's new TWI due to ongoing work at the international level to	<a href="#">Dioxin position paper (COT, 2021)</a>  <a href="#">Risk for animal and human health related to the presence of dioxins and dioxin-like PCBs in feed and food (EFSA, 2018)</a>

Chemical considered	Summary of COT conclusions	Web link
	<p>review the basis and values of the WHO toxic equivalent factors (TEFs). A finalised assessment by the EC is not expected until 2022, at the earliest.</p> <p>COT agreed to undertake its own assessment of dioxin and dioxin-like compounds, however in the meantime the committee did not consider it necessary to alter its existing advice. Any action now would take several years to be reflected in changes in body burden, due to the long half-life of dioxin.</p>	<p><a href="#">EFSA scientific opinion on the risk for animal and human health related to the presence of dioxins and dioxin-like PCBs in feed and food (EFSA, 2018)</a></p>
Hexabromocyclododecane (HBCDDs)	<p>COT concluded that while the <b>level of HBCDDs in the diet of young children was not a cause for concern</b>, the possibility of high levels in household dust continues to be so. Levels in dust should be monitored in houses to determine whether they decrease, now that production and usage of HBCDDs has largely ceased.</p>	<p><a href="#">Hexabromocyclododecanes statement (COT, 2015) and addendum (COT, 2016)</a></p>
Iodine	<p>COT concluded that at current intakes, excess iodine is <b>unlikely to pose a toxicological risk to health</b>.</p>	<p><a href="#">The potential risks from excess iodine (COT, 2017)</a></p>
Lead	<p>COT concluded that for young children, the <b>risk from dietary exposure alone is small</b> and there is no need for specific dietary advice relating to lead. However, when the possible contribution from soil and dust was taken into account, the possibility of adverse health effects cannot be excluded.</p>	<p><a href="#">Potential risks from lead in the infant diet (COT, 2013) and addendum (COT, 2016)</a></p>
Manganese	<p>COT was <b>unable to draw firm conclusions</b> on the potential effects of dietary exposure on the neurodevelopment of children aged 1 to 5 years because it was not possible to relate the adverse effects observed in humans to</p>	<p><a href="#">The health effects of manganese in the diets of infants and young children (COT, 2018)</a></p>

Chemical considered	Summary of COT conclusions	Web link
	<p>dietary exposures. Further data is required to refine this risk assessment, although any risk at current dietary exposures is likely to be low.</p>	
Methylmercury	<p>COT concluded that when taking into consideration the high degree of conservatism in the exposure modelling, there was <b>low risk to health</b> from the potential minor exceedance of the TWI in children limit of quantification. However, it would be prudent to maintain existing advice regarding consumption of large predator fish.</p>	<p><a href="#">Potential risks from methylmercury in the diet of infants and young children (COT, 2018)</a></p>
Nickel	<p>COT concluded that <b>chronic exposure to nickel from food was of no toxicological concern</b> to the long-term health of young children aged 1 to 5 years. Acute dietary exposure to nickel in sensitised individuals could trigger or exacerbate potentially unpleasant dermal effects.</p>	<p><a href="#">Potential risks from nickel in the diet of infants and young children (COT, 2018)</a></p>
Ochratoxin A (OTA)	<p>COT concluded that in young children consuming commercial foods for these age groups, exposures were well below the TWI and hence there was <b>no toxicological concern</b>.</p>	<p><a href="#">Potential risks from ochratoxin A (OTA) in the diet of infants and young children (COT, 2018)</a></p>
Perfluorooctanesulfonic (PFOS) acid and Perfluorooctanoic (PFOA) acid	<p>The EFSA panel had concluded that, for both compounds, exposures in a considerable proportion of the population exceed the proposed TWIs and these exceedances at the upper level of the estimates are of concern to human health. However, EFSA also noted that the present exposure assessment is highly uncertain as analytical methods are</p>	<p><a href="#">EFSA scientific opinion on risk to human health related to the presence of perfluorooctanesulfonic acid and perfluorooctanoic acid in food (EFSA, 2018)</a></p>

Chemical considered	Summary of COT conclusions	Web link
	<p>currently not sufficiently sensitive. Furthermore, it is unclear what impact processing has on exposure as well as the impact of co-exposure to multiple perfluoroalkyl substances (PFAS) on health-related outcomes. COT agreed with the uncertainties surrounding PFOS and PFOA and concluded that they would await EFSA's publication on PFAS<sup>1</sup>. The COT<sup>2</sup> have reviewed the EFSA scientific opinion on 'the risks to human health of perfluoroalkyl substances (PFAS) in food' published in 2020 and an updated COT statement will be published in due course.</p>	<p><a href="#">PFAS opinion for public consultation</a></p>
Phthalates	<p>EFSA (2019) establish a group TDI for DINP with DEHP, DBP, BBP in a low tier cumulative risk assessment, based on the reproductive effects and a plausible common mode of action. Exposures were below the TDI for European consumers of any age, including the most sensitive groups.</p> <p>COT considered it reasonable to group those 4 phthalates and that the group TDI and the relative potency factors were appropriate for DEHP, DBP and BBP. Furthermore, COT was content that the exposures estimated by EFSA did not indicate a health concern using the group TDI but noted that the uncertainty assessment in the draft opinion did not adequately reflect on the conclusions on DINP.</p>	<p><a href="#">EFSA draft scientific opinion on the risk assessment of DBP, BBP, DEHP, DINP and DIDP for use in food contact materials (EFSA, 2019)</a></p>
Plant-based drinks	<p>COT assessment on the potential risks posed by soya, oat and almond drinks consumed in the diets of infants and</p>	<p><a href="#">Overarching statement on consumption of plant-based drinks</a></p>

Chemical considered	Summary of COT conclusions	Web link
	young children concluded that neither the safety of these drinks, nor the suitability of the current guidance, could be confirmed from a toxicological perspective.	<a href="#">in children aged 6 months to 5 years (COT, 2021)</a>
Polybrominated biphenyls (PBBs)	COT concluded that, taking into account all of the uncertainties surrounding the exposure estimates, the contributions made by planar PBBs to the TDI for dioxin-like compounds were minor, and the large margins of exposure in the assessment of non-planar PBBs <b>did not indicate a cause for concern.</b>	<a href="#">Polybrominated biphenyls (PBBs) in the infant diet (COT, 2015)</a>
Polybrominated diphenyl ethers (PBDEs)	COT concluded that the <b>exposures from breast milk (12 to 18 months of age) and dust and soil (1 to 5 years) are of potential concern. Exposure from food was unlikely to be of concern.</b> However, given that PBDEs are no longer used commercially, the levels are therefore expected to decrease and are the sources of PBDEs for exposure in young children, the options for risk management are limited. <b>COT however recommended for monitoring to be continued to ensure levels are declining as expected.</b>	<a href="#">Potential risks from polybrominated diphenyl ethers (PBDEs) in the infant diet (COT, 2015) and addendum (2017)</a>
T-2 toxin, HT-2 toxin and neosolaniol	Whilst an effect on health cannot be entirely excluded at the 97.5 <sup>th</sup> percentile exposure, it is doubtful that children would be regularly exposed to these levels. Overall, COT therefore concluded that dietary exposure levels of T2, HT2 or NEO were <b>unlikely to be of any toxicological concern</b> in young children.	<a href="#">T-2 toxin, HT-2 toxin and neosolaniol in the diets of infants and young children (COT, 2018)</a>

<sup>1</sup> COT meeting minutes: 23rd March 2021

<sup>2</sup> COT meeting minutes: 4th May 2021

# Conclusions

- 10.5 COT assessed a number of chemicals and their potential risk from the diet of infants (aged 0 to 12 months) and young children (aged 1 to 5 years). The following paragraphs provide the conclusions for the latter age group; conclusions for infants aged 0 to 12 months can be found in the SACN report [‘Feeding in the first year of life’](#) (SACN, 2018).
- 10.6 COT refers to and confirms its previous evaluations for legacy chemicals, soya phytoestrogens, and vitamin A. As children aged 1 to 5 years would not be expected to be consuming high-caffeine beverages or alcohol, COT concluded that no further assessment for these 2 chemicals in this age group was required.
- 10.7 The additives regulation applies to all foods produced, including foods specifically for young children. Therefore, COT deemed it not necessary to assess food additives again in these age groups.
- 10.8 The data collected by the FSA on perchlorate and chlorate have been submitted to, and form part of, EFSA’s evaluations. While further data collection has been undertaken for chlorate, the data are unlikely to change the (UK) exposure assessment undertaken by EFSA or conclusions drawn from them. COT therefore did not consider it necessary to undertake a full risk assessment for either chemical itself. In agreement with EFSA, COT concluded that while there are considerable uncertainties in the assessment there is potential concern from dietary exposure to chlorate and perchlorate.
- 10.9 Given the limited UK-specific occurrence data, COT assessed 3-MCPD, its fatty acid esters and glycidol based on the latest EFSA evaluation. Overall, the committee agreed that some of EFSA’s MOE values for glycidol and exceedances of the TDI for 3-MPCD are of potential concern. However, as concluded by EFSA, the impacts of the uncertainties in these risk assessments for glycidol and 3-MCPD are high, for example uncertainty in the reference point used as a basis for the calculation of the MOE values for glycidol, and the long-term effects of 3-MCPD on the male reproductive system, as well as in the occurrence data.
- 10.10 There have been efforts to reduce concentrations of furan (and methylfurans) in food over recent years but the evidence so far is not sufficient to demonstrate whether there has been a decrease in dietary exposure. The exposures in COT’s assessment are of potential toxicological concern and efforts to reduce furan and methylfurans should therefore continue. However, there are numerous uncertainties in the assessment and COT acknowledges that its assessment is based on worst case assumptions.
- 10.11 For exposure of young children to acrylamide from infant formula and food, COT concluded that there was no cause for concern regarding neurotoxicity. Although human studies do not prove that acrylamide causes cancer, there is a potential

concern regarding carcinogenicity relating to exposures in this age group based on extrapolations from experimental studies.

- 10.12 Aflatoxin levels in all samples in the FSA's TDS survey were below their respective LOQ. However, given that aflatoxins are genotoxic and carcinogenic their presence in food is always undesirable and when exposure was estimated based on their LOQs, it was not possible to exclude a safety concern.
- 10.13 COT concluded that the total exposure to inorganic arsenic, from dietary and non-dietary sources, in young children aged 1 to 5 years was of potential concern to health.
- 10.14 Given the data gaps and limitations in the information for deoxynivalenol and its acetylated or modified forms, citrinin, patulin, manganese and tropane alkaloids, a potential health effect currently cannot be excluded.
- 10.15 For bisphenol A, dioxins, phthalates and perfluorooctanesulfonic acid perfluorooctanoic acid and perfluoroalkyl substances, COT decides to wait for EFSA's re-evaluation or is in the process of commenting on said publications. Following the assessment of EFSA's new opinion on dioxin and dioxin-like compounds, the committee has agreed to undertake its own review. A position statement has been published on the COT website.
- 10.16 Exposures to aluminium, cadmium, chromium, copper, iodine, lead, nickel, selenium, zinc, hexachlorocyclohexane, hexabromocyclododecane, methylmercury, ochratoxin A, polybrominated biphenyls, polycyclic aromatic hydrocarbons, tetrabromobisphenol, tropane alkaloids, T-2 toxin, HT-2 toxin, the 5 most common sweeteners in the UK (aspartame, acesulfame K, saccharine, sorbitol and xylitol, stevia and sucralose) and several mycotoxins (cyclopiazonic acid, diacetoxyscirpenol, ergot alkaloids, fumonisins, fusarenon-X, moniliformin, nivalenol, sterigmatocystin and zearalenone) are not of toxicological concern.
- 10.17 In 2021, a joint SACN-COT working group on plant-based drinks was established to conduct a benefit:risk assessment considering both nutritional and toxicological aspects associated with the consumption of plant-based drinks by the UK population. Further details on the working group and its assessment are available on the SACN website. Findings from this assessment will be considered by SACN and COT and the final evidence evaluation will be agreed by both committees ahead of publication (in line with the process outlines in the updated SACN (2023)).

# 11 Overall summary and conclusions

## Background

- 11.1 This report considered the scientific basis of current UK recommendations for feeding young children aged 1 to 5 years. This report forms part of a wider piece of work considering the evidence underpinning recommendations for feeding children up to 5 years of age, of which the first part, [‘Feeding in the first year of life’](#), was published in 2018. The report does not include a review of the evidence informing the Dietary Reference Values (DRVs) for children under 5 years of age; the existing DRVs have been used to assess the adequacy of the diets of children aged 1 to 5 years in the UK.
- 11.2 This report considered evidence obtained through literature searches for systematic reviews (SRs) and meta-analyses (MAs) examining the relationship between the diet of young children and later health outcomes. Most of the evidence identified was from observational (prospective cohort) or non-randomised studies of interventions.
- 11.3 This report also considered evidence on young child feeding in the UK from large national surveys, namely the 2011 Diet and Nutrition Survey of Infants and Young Children (DNSIYC) for children aged 12 to 18 months, and the National Diet and Nutrition Survey (NDNS) rolling programme (mainly from years 2016 to 2019) for children aged 18 to 60 months. Additional consideration was also given to data from UK national child measurement programmes and health surveys in relation to the prevalence of overweight and obesity among children aged 4 and 5 years as well as data from national dental health surveys in relation to the prevalence of dental caries in children up to 5 years of age.
- 11.4 The section below summarises findings from the surveys and evidence from SRs that was graded ‘adequate’, ‘moderate’, ‘limited’ or ‘inconsistent’. For evidence graded ‘insufficient’ see Annex 10, Table 10.36. The approach taken to assess the certainty of SR evidence is described in [Grading of the evidence from systematic reviews](#) chapter 2.
- 11.5 Throughout this summary, data are interpreted against the UK Dietary Reference Values (DRVs). DRVs describe the distribution of nutrient and energy requirements of different groups of people within the UK population; they are not recommendations for individuals. They comprise:

- Estimated average requirement (EAR): Estimated Average Requirement of a group of people for energy or protein or a vitamin or mineral. About half of a defined population will usually need more than the EAR, and half less.
- Reference nutrient intake (RNI): The average daily intake of a nutrient sufficient to meet the needs of almost all members (97.5%) of a healthy population. Values set may vary according to age, gender and physiological state
- Lower reference nutrient intake (LRNI): The estimated average daily intake of a nutrient which can be expected to meet the needs of only 2.5% of a healthy population. Values set may vary according to age, gender and physiological state.

## Overall summary of dietary survey data and systematic review evidence

- 11.6 This section summarises the findings on food and drink consumption, and nutrient intakes and status from DNSIYC and NDNS, as well as SR evidence that was graded ‘adequate’, ‘moderate’ and ‘limited’ (also see Table 11.1).

### Energy and macronutrients

#### Energy

- 11.7 Data from DNSIYC and NDNS indicated that 90% of children aged 12 to 24 months and 70% of children aged 24 to 35 months had reported energy intakes above the EAR. By age 36 to 47 months, approximately half of children had reported intakes above the EAR. By age 48 to 60 months less than half of children had reported intakes above the EAR.
- 11.8 There was ‘moderate’ evidence from SRs that larger portion sizes of snacks and meals provided in preschool settings are associated with higher food and energy intakes in the short term (less than 6 months).

#### Carbohydrates

- 11.9 The current UK government recommendation for total carbohydrate intake is that the population average intake should be approximately 50% of total dietary energy intake (TDEI). Data from DNSIYC and NDNS indicated that this was achieved in most age groups. Mean total carbohydrate intake contributed on average 49% TDEI in children aged 12 to 47 months and 51% TDEI in children aged 48 to 60 months.

- 11.10 The current UK government recommendation for free sugars is that the population average intake should not exceed 5% TDEI. This recommendation currently applies from age 2 years and above. Data from DNSIYC and NDNS indicated that mean intake of free sugars was double the maximum recommendation in children aged 12 to 47 months (at approximately 10% TDEI) and in children aged 48 to 60 months (at approximately 12% TDEI). The vast majority of children in all age groups ( $\geq 80\%$ ) had intakes above the 5% recommendation.
- 11.11 The DRV for dietary fibre for children aged 2 to 5 years is 15 grams per day. Data from NDNS indicated that mean dietary fibre intake was lower than recommended in children aged 18 to 47 months (at approximately 10 grams per day) and in children aged 48 to 60 months (at approximately 13 grams per day). The vast majority of children aged 18 to 60 months (88% and 72% of children aged 18 to 47 months and 48 to 60 months, respectively) had dietary fibre intakes below the DRV.
- 11.12 There was 'adequate' evidence from SRs that higher free sugars in children aged 1 to 5 years is associated with increased dental caries development (increment, incidence or prevalence) in childhood and adolescence.

## **Dietary fat**

- 11.13 The DRVs for dietary fat intake currently apply in full from age 5 years onwards, and do not apply before age 2 years. A flexible approach is currently recommended to the timing and extent of dietary change for individual children between the ages of 2 and 5 years.
- 11.14 The DRV for total dietary fat is that the population average intake should be no more than 33% TDEI. Data from DNSIYC and NDNS indicated that the mean intake of total dietary fat as a % TDEI was approximately 35% in children aged 12 to 47 months and approximately 34% in children aged 48 to 60 months. Although the DRV currently applies in full from age 5 years, and does not apply before age 2 years, it was notable that 69% of children aged 12 to 47 months had intakes above the DRV.
- 11.15 The DRV for saturated fat intake is that the population average intake should be no more than 10% TDEI. Data from DNSIYC and NDNS indicated that mean saturated fat intake was approximately 16% TDEI in children aged 12 to 18 months and approximately 15% in children aged 48 to 60 months. Although the DRV currently applies in full from age 5 years, and does not apply before age 2 years, it was notable that  $>90\%$  of children aged 12 to 60 months had intakes above the DRV.
- 11.16 There was 'limited' evidence from SRs of no association between total fat intake in children aged 1 to 5 years and change in BMI or body weight in the shorter term (1

to 3 years). The role of TDEI is uncertain in this relationship (see chapter 3, [background to macronutrients](#) for an explanation).

- 11.17 No additional evidence from SRs was identified on saturated fat intake and health outcomes since the SACN report '[Saturated Fats and Health](#)' (SACN, 2019). The SR evidence in children included in the '[Saturated Fats and Health](#)' report identified only 1 RCT that included children aged 1 to 5 years and findings from this study could not be disaggregated from those in older children.

## Protein

- 11.18 The reference nutrient intake (RNI) for protein is 14.5 grams per day for children aged 1 to 3 years and 19.7 grams per day for children aged 4 and 5 years. Data from DNSIYC and NDNS indicated that mean protein intake in children aged 12 to 18 months was 38 grams per day, more than twice the RNI, rising to 41 grams per day in children aged 18 to 47 months, which is close to 3 times the RNI for this age group. Children aged 48 to 60 months had a mean protein intake of approximately 46 grams per day, more than twice the RNI for this age group.
- 11.19 There was 'moderate' evidence from SRs that higher total protein intake in children aged 1 to 5 years is associated with higher BMI in childhood. The role of TDEI is uncertain in this relationship (see [Background to macronutrients](#) in chapter 3 for an explanation).
- 11.20 There was 'limited' evidence from SRs that higher animal protein intake in children aged 1 to 5 years is associated with earlier pubertal timing (menarche in girls or voice break in boys).

## Micronutrients

- 11.21 The target average salt intake (estimated from RNI values for sodium for infants and young children) is 2 grams per day for children aged 1 to 3 years and 3 grams per day for children aged 4 to 6 years. Seventy six percent of children aged 18 to 47 months had salt intakes above the target average salt intake for that age group. For children aged 48 to 60 months, 47% of children had salt intakes above the target average salt intake.
- 11.22 The RNI for iron is 6.9 mg per day for children aged 1 to 3 years and 6.1 mg per day for children aged 4 to 6 years. The RNI for zinc is 5.0 mg per day for children aged 7 months to 3 years and 6.5 mg per day for children aged 4 to 6 years. The RNI for vitamin A is 400 µg (retinol equivalents) per day for children aged 1 to 6 years.
- 11.23 NDNS data indicated that while mean intakes of iron, zinc and vitamin A were above the RNI for these micronutrients in almost all age groups, between 8% and

11% of children aged 18 to 47 months had intakes below the LRNI for iron, zinc and vitamin A; and 20% of children aged 48 to 60 months had intakes below the LRNI for zinc. These findings should be interpreted with caution as there was some evidence to suggest energy underreporting in children with intakes below the LRNI for these micronutrients.

- 11.24 Analyses of NDNS data (years 2008 to 2019 of the rolling programme) indicated that inadequate intakes of iron, zinc, vitamin A and vitamin D may be more prevalent among children from lower socioeconomic status households and certain ethnic groups (Asian or Asian British, and Black or Black British). Children with intakes below the LRNI did not obtain any vitamin A from dietary supplements. The UK government recommends that all children aged 6 months to 5 years should be given a vitamin supplement containing vitamin A.
- 11.25 Despite NDNS data indicating that mean intakes of vitamin A were above the RNI in all age groups, the potential risks from intakes at these levels are unlikely to be a cause for concern (see chapter 4, paragraphs 4.177 and 4.178).
- 11.26 Data from DNSIYC and NDNS indicated that although 11% of children aged 12 to 18 months and over 24% of children aged 18 to 60 months had iron deficiency, less than 4% of children in all age groups had iron deficiency anaemia. It should be noted that there are uncertainties in the iron DRVs for children.
- 11.27 NDNS data indicated that 7% of children aged 18 to 47 months had plasma retinol concentrations between 0.35  $\mu\text{mol/L}$  and 0.70  $\mu\text{mol/L}$ , the range associated with mild vitamin A deficiency in adults (there is no equivalent threshold in children).
- 11.28 The RNI for vitamin D for children aged 1 to 5 years is 10 $\mu\text{g}$  (400 IU) per day. Data from DNSIYC and NDNS indicated that the mean vitamin D intake of children aged 12 to 18 months was 55% of the RNI and around 40% in children aged 18 to 60 months. Data from NDNS indicated that only 25% of children aged 18 to 36 months took a vitamin D supplement. The UK government recommends that children aged 1 to 5 years should be given a daily supplement containing 10 $\mu\text{g}$  vitamin D.
- 11.29 Analysis of NDNS data (years 2012 to 2017) for children aged 18 to 36 months indicated that vitamin D intakes decreased with increasing deprivation (as measured by equivalised household income). Moreover, although the sample size was too small to draw firm conclusions, NDNS data (years 2008 to 2019) indicated that, compared with white children, young children from other ethnic groups were likely to be at higher risk of vitamin D deficiency.
- 11.30 NDNS data indicated that 9% of children aged 18 to 47 months had serum 25(OH)D concentrations below 25 nmols/l which is the threshold for increased risk of rickets and osteomalacia. Analysis of NDNS data (years 2012 to 2017) for children aged 18 to 36 months indicated that serum 25(OH)D concentrations

decreased with increasing deprivation (as measured by equivalised household income).

- 11.31 The RNI for vitamin C for children aged 1 to 5 years is 30mg per day. Data from DNSIYC and NDNS indicated that vitamin C intakes in children aged 12 to 60 months were adequate and no children had intakes below the LRNI. There was also no apparent relationship between vitamin C intakes and deprivation (assessed by the Index of Multiple Deprivation for England).
- 11.32 The latest available data (January 2023) indicated that uptake of Healthy Start vitamins containing vitamins A, C and D by local authority ranged from 46% to 80% (median 62%) in England; 58% to 73% (median 66%) in Wales; and 49% to 56% (median 54%) in Northern Ireland.
- 11.33 There was 'limited' evidence from SRs that fortification with iron and other micronutrients (including zinc, vitamin A and vitamin C) of milk, or micronutrient sprinkles reduces the prevalence of anaemia in children aged 6 to 36 months.
- 11.34 There was 'limited' evidence from SRs that vitamin D fortification of milk or formula milk improves vitamin D status or decreases the risk of vitamin D deficiency in children aged 1 to 5 years.

## **Foods**

### **Vegetables and fruit**

- 11.35 There are currently no UK government recommendations on portion sizes for vegetables and fruit for young children. However, it is recommended that from about 6 months of age, gradual diversification of the diet to provide increasing amounts of vegetables and fruit is encouraged.
- 11.36 Data from DNSIYC and NDNS indicated that nearly all children in all age groups consumed vegetables or fruit or both over the 4 day survey period.
- 11.37 Data from DNSIYC indicated that children aged 12 to 18 months consumed, on average, 170 grams per day of vegetables (excluding potatoes) and fruit (excluding fruit juice). Data from NDNS indicated that for children aged 18 to 47 months, and aged 48 to 60 months, mean daily consumption was 178 grams and 217 grams, respectively. In all age groups fruit made a greater contribution to intakes than vegetables.
- 11.38 Time trend analysis of NDNS data indicated that for children aged 18 to 36 months, there was a significant reduction in vegetable consumption over a 9-year period (2008 to 2017) while fruit consumption remained broadly unchanged (analyses were not performed for other age groups).

- 11.39 Consumption of total vegetables and fruit in children aged 18 to 60 months decreased with increasing deprivation (as assessed by the Index of Multiple Deprivation, England).

### **Dairy products**

- 11.40 Data from DNSIYC and NDNS indicated that nearly all children aged 12 to 60 months consumed dairy products over the 4 day survey period. Dairy products (excluding formula milks) contributed approximately 27% TDEI, 22% TDEI and 15% TDEI in children aged 12 to 18 months, 18 to 47 months, and 48 to 60 months, respectively. Of the main dairy products examined, cows' milk and other dairy milks were the largest contributors to TDEI in children aged 12 to 60 months.

### **Foods rich in starchy carbohydrates**

- 11.41 Data from DNSIYC and NDNS indicated that nearly all children aged 12 to 60 months consumed foods rich in starchy carbohydrates over the 4 day survey period. Foods rich in starchy carbohydrates contributed 17% TDEI in children aged 12 to 18 months, and over 20% TDEI in children aged 18 to 60 months. Of the main sources of starchy carbohydrates examined, bread made the largest contribution to TDEI followed by breakfast cereals (with a total sugars content less than 22.5 grams per 100g) in children aged 12 to 60 months.

### **Non-dairy sources of protein**

- 11.42 Data from DNSIYC and NDNS indicated that nearly all children aged 12 to 60 months consumed non-dairy sources of protein. Non-dairy sources of protein contributed approximately 11% TDEI, 15% TDEI and 16% TDEI in children aged 12 to 18 months, 18 to 47 months and 48 to 60 months, respectively. Of the main non-dairy sources of protein examined, processed and unprocessed meat were the largest contributors in all age groups and the contribution of processed meat was higher in the older age groups.

### **Foods that are energy dense and high in saturated fat, salt or free sugars**

- 11.43 Data from DNSIYC and NDNS indicated that nearly all children aged 12 to 60 months consumed foods that are energy dense and high in saturated fat, salt or free sugars.
- 11.44 Based on the food groups examined, these foods contributed approximately 16% TDEI, 24% TDEI and 30% TDEI in children aged 12 to 18 months, 18 to 47 months and 48 to 60 months, respectively. Of the foods that were examined, biscuits, buns, cakes and pastries was the largest food group contributor to TDEI.

## **Commercially manufactured foods and drinks specifically marketed for infants and young children**

- 11.45 A PHE evidence review (2019) found that the nutrient composition of many commercially manufactured foods and drinks specifically marketed for infants and young children was inconsistent with UK dietary recommendations for this age group. Some products available in the UK had added sugar or salt or contained ingredients that are high in sugar or salt; this was particularly common with finger foods. The review found that, at age 1 year, sweet finger foods provided more energy than is recommended for snack occasions across the day.
- 11.46 The PHE review also highlighted that commercially manufactured finger foods have been the main driver in the growth of the infant food market in recent years.
- 11.47 Data from DNSIYC and NDNS indicated that 65% of children aged 12 to 18 months consumed commercially manufactured foods and drinks specifically marketed for infants and young children, while 20% of children aged 18 to 47 months consumed these products. Among consumers, these products were sizeable contributors to intakes of energy and free sugars.
- 11.48 In children aged 12 to 18 months and those aged 18 to 47 months, consumers of these products obtained 10% TDEI and approximately 5% TDEI, respectively, from these products.
- 11.49 Among consumers aged 12 to 18 months, these products provided 20% of free sugars intake, while for consumers aged 18 to 47 months, these products provided 12% of free sugars intake.

## **Dietary patterns**

- 11.50 There was 'limited' evidence from SRs that dietary patterns classified as 'unhealthy' are associated with higher body fat measures in children aged 1 to 5 years.

## **Drinks**

### **Breastfeeding beyond the first year of life**

- 11.51 Of the 4 UK countries, only Scotland and Northern Ireland collect breastfeeding data into the second year of life. Data from Public Health Scotland for 2021 to 2022 indicated that 22% of children aged 13 to 15 months were still receiving breast milk. Data from the Northern Ireland Public Health Agency indicated that 11.2% of children born in 2020 were still receiving breast milk at age 12 months.

- 11.52 There was 'limited' evidence from SRs that breastfeeding for 12 months or longer was not associated with development of early childhood caries compared with breastfeeding for less than 12 months.
- 11.53 There was 'moderate' evidence from SRs that breastfeeding beyond 12 months protects against the development of malocclusion.

## **Formula milks**

- 11.54 Data from DNSIYC and NDNS indicated that formula milks (mainly follow-on formula and milks marketed to children over the age of 1 year, also known as 'toddler milks' and 'growing-up milks') were consumed by 36% of children aged 12 to 18 months, and 7% of children aged 18 to 47 months. There were no consumers of formula milks in the 48 to 60 month age group.
- 11.55 For children aged 12 to 18 months, consumers of formula milks (36% of this age group) obtained approximately 50% of their free sugars intake from these products.

## **Milk**

- 11.56 Data from DNSIYC and NDNS indicated that nearly all children consumed milk (which includes all types of cows' milk and other dairy milks) over the 4 day survey period.
- 11.57 Milk contributed approximately a fifth of TDEI in children aged 12 to 47 months and approximately 15% in the 48 to 60 month age group.
- 11.58 Substitution analysis using data from DNSIYC indicated that replacing whole cows' milk with semi-skimmed cows' milk for children aged 12 to 18 months would be unlikely to have a detrimental effect on nutrient intakes at the population level. By contrast, replacing whole milk with skimmed or 1% cows' milk may result in a greater risk of inadequate intakes of vitamin A in young children.
- 11.59 There was 'limited' evidence from SRs of no association between total milk intake in children aged 1 to 5 years and BMI in childhood.

## **Fruit juice**

- 11.60 Data from DNSIYC and NDNS indicated that 26% of children aged 12 to 18 months and over 40% of children aged 18 to 60 months consumed fruit juice (100% fruit juice and smoothies) over the 4 day survey period.
- 11.61 Fruit juice (100% fruit juice and smoothies) contributed between 2% TDEI in consumers aged 12 to 18 months and approximately 3% TDEI in consumers aged 18 to 60 months.

- 11.62 Fruit juice (100% fruit juice and smoothies) contributed 5%, 11% and 7% to free sugars intakes in children aged 12 to 18 months, 18 to 47 months and 48 to 60 months, respectively, at the population level.
- 11.63 There was 'limited' evidence from SRs that higher fruit juice consumption in children aged 1 to 5 years is associated with increased BMI in childhood when unadjusted for TDEI, and 'limited' evidence that fruit juice consumption in children aged 1 to 5 years is not associated with BMI in childhood, following adjustment for TDEI (see chapter 6, paragraph 6.96).

## **Sugar-sweetened beverages**

- 11.64 Data from DNSIYC and NDNS indicated that 26% of children aged 12 to 18 months and over 20% of children aged 18 to 60 months consumed SSBs over the 4 day survey period.
- 11.65 SSBs contributed 1.6% TDEI in consumers aged 12 to 18 months and 1.7% TDEI in consumers aged 18 to 47 months. SSBs contributed 0.5% TDEI at a population level in children aged 48 to 60 months (data were insufficient to present for consumers only in this age group).
- 11.66 SSBs contributed approximately 3% and 4%, respectively, to free sugars intakes in children aged 12 to 18 months and 18 to 60 months at a population level.
- 11.67 Time trend analysis of NDNS data (years 2008 to 2017) in children aged 18 to 36 months indicated a decrease in the percentage of consumers of SSBs for the 9-year period. No time trend data were available for the other age groups.
- 11.68 There was 'adequate' evidence from SRs that higher SSB consumption in children aged 1 to 5 years is associated with a greater odds of overweight or obesity in childhood compared with lower SSB consumption, adjusted for TDEI.
- 11.69 There was 'moderate' evidence that higher SSB consumption in children aged 1 to 5 years is associated with a greater increase in BMI (or BMI z-score or weight-for-height z-score) in childhood and adolescence compared with lower SSB consumption, unadjusted for TDEI (see chapter 3, paragraph 3.50).
- 11.70 There was 'limited' evidence from SRs that higher SSB consumption in children aged 1 to 5 years is associated with increased early childhood caries.

## **Eating and feeding behaviours**

- 11.71 There was 'moderate' evidence from SRs that feeding practices (including repeated exposure and pairing vegetables with positive stimuli) increases children's vegetable consumption in the short term (up to 8 months).

- 11.72 There was ‘moderate’ evidence from SRs that repeated taste exposure (around 8 to 10 times) to a vegetable is the most effective feeding practice at increasing vegetable consumption in children aged up to 5 years in the short term (less than 8 months).
- 11.73 There was ‘moderate’ evidence from SRs that repeated taste exposure to vegetables paired with liked foods or additional flavours or energy increases vegetable consumption, although this strategy may be less effective in increasing vegetable consumption than repeated taste exposure to vegetables in their plain form.
- 11.74 There was ‘inconsistent’ evidence from SRs on the effect of adult modelling of food consumption (including vegetables and fruit) on children’s food acceptance or consumption in the short term.

## **Excess weight and obesity**

- 11.75 Data from child measurement programmes in England and Scotland for the collection year 2021 to 2022 indicated that the prevalence of overweight and obesity combined in children aged 4 to 5 years was 22.3% and 24.1%, respectively. The prevalence of obesity in England and Scotland (at 10.1% and 11.7%, respectively) decreased from that in the collection year 2020 to 2021 when measurements were taken during the beginning of the COVID-19 pandemic but remained higher than before the pandemic. In Wales, limited data from the collection year 2020 to 2021 also indicated that the prevalence of obesity (approximately 18%) had increased compared with the pre-pandemic collection year 2018 to 2019 (no comparable data are available for Northern Ireland). Data from these measurement programmes also indicated that deprivation is a major risk factor for obesity in childhood, while increased BMI in early childhood is a strong predictor of obesity in later childhood.
- 11.76 There was ‘limited’ evidence from SRs that adiposity rebound occurring before age 5 years is associated with a higher BMI or risk of obesity in adulthood.
- 11.77 There was ‘adequate’ evidence from SRs that higher child BMI or weight status at age 1 to 5 years is associated with higher adult BMI or risk of adult overweight or obesity.
- 11.78 There was ‘moderate’ evidence from SRs of no association between child BMI at age 6 years and under and incidence of coronary heart disease in adulthood.
- 11.79 There was ‘moderate’ evidence from SRs of no association between child BMI at age 6 years and under and incidence of stroke in adulthood.

## Oral health

- 11.80 Dental caries in children remains a major public health problem. The latest available survey data indicated that 11% of children aged 3 years and 23% of children aged 5 years in England experienced obvious tooth decay. In Scotland, 27% of children aged 5 years had obvious tooth decay, while in Wales and Northern Ireland, the figures were 34% and 40%, respectively. Almost 9 out of 10 hospital tooth extractions among children aged 0 to 5 years are due to preventable tooth decay and tooth extraction is still the most common hospital procedure in children aged 6 to 10 years.
- 11.81 There was 'adequate' evidence from SRs that higher intake of free sugars in children aged 1 to 5 years is associated with increased dental caries (increment, incidence or prevalence) in childhood and adolescence.
- 11.82 There was 'limited' evidence from SRs that higher SSB consumption in children aged 1 to 5 years is associated with increased early childhood caries (incidence, prevalence or experience).
- 11.83 There was 'limited' evidence from SRs that breastfeeding for 12 months or longer was not associated with development of early childhood caries compared with breastfeeding for less than 12 months.
- 11.84 There was 'moderate' evidence from SRs that breastfeeding beyond 12 months protects against the development of malocclusion.

**Table 11.1 Systematic review evidence in children aged 1 to 5 years graded 'moderate' or 'adequate'**

Topic area	Systematic review finding	Certainty of evidence
Energy	Larger portion sizes of snacks and meals provided in preschool settings are associated with higher food and energy intakes in the short term (less than 6 months)	Moderate
Macronutrients	Higher total protein intake in children aged 1 to 5 years is associated with higher body mass index (BMI) in childhood	Moderate
Macronutrients	Higher free sugars intake is associated with increased dental caries (increment, incidence or prevalence) in childhood and adolescence	Adequate
Drinks	Higher sugar-sweetened beverage (SSB) consumption in children aged 1 to 5 years is associated with greater odds of overweight or obesity in childhood  Higher SSB consumption in children aged 1 to 5 years is associated with a greater increase in BMI in childhood and adolescence	Adequate  Moderate
Eating and feeding behaviours	Feeding practices (including repeated taste exposure, pairing with positive stimuli such as liked foods, modelling of vegetable consumption and offering the child non-food rewards) increase vegetable consumption in children aged 1 to 5 years (in the short term, up to 8 months)	Moderate
Eating and feeding behaviours	Repeated taste exposure to vegetables increases vegetable consumption in children aged 1 to 5 years (in the short term, up to 8 months)	Moderate
Excess weight and obesity	Higher child BMI or weight status at age 1 to 5 years is associated with higher adult BMI or risk of overweight or obesity	Adequate
Excess weight and obesity	Child BMI at age 6 years and under is not associated with incidence of coronary heart disease in adulthood	Moderate
Excess weight and obesity	Child BMI at age 6 years and under is not associated with incidence of stroke in adulthood	Moderate
Oral health	Breastfeeding beyond 12 months is associated with lower odds of malocclusion (teeth that are not aligned correctly)	Moderate

## Overall conclusions

- 11.85 In 1994, the Committee on Medical Aspects of Food and Nutrition Policy (COMA) published its report '[Weaning and the weaning diet](#)' and, since then, has been the basis for much of the advice on feeding young children in the UK.
- 11.86 The current diet of young children in the UK, as captured in both the Diet and Nutrition Survey in Infants and Young Children (DNSIYC) and the National Diet and Nutrition Survey (NDNS) does not meet current dietary recommendations for several nutrients.
- 11.87 The following conclusions are informed by the main findings from DNSIYC and NDNS and SR evidence that was graded 'adequate' and 'moderate' (Table 11.1).

## Energy and macronutrients

- 11.88 Evidence from DNSIYC and NDNS indicated that
- mean intakes of energy for children aged 12 to 35 months were above the EAR
  - mean intakes of free sugars for children aged 18 to 60 months were above the current recommendation of no more than 5% TDEI
  - mean intakes of dietary fibre for children aged 18 to 60 months were below the recommended intake of 15 grams per day
  - mean intakes of saturated fats were above the current recommendation of no more than 10% TDEI (which applies in full from age 5 years)
  - mean intakes of protein were above the RNI.
- 11.89 Evidence identified from SRs indicates that:
- larger portion sizes of snacks and meals provided in preschool settings are associated with higher food and energy intakes in the short term (less than 6 months)
  - higher free sugars intake in children aged 1 to 5 years is associated with increased dental caries (increment, incidence or prevalence) in childhood and adolescence
  - higher total protein intake in children aged 1 to 5 years is associated with higher BMI in childhood
  - higher childhood BMI is associated with higher risk of adult overweight or obesity.
- 11.90 These findings are of concern in relation to wider evidence on
- the high prevalence of overweight and obesity in childhood in the UK particularly in lower socioeconomic groups and in some ethnic groups

- the high prevalence of dental caries in children in the UK.

## **Micronutrients**

- 11.91 Evidence from DNSIYC and NDNS indicated that mean salt intake was above the target average salt intake in children aged 18 to 47 months, where 76% of children in this age group had intakes above the target salt intake.
- 11.92 Evidence from DNSIYC and NDNS indicated that certain groups of children, including children from lower socioeconomic status households (as measured by the Index of Multiple Deprivation) and some ethnic groups, may be at risk of inadequate intakes of iron, zinc, vitamin A, and vitamin D, and low vitamin D status. Conversely, intakes of vitamin C exceeded the RNI across all age groups.
- 11.93 Evidence from NDNS indicated that use of vitamin D supplements in the general population of children aged 1 to 5 years was low (no comparable data were available for supplements containing vitamin A or C); while the latest available data indicated variable uptake of Healthy Start vitamins (containing vitamins A, C and D).

## **Foods**

### **Vegetables and fruit**

- 11.94 Currently there are no UK government recommendations on portion sizes for vegetables and fruit for young children. Evidence from NDNS indicated that children ate more fruit than vegetables. Consumption of total vegetables and fruit decreased with increasing deprivation. Encouraging consumption of vegetables as children grow and develop more independence around food is important to support children to meet population dietary recommendations.
- 11.95 Evidence identified from SRs indicated that repeated taste exposure to a vegetable (around 8 to 10 times) can increase consumption of that vegetable in the short term (less than 8 months). No SR evidence was identified on the efficacy of this feeding practice in increasing vegetable consumption in the longer term.

### **Dairy products**

- 11.96 Evidence from DNSIYC indicated that the food group (sugar-sweetened) 'yoghurts, fromage frais and dairy desserts' was among the top contributors to free sugars intake in children aged 1 to 1.5 years, providing 18% of free sugars intake at a population level.

## **Foods that are energy dense and high in saturated fat, salt or free sugars**

- 11.97 Evidence from NDNS indicated that foods that are energy dense and high in saturated fat, salt or free sugars contributed approximately 16% TDEI, 24% TDEI and 30% TDEI in children aged 12 to 18 months, 18 to 47 months and 48 to 60 months, respectively. Of these, biscuits, buns, cakes and pastries were the largest contributor to TDEI.

## **Commercially manufactured foods and drinks marketed specifically for infants and young children**

- 11.98 Evidence from DNSIYC indicated that among children aged 12 to 18 months who consumed commercially manufactured foods and drinks marketed specifically for infants and young children (65% of this age group), these products provided approximately 20% of free sugars intakes.
- 11.99 A PHE evidence review (2019) found that the nutrient composition of many of these products was inconsistent with UK dietary recommendations for this age group, particularly for sugar and salt. The PHE review highlighted that commercially manufactured finger foods have been the main driver in the growth of the infant food market in recent years.

## **Drinks**

- 11.100 Evidence from DNSIYC and NDNS indicated that
- formula milks (mainly follow-on formula and milks marketed for children over the age of 1 year, also known as ‘toddler milks’ and ‘growing-up milks’) were consumed by 36% of children aged 12 to 18 months and contributed 50% of free sugars intake in consumers (18% of free sugars intake at a population level)
  - fruit juice (100% fruit juice and smoothies) contributed nearly 11% to free sugars intake in children aged 18 to 47 months and less than 10% in the other age groups at a population level
- 11.101 Substitution analysis using data from DNSIYC indicated that replacing whole cows’ milk with semi-skimmed cows’ milk for children aged 12 to 18 months would be unlikely to have a detrimental effect on nutrient intakes at the population level. By contrast, replacing whole milk with skimmed or 1% milk may result in a greater risk of inadequate intakes of vitamin A in young children.
- 11.102 Evidence identified from SRs indicated that higher sugar-sweetened beverage (SSB) consumption in children aged 1 to 5 years is associated with a greater odds of overweight or obesity in childhood.

- 11.103 Evidence identified from SRs indicated that continued breastfeeding beyond the age of 1 year is protective against malocclusion (teeth that are not correctly aligned).

## **Risks of chemical toxicity**

- 11.104 The Committee on Toxicology of Chemicals in Food, Consumer Products and the Environment (COT) assessed toxicity issues from the infant diet for a number of nutrients, substances and contaminants in breast milk, infant formula and solid foods. They concluded there were unlikely to be concerns over toxicity in the diet of young children for substances considered at current levels of exposure. Issues where COT has identified that there is potential concern are described in chapter 10.
- 11.105 Nutritional and toxicological aspects associated with the consumption of plant-based drinks by children aged 1 to 5 years are being considered in a joint benefit:risk assessment being undertaken by SACN and COT. Findings are expected to be published in 2024 and will include recommendations on plant-based drink consumption. More information on the work of the joint SACN-COT working group is available [here](#).
- 11.106 SACN's Feeding in the first year of life report (2018) considered findings from a benefit:risk assessment on timing of the introduction of peanut and hen's egg into the infant diet and the risk of developing allergy to these foods. The available evidence indicated that the deliberate exclusion or delayed introduction of peanut or hen's egg beyond 6 to 12 months of age may increase the risk of allergy to the same foods. These findings will have a bearing on children in the older age group (1 to 5 years).

## **General limitations in the evidence base**

- 11.107 A range of limitations was identified in the evidence base provided by SRs and dietary surveys. These are summarised below.

### **General limitations of the systematic review evidence**

- 11.108 There was either no or insufficient SR evidence for a number of dietary exposures (including saturated fat and dietary fibre) and health outcomes (including paediatric cancers, allergy and autoimmune diseases, and bone and skeletal health) which were included in the scope and literature search for this risk assessment.
- 11.109 Many of the SRs identified for this report had a broad search strategy that included population groups outside the age range of interest for this report (children aged 1 to 5 years) and it was difficult to determine whether their search strategy for the target population was comprehensive.

- 11.110 Most of the SR evidence that was specific to children aged 1 to 5 years was observational (from prospective cohort studies) or from non-randomised studies of interventions and may have been subject to confounding and selection bias.
- 11.111 The evidence base on many topic areas was highly heterogeneous in terms of exposures, dietary assessment methods, outcome measures, populations, settings, and study designs, which prevented the pooling of results by MA or other methods of quantitative synthesis.
- 11.112 Due to the lack of quantitative syntheses in the included SRs, risk of publication bias was seldom formally assessed.
- 11.113 The SR evidence identified on micronutrients was drawn almost exclusively from supplementation and food fortification trials designed for populations in low income, lower-middle or upper-middle income countries (defined according to the World Bank classification system) and therefore may not be generalisable to children living in the UK.
- 11.114 Primary studies, particularly those conducted in high income countries, seldom considered whether the impact of dietary exposures on nutritional status (for example, vitamin D) or health outcomes differed among different ethnic groups.
- 11.115 The majority of primary studies had short follow-up periods, limiting the ability to draw conclusions about the longer-term health effects of nutrient or dietary intake in children aged 1 to 5 years.

### **General limitations of the evidence from dietary surveys**

- 11.116 DNSIYC was conducted in 2011. Dietary patterns may have changed significantly in the period since the data were collected.
- 11.117 The number of children that provided blood samples for status measures in NDNS was small and may not be representative of the wider population. Children who gave a blood sample were more likely to come from higher socioeconomic status households.
- 11.118 Misreporting of food consumption, specifically underreporting, and therefore underestimation of total dietary energy intake (TDEI) (known as underreporting) in self-reported dietary methods is a well-documented source of bias and is an important consideration when interpreting survey data.

# 12 Recommendations

- 12.1 The following recommendations are suitable for children aged 1 to 5 years who are able to consume a varied diet and are growing appropriately for their age.
- 12.2 Between 1 to 2 years of age, children's diets should continue to be gradually diversified in relation to foods, dietary flavours and textures. A flexible approach is recommended to the timing and extent of dietary diversification, taking into account the variability between young children in developmental attainment and the need to satisfy their individual nutritional requirements. [SACN 2023, SACN 2018]
- 12.3 Current UK dietary recommendations as depicted in the Eatwell Guide should apply from around age 2 years [SACN 2023], with the following exceptions:
- UK dietary recommendations on average intake of free sugars (that free sugars intake should not exceed 5% of total dietary energy intake) should apply from age 1 year [SACN 2023]
  - milk or water, in addition to breast milk, should constitute the majority of drinks given to children aged 1 to 5 years [SACN 2023]
  - pasteurised whole and semi-skimmed cows' milk can be given as a main drink from age 1 year [SACN 2023], as can goats' and sheep's milks [SACN 2023, COMA 1994].
  - pasteurised skimmed and 1% cows' milk should not be given as a main drink until 5 years of age. These lower fat milks can be used in cooking. [SACN 2023, COMA 1994]
  - children aged 1 to 5 years should not be given rice drinks as they may contain too much arsenic [SACN 2023 endorses COT 2016, 2021]
  - children aged 1 to 5 years should not be given sugar-sweetened beverages [SACN 2023]
  - dairy products (such as yoghurts and fromage frais) given to children aged 1 to 5 years should ideally be unsweetened. [SACN 2023, COMA 1994]
- 12.4 Formula milks (including infant formula, follow-on formula, 'growing-up' or other 'toddler' milks) are not required by children aged 1 to 5 years. [SACN 2023 endorses WHO 2013]. Specialised formula, including low-allergy formula, are also usually not required after the first year of life. [SACN 2023]
- 12.5 Foods (including snacks) that are energy dense and high in saturated fat, salt or free sugars should be limited in children aged 1 to 5 years in line with current UK dietary recommendations. [SACN 2023]

- 12.6 Commercially manufactured foods and drinks marketed specifically for infants and young children are not needed to meet nutritional requirements. [SACN 2023]
- 12.7 Salt should not be added to foods given to children aged 1 to 5 years. Children aged 1 to 3 years should, on average, aim to have no more than 2g of salt per day; the figure for children aged 4 to 6 years is 3g per day. [SACN 2023, SACN 2003]
- 12.8 Children aged 1 to 5 years should be presented with unfamiliar vegetables on multiple occasions (as many as 8 to 10 times or more for each vegetable) to help develop and support their regular consumption. [SACN 2023]
- 12.9 Deliberate exclusion of peanut or hen's egg (and foods containing these) beyond 12 months of age may increase the risk of allergy to the same foods. Importantly, once introduced, these foods should continue to be consumed as part of the child's usual diet in order to minimise the risk of allergy to peanut or hen's egg developing after initial exposure. [SACN 2023, SACN-COT 2018]
- 12.10 Children aged 1 to 5 years should continue to be offered a wide range of foods that are good sources of iron. They do not require iron supplements unless advised by a health professional. [SACN 2023, SACN 2018]
- 12.11 Children aged 1 to 5 years should be given a daily supplement of 10µg (400 IU) vitamin D and 233µg vitamin A unless, contrary to recommendations, they are consuming more than 500ml of formula milk per day (see above). [SACN 2023, SACN 2016, COMA 1994]
- 12.12 Vitamin C supplements are not necessary for the general population. However, there is no evidence that taking vitamin C supplements at the current recommended level of supplementation has any adverse effects. [SACN 2023]
- 12.13 It is recommended that government considers a range of strategies and actions to improve the diets of children aged 1 to 5 years, and continues to monitor dietary intakes, and the nutritional, weight and oral health status of young children as outlined below.
- 12.14 Consider strategies to support and promote:
- continuation of breastfeeding into the second year of life [SACN 2023]
  - current UK dietary recommendations to children aged 1 to 5 years [SACN 2023]
  - feeding of an appropriate and diverse diet to children aged 1 to 5 years that meets nutritional requirements but does not exceed energy requirements [SACN 2023]
  - awareness and uptake of current advice on vitamins D and A supplements at the current recommended levels in children aged 1 to 5 years, particularly in at-risk groups such as children from some ethnic groups and lower socioeconomic status households [SACN 2023]

- good oral health in children aged 1 to 5 years [SACN 2023]

12.15 Consider strategies to reduce consumption of:

- free sugars and excess protein in children aged 1 to 5 years [SACN 2023]
- foods (including snacks) that are energy dense and high in saturated fat, salt or free sugars in children aged 1 to 5 years, while encouraging uptake of healthier snacks [SACN 2023]
- sugar-sweetened beverages in children aged 1 to 5 years [SACN 2023]

12.16 Actions for consideration:

- develop and communicate age-appropriate portion sizes for food and drinks, including for vegetables, fruit, fruit juice and milk, for children aged 1 to 5 years [SACN 2023]
- review advice on the need for vitamin C supplements for children aged 1 to 5 years [SACN 2023]
- support parents or caregivers of children aged 1 to 5 years following vegetarian, vegan and plant-based diets to ensure the nutritional requirements (including for iron, iodine, calcium and vitamin B12) of their children are met [SACN 2023]

12.17 Monitoring of children aged 1 to 5 years for consideration:

- collect detailed, nationally representative data on nutrient intakes and status [SACN 2023]
- collect detailed data on nutrient intake and status of population subgroups, including ethnically diverse populations and socially disadvantaged groups, [SACN 2023]
- monitor the nutritional impact of a population shift towards adopting vegetarian, vegan and plant-based diets [SACN 2023]
- continue to monitor the prevalence of both overweight and obesity and the extent of excess energy intakes [SACN 2023]
- continue to monitor the oral health [SACN 2023]
- monitor intakes of low or no calorie sweeteners [SACN 2023]

# 13 Research recommendations

- 13.1 A number of gaps in the evidence were identified during the development of this report. Areas recommended for future research are summarised below. In addition, a number of limitations in study design were identified. Future research should adhere to the overarching principles outlined in Box 13.1.
- 13.2 Consideration of the potential short- and long-term health effects in young children of consuming:
- fruit juice, and formula milks marketed for children over the age of 1 year in relation to free sugars intakes
  - commercially manufactured foods and drinks specifically marketed for infants and young children
  - low or no calorie sweeteners
  - saturated fat, mono and polyunsaturated fats
  - dietary fibre
  - animal compared with vegetable protein.
- 13.3 Consideration of the potential short- and long-term health effects of vegetarian and vegan diets, and plant-based foods, drinks and diets in young children.
- 13.4 Consideration of the impact of suboptimal micronutrient intakes and status (including iron, vitamin A and vitamin D) on growth, and developmental and health outcomes (including oral health) of young children.
- 13.5 Consideration of the developmental and cultural factors associated with the acceptance and consumption of healthier foods in order to inform interventions to influence young children's eating behaviours.
- 13.6 Consideration of whether the way foods and drinks are presented to young children (for example, the use of straws or pouches with nozzles) has an impact on energy intake, oral health and developmental attainment.

### **Box 13.1. Overarching principles in conducting and reporting research in feeding young children**

For all future nutrition research:

- conduct research in accordance with best practice principles/guidance such as the [CONSORT](#) checklist for reporting a randomised trial and [PRISMA](#) checklist for a systematic review. For example, adopt transparent reporting practices by registering study or review protocols, statistical analysis plans and sources of funding on an open access research registration platform such as PROSPERO or Open Science Framework at the outset, ensure that any subsequent amendments to protocols and analysis plans are declared, and include a data access statement.
- increase representation of different ethnic groups within study populations, including, where necessary, undertaking focused research in specific population groups
- report power calculations to inform the robustness of null effects in intervention or association studies
- collect data using validated methods with sufficient frequency to capture the timing of events of interest accurately and precisely, given the transitional nature of young children's diets
- standardise methodology for assessing diet quality in young children in the UK

For observational research:

- minimise the risk of reverse causation by employing prospective data collection and adjusting outcomes for key baseline values
- evaluate and control for potential confounding factors to accurately identify the impact of diet, feeding practices and eating behaviours at this crucial stage of development
- exercise considerable care when making statistical adjustment, to ensure that the strength and complexity of sociodemographic confounding of young child feeding are fully accounted for
- include appropriate comparative groups (for example, children consuming cows' milk) in studies investigating the health effects of breastfeeding beyond the first year of life

# 14 Abbreviations

AR	Adiposity rebound
BF	Body fat
BFMI	Body fat mass index
BMI	Body Mass Index
BMI z-score	Body Mass Index z-score
BMI SDS	Standardised BMI
BP	Blood pressure
CDC	US Centers for Disease Control
CEBQ	Child Eating Behaviour Questionnaire
CFU	Colony-forming unit
CHD	Coronary heart disease
CI	Confidence interval
COMA	Committee on Medical Aspects of Food and Nutrition Policy
COT	Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment
CS	Cross-sectional study
CC	Case control study
DMFT or dmft	Decayed, missing, filled teeth (lower case acronym refers to primary dentition)
dmfs	Decayed, missing, filled surfaces in primary dentition
DNSIYC	Diet and Nutrition Survey in Infants and Young Children
DRV	Dietary Reference Value
EAR	Estimated Average Requirements
ECC	Early childhood caries

EFSA	European Food Safety Authority
EU	European Union
FFQ	Food frequency questionnaire
FSIQ	Full-Scale Intelligence Quotient
g/day	Grams per day
Hb	Haemoglobin
HDL-C	High density lipoprotein cholesterol
HIC	High income country
HOME score	Home Observation Measurement of the Environment
HR	Hazard ratio
HRP	Household reference person
ID or IDA	Iron deficiency or iron deficiency anaemia
IQ	Intelligence quotient
ITT	Intention-to-treat analysis or population
IU	International units
Kcal	Kilocalorie
Kg	Kilogram
Kj	Kilojoule
KS2	Key stage 2
LC-PUFA	Long chain polyunsaturated fatty acids
LDL-C	Low density lipoprotein cholesterol
LIC	Low income country
LMIC	Lower middle income country
LRNI	Lower Reference Nutrient Intake
MA	Meta-analysis

MD	Mean difference
MMN	Multiple micronutrient(s)
NCMP	National Child Measurement Programme
NDNS	National Diet and Nutrition Survey
NR	Not reported
OR	Odds ratio
Oz	Ounce
PCS	Prospective cohort study
PLGV	Peak linear growth velocity
PP	Per protocol analysis
PPVT III	Peabody Picture Vocabulary test
PR	Prevalence ratio
PUFA	Polyunsaturated fatty acids
RCT	Randomised control trial
RE	Retinol equivalents
RNI	Reference Nutrient Intake
RoB	Risk of bias
RR	Relative Risk or risk ratio
SACN	Scientific Advisory Committee on Nutrition
SD	Standard deviation
SDS	Standard deviation score
SE	Standard error
S-ECC	Severe early childhood caries
SES	Socioeconomic status
SMD	Standardised mean difference

SMCN	SACN Subgroup on Maternal and Child Nutrition
SSB	Sugar-sweetened beverage
TAG	Triacylglycerol
T2D	Type 2 diabetes
TDEI	Total dietary energy intake
UK	United Kingdom
UMIC	Upper Middle Income Country
VIQ	Verbal Intelligence Quotient
WAZ	Weight-for-age z-score
WISC	Wechsler Intelligence Scale for Children
WHO	World Health Organization
WHZ	Weight-for-height z-score
WLZ	Weight-for-length z-score
WMD	Weighted mean difference

# 15 Glossary

Ad libitum diet	A diet in which the amount of food is not restricted.
Artificial sweeteners	Also referred to as non-nutritive sweeteners, non-sugar sweeteners, low calorie sweeteners or intense sweeteners, describing chemical low or no calorie substances that can be used to sweeten foods and drinks in place of sugar. The term 'artificial sweeteners' is also used in the UK government advice ( <a href="#">NHS</a> ) and therefore was adopted in this report. However, due to the lack of agreed terminology on artificial sweeteners, the terms adopted by the SR authors are used in the evidence section.
Breastfeeding	The feeding of an infant with milk taken from the breasts, either directly by the infant or expressed and given to the infant via a bottle or other drinking vessel.
Breastfeeding intensity	Breastfeeding intensity is defined as the proportion of daily feedings that are breast milk.
Body mass index (BMI)	An individual's weight in kilograms divided by the square of height in metres (kg/m <sup>2</sup> ). Often used as an indicator of adiposity with recognised limitations (Pietrobelli et al, 1998).
Bottle feeding	Feeding an infant from a bottle, whatever is in the bottle, including expressed breast milk, water, infant formula, etc.
Bioavailability	Bioavailability is defined as the efficiency with which a dietary component is used systemically through normal metabolic pathways. It is expressed as a % of intakes and is known to be influenced by dietary and host factors.
Cardiovascular disease	Cardiovascular disease is the most common cause of death in the UK and includes coronary heart disease, angina, heart attack and stroke.

Catch-up growth	Rapid growth following a period of restriction. Ultimately, it may redress wholly or partly the accrued deficit in weight or size though there may be consequences for body composition and metabolic capacity. This phenomenon is also often seen in children who are born small-for-gestational-age or with a low birthweight.
Cohort study	Systematic follow-up of a group of people for a defined period of time or until a specified event. Also known as a longitudinal study. A cohort study may collect data prospectively or retrospectively.
Complementary feeding	The WHO defines complementary feeding as “the process starting when breast milk alone is no longer sufficient to meet the nutritional requirements of infants” so that “other foods and liquids are needed, along with breast milk.” (PAHO, 2003). For the purposes of this report, complementary feeding refers to the period when solid foods are given in addition to either breast milk or infant formula to complement the nutrients provided by breast milk (and/or infant formula) when breast milk (and/or infant formula) alone is not sufficient to meet the nutritional requirements of the growing infant. Complementary feeding replaces the term ‘weaning’ which can be misinterpreted to mean the cessation of breastfeeding rather than the introduction of solid foods. Complementary feeding includes all liquids, semi-solid and solid foods, other than breast milk and infant formula.
Confounding factor	An unmeasured variable that influences both the exposure of interest (for example, nutrient intake) and the outcome (for example, body weight). These include gender, physical activity, social and economic influences, and ethnicity.
Crossover study design	A study design in which participants receive multiple interventions, and the effect of the interventions are measured on the same individuals.
Dairy	Dairy refers to milk produced by an animal, specifically a mammal such as goats, sheep, cows or even camels and water buffalo. All mammalian milk is considered dairy but there are differences in butterfat content, lactose, and protein.

Diabetes	A metabolic disorder involving impaired metabolism of glucose due to either failure of secretion of the hormone insulin, insulin-dependent or type 1 diabetes, OR impaired responses of tissues to insulin, non-insulin-dependent or type 2 diabetes.
Diet and Nutrition Survey of Infants and Young Children (DNSIYC)	Survey providing detailed information on the food consumption, nutrient intakes and nutritional status of infants and young children aged 4 up to 18 months living in private households in the UK. Fieldwork was carried out between January and August 2011.
Dietary diversity score	A hypothesis-driven approach of assessing diet quality. This method considers the number of portions from each food group (for example dairy, meat, cereals, fruits and vegetables) or foods consumed on regular basis (Gherasim et al, 2020). The underlying principle behind measuring dietary diversity is that to achieve a 'balanced diet', variety in dietary sources is needed. However, there is no standardised method of measuring dietary diversity (Gil et al, 2015).
Dietary guideline	The role of dietary guidelines is to assist populations to follow a healthy balanced diet with adequate nutrient intake and focus on prevention of non-communicable diseases.
Diversification of the diet	Diversification of the diet refers to the progression from an exclusively milk-based diet to an eating pattern which includes a wide range of foods.
Doubly labelled water (DLW) method	Doubly labelled water is water in which both the hydrogen (H) and oxygen ( $^{16}\text{O}$ ) have been partly or completely replaced for tracing purposes (that is, labelled) with 'heavy', non-radioactive forms of these elements: $^2\text{H}$ and $^{18}\text{O}$ . The DLW method measures the rate of disappearance of these 2 tracers given to an individual in water as they are washed out of the body. $^{18}\text{O}$ disappears faster from the body than $^2\text{H}$ because it is lost in both urine and as carbon dioxide in breath. $^2\text{H}$ is only lost from the body in urine. The difference between how fast $^2\text{H}$ and $^{18}\text{O}$ disappear provides a measurement of carbon dioxide production and this can then be converted into the amount of energy used.

## Dietary Reference Values (DRVs)

DRVs provide benchmark levels of nutrient requirements which can be used to compare mean values for population intakes. Although information is usually inadequate to calculate precisely and accurately the range of requirements for a nutrient in a group of individuals, it has been assumed to be normally distributed. This gives a notional mean requirement or Estimated Average Requirement (EAR) with the Reference Nutrient Intake (RNI) defined as two notional standard deviations above the EAR. Intakes above the RNI will almost certainly be adequate to meet the needs of 97.5% of the population. The Lower Reference Nutrient Intake (LRNI), which is two notional standard deviations below the EAR, represents the lowest intakes which will meet the needs of approximately 2.5% of individuals in the group. Intakes below this level are almost certainly inadequate for most individuals.

dmfs or DMFS

Decayed, missing, filled surfaces (in primary dentition, lower case; in permanent dentition, upper case)

dmft or DMFT

Decayed, missing, filled teeth (in primary dentition, lower case; in permanent dentition, upper case)

Dyslipidaemia

Dyslipidaemia is an abnormal amount of lipids (triacylglycerols, cholesterol or phospholipids) in the blood.

Dual-energy X-ray absorptiometry (DEXA)

A technique used to measure bone mineral density.

Early childhood caries (ECC)

ECC is defined as one or more decayed, missing or filled tooth surface in any primary tooth of children aged under 71 months. In children younger than 3 years of age, any sign of decay on the smooth surface of the teeth is indicative of severe early childhood caries (S-ECC) (AADP, 2021).

Eating Assessment in Toddlers (EAT) diet score

Based on Dietary Guidelines for Children and Adolescents in Australia; a higher EAT score indicates higher diet quality

Equivalised household income	Equivalisation is a standard methodology that adjusts for household income to account for different demands on resources by considering the household size and composition.
Estimated average requirement (EAR)	Estimated Average Requirement of a group of people for energy or protein or a vitamin or mineral. About half of a defined population will usually need more than the EAR, and half less.
Fat free mass (FFM)	The non fat component of body composition comprising muscle, bone, skin and organs.
Fat mass (FM)	The component of body composition made up of fat.
Formula milks	<p>Infant formula, follow-on formula, follow-up formula, 'growing-up' milk ('toddler' milk)</p> <p>Infant formula is a breast milk substitute commercially manufactured to Codex Alimentarius or European Union standards. Infant formula (based on either cows' milk or goats' milk) is the only suitable alternative to breast milk for babies who are under 12 months old. Follow-on formula is not suitable for babies under 6 months old and does not need to be introduced after 6 months. Beyond 1 year, infant and follow-on formula are not needed. 'Growing-up' milk ('toddler' milk) are marketed as an alternative to whole cows' milk for children aged 1 year and older. There is no evidence to suggest that these products provide extra nutritional benefits for young children.</p>
Free sugars	All sugars naturally present in fruit and vegetable juices, concentrates, smoothies, purées and pastes, powders, extruded fruit and vegetable products and similar products in which the structure has been broken down; all sugars in drinks (except for dairy-based drinks); and lactose and galactose added as ingredients (Swan et al, 2018).
Full-Scale Intelligence Quotient (FSIQ)	A broad measure of intelligence achieved through administration of a standardized intelligence test.

Healthy Start

UK-wide government scheme to offer a nutritional safety net for pregnant women, new mothers and children under 4 years of age in very low income families, and encourage them to eat a healthier diet. The scheme provides vouchers to put towards the cost of milk, fruit and vegetables or infant formula, and coupons for free Healthy Start vitamin supplements.

High income country (HIC)

The World Bank defines economies into four income groupings: low, lower-middle, upper-middle, and high. Income is measured using gross national income (GNI) per capita, in US dollars, converted from local currency using the World Bank Atlas method. Estimates of GNI are obtained from economists in World Bank country units; and the size of the population is estimated by World Bank demographers from a variety of sources, including the United Nation's biennial World Population Prospects. In 2023, a HIC was defined as having a GNI per capita of \$13,205 or more ([New World Bank country classifications by income level: 2022-2023](#)).

Home Observation Measurement of the Environment (HOME) score

The primary measure of the quality of a child's home environment. It has been used as both an input in helping to explain other child characteristics or behaviours and as an outcome for researchers whose objective is to explain associations between the quality of a child's home environment and earlier familial and maternal traits and behaviours.

Index of Multiple Deprivation (IMD)

The Index of Multiple Deprivation (IMD) is the official measure of relative deprivation in England and is part of a suite of outputs that form the [Indices of Deprivation](#) (IoD). It follows an established methodological framework in broadly defining deprivation to encompass a wide range of an individual's living conditions. People may be considered to be living in poverty if they lack the financial resources to meet their needs, whereas people can be regarded as deprived if they lack any kind of resources, not just income.

Infant

A child under the age of 12 months.

Infant Feeding Survey (IFS)	National survey of infant feeding practices conducted every 5 years from 1975 to 2010. The survey provided national estimates of the incidence, prevalence, and duration of breastfeeding (including exclusive breastfeeding) and other feeding practices adopted by mothers in the first 8 to 10 months after their infant was born. In the more recent surveys these estimates were provided separately for England, Wales, Scotland and Northern Ireland, as well as for the UK as a whole.
Infant formula	See 'Formula milks'
Intervention study	Comparison of an outcome (for example, disease) between two or more groups deliberately subjected to different exposures (for example, dietary modification or nutrient supplementation).
Intrinsic sugars	Sugars that are naturally incorporated into the cellular structure of foods.
Key Stage 2 (KS2)	Formal assessments tests in English (grammar, punctuation, spelling and reading) and maths that children in the UK take in year 6 (at age 11 years).
Kilocalorie	Units used to measure the energy value of food, 1kcal = 4.18kJ
Kilojoule or megajoule	Units used to measure the energy value of food, 1kJ=1000 joules, 1MJ = 1 million joules
Linear growth	An increase in the length or height of an infant or child.
Longitudinal study	In a longitudinal study, individual subjects are followed through time with continuous or repeated monitoring exposures, health outcomes, or both.
Low birthweight	Low birthweight is defined as less than 2,500g (up to and including 2,499g). Infants may be low birthweight because they are born too early or are unduly small for gestational age.

Low middle income country (LMIC)

The World Bank defines economies into four income groupings: low, lower-middle, upper-middle, and high. Income is measured using GNI per capita, in US dollars, converted from local currency using the World Bank Atlas method. Estimates of GNI are obtained from economists in World Bank country units; and the size of the population is estimated by World Bank demographers from a variety of sources, including the UN's biennial World Population Prospects. In 2023, a LMIC was defined as having a GNI per capita of \$1,086 to \$4,256. ([New World Bank country classifications by income level: 2022-2023](#)).

Low income country (LIC)

The World Bank defines economies into four income groupings: low, lower-middle, upper-middle, and high. Income is measured using GNI per capita, in US dollars, converted from local currency using the World Bank Atlas method. Estimates of GNI are obtained from economists in World Bank country units; and the size of the population is estimated by World Bank demographers from a variety of sources, including the UN's biennial World Population Prospects. In 2023, a LIC was defined as having a GNI per capita of \$1,085 or less. ([New World Bank country classifications by income level: 2022-2023](#)).

Lower reference nutrient intake (LRNI)

The estimated average daily intake of a nutrient which can be expected to meet the needs of only 2.5% of a healthy population. Values set may vary according to age, gender and physiological state (for example, pregnancy or breastfeeding).

Macronutrients

Nutrients that provide energy, including fat, protein and carbohydrate.

Malocclusion

Malocclusion describes the alignment of teeth which are considered not to be in a normal position in relation to adjacent teeth (that is, the teeth are not correctly aligned).

Margin of exposure

This [approach](#) provides an indication of the level of health concern about a substance's presence in food. EFSA's Scientific Committee states that, for substances that are genotoxic and carcinogenic, an MOE of 10,000 or higher is of low concern for public health.

Meta-analysis	A quantitative pooling of estimates of effect of an exposure on a given outcome, from different studies identified from a systematic review of the literature
Micronutrients	Essential nutrients required by the body in small quantities, including vitamins and minerals.
National Diet and Nutrition Survey	A continuous cross-sectional survey of food consumption, nutrient intakes and nutritional status in children aged 18 months upwards (as well as adults and adolescents) in the UK.
Non-milk extrinsic sugars	Extrinsic sugars are those sugars not contained within the cellular structure of a food. The extrinsic sugars in milk and milk products (that is, lactose) were deemed to be exempt from the classification of sugars in relation to the dietary reference values set by COMA in 1991. Non-milk extrinsic sugars added to foods (for example, sucrose, glucose and fructose) and sugars naturally present in fruit juices (for example, glucose and fructose).
Nutrient deficiency	Impaired function due to inadequate supply of a nutrient required by the body.
Odds ratio (OR)	A measure of association between an exposure and an outcome. The OR represents the odds that an outcome will occur given a particular exposure, compared with the odds of the outcome occurring in the absence of that exposure. The OR is adjusted to address potential confounding.
Percentage point	A percentage point is the unit for the arithmetic difference between two percentages. For example, the difference between 30% and 33% is 3 percentage points.
Pre-post study	Also known as a before-after study. A study that measures outcomes in a group of participants before introducing an intervention, and then again afterwards. Any changes in the outcomes are attributed to the intervention. This study design cannot rule out that something other than the intervention may have caused a change. Randomised controlled trials (RCTs) are considered the most reliable way to show that your digital product has caused an outcome. However, it is not always possible to run an RCT. Before-and-after studies are more flexible and generally cheaper to run.

Randomised controlled trial (RCT)	A study in which eligible participants are assigned to two or more treatment groups on a random allocation basis. Randomisation assures the play of chance so that all sources of bias, known and unknown, are equally balanced.
Reference nutrient intake (RNI)	The average daily intake of a nutrient sufficient to meet the needs of almost all members (97.5%) of a healthy population. Values set may vary according to age, gender and physiological state (for example, pregnancy or breastfeeding).
Retinol equivalents (RE)	To take account of the contribution from provitamin A carotenoids, the total vitamin A content of the diet is usually expressed as micrograms ( $\mu\text{g}$ ) of retinol equivalents (RE): $1\mu\text{g RE} = 1\mu\text{g retinol} = 6\mu\text{g beta-carotene} = 12\mu\text{g other carotenoids with provitamin A activity}$
Relative risk (RR)	The ratio of the rate of disease or death among people exposed to a factor, compared with the rate among the unexposed, usually used in cohort studies (World Cancer Research Fund & American Institute for Cancer Research, 2007).
Responsive feeding	A form of 'responsive parenting', in which parents are aware of their child's emotional and physical needs and react appropriately to their child's signals of hunger and fullness.
Risk factor	A factor demonstrated in epidemiological studies to influence the likelihood of disease in groups of the population.
Safe intake	Safe Intakes are set for some nutrients if there is insufficient reliable data to establish DRVs. They are based on a precautionary approach and are 'judged to be a level or range of intake at which there is no risk of deficiency, and below a level where there is a risk of undesirable effects (DH, 1991).
Solid foods	Foods other than breast milk or formula milk introduced to the infant diet at the commencement of complementary feeding.

Sugar-sweetened beverage	In this report, a sugar-sweetened beverage (SSB) is any (non-dairy) beverage (carbonated drinks, fruit-based drinks, squashes, flavoured water) where free sugars have been specifically added as a sweetener. Where possible, these are distinguished from 100% fruit juices (with naturally occurring levels of sugars).
Systematic review	An extensive review of published literature on a specific topic using a defined search strategy, with a priori inclusion and exclusion criteria.
Tolerable upper level (TUL)	A tolerable upper intake level (TUL) is intended to specify the level above which the risk for harm begins to increase and is defined as the highest average daily intake of a nutrient that is, likely to pose no risk of adverse health effects for nearly all persons in the general population, when the nutrient is consumed over long periods of time, usually a lifetime.
Total dietary energy intake (TDEI)	In this report, TDEI is used for consistency with previous SACN reports. However, in young children, this is equivalent to total energy intake because this age group, unlike adults, does not obtain energy from alcohol.
Upper middle income country (UMIC)	The World Bank defines economies into four income groupings: low, lower-middle, upper-middle, and high. Income is measured using GNI per capita, in US dollars, converted from local currency using the World Bank Atlas method. Estimates of GNI are obtained from economists in World Bank country units; and the size of the population is estimated by World Bank demographers from a variety of sources, including the UN's biennial World Population Prospects. In 2023, a UMIC was defined as having a GNI per capita of \$4,256 to \$13,205. ( <a href="#">New World Bank country classifications by income level: 2022-2023</a> ).
Verbal Intelligence Quotient (VIQ)	A numerical measurement of child's spoken language capabilities and limitations. It is used to gauge child's ability to reason out and understand others through spoken words.

Weaning	The process of expanding the diet to include foods and drinks other than breast milk or infant formula (DH, 1994b). The term complementary feeding is preferred to describe diversification of the diet because 'weaning' has also been used to describe curtailment of breastfeeding.
Wechsler Intelligence Scale for Children (WISC)	An individually administered intelligence test for children between the ages of 6 and 16. It generates a Full Scale IQ that represents a child's general intellectual ability.
Young child	A child aged between 12 and 36 months (1 and 3 years).
Z-score	The z-score (or standard deviation (SD) score) is defined as the difference between an observed value for an individual and the median value of the reference population, divided by the standard deviation value of the reference population. Z-scores are used for height, weight and head circumference.

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