Scientific Advisory Committee on Nutrition

‘Feeding young children aged 1 to 5 years’

Contents

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Ms Debby Webb (OHID, Department of Health and Social Care)

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Membership of SACN

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1 Introduction

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, and to examine the evidence relating to the influence of the infant diet on development of allergic and autoimmune disease.

1.4 This draft 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).

1.5 In keeping with SACN's terms of reference, this draft report is restricted to risk assessment and only evidence in young children in health has been considered. The draft report considers evidence identified on:

- young child energy, macronutrient and micronutrient requirements
- eating and feeding behaviours
- dietary patterns and consumption of individual foods
- weight status
- oral health.

1.6 Risks of chemical toxicity arising from the young child diet are also covered.

1.7 In line with the SACN Framework for the Evaluation of Evidence (SACN, 2012; SACN, 2020), the draft report does not provide advice on how recommendations are taken forward for policy, that is, risk management. 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 draft report.

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

Terms of reference

1.9 The terms of reference for this review are defined below.

- To review the scientific basis of current recommendations for complementary and young child feeding up to 5 years (60 months) of age[1]. This draft report covers young children aged 1 to 5 years of age[2]
- 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)[3]
- To make recommendations for policy, practice and research.

1.10 As noted above, this draft report is 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). 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 will impact on nutritional status and health outcomes in the second year of life and beyond.

1.11 The key dietary factors considered in this draft report are:

- Energy requirements
- Macronutrients
- Micronutrients (focus on vitamins A and D[4], iron and zinc)
- Dietary patterns (including consideration of vegetarian and vegan diets, and consumption of different food groups)

[1] To note that this should be understood as 5 **completed** years of age.

[2] 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.

[3] As this report covers the 1 to 5 age group, it was not considered relevant to address this term of reference

[4] 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.

- Chemical contaminants (or the risk of chemical toxicity)
- The latest available nutritional intakes and status of children aged 1 to 5 years (12 to 60 months) in the UK

Health outcomes considered

1.12 The health outcomes considered in this draft report are divided into those relating to childhood health and those relating to future health.

1.13 Childhood health outcomes are:

- Growth and body composition
  - linear growth
  - body composition (lean mass, adiposity, body mass index)
  - underweight or overweight or obesity
- Neurological outcomes, including cognitive outcomes
- Bone or skeletal health outcomes
- Oral health
- Development of eating habits and feeding behaviours
- Morbidities (including respiratory diseases)

1.14 Future health outcomes are:

- Obesity or body composition
- Cancer
- Cardiovascular outcomes (coronary heart disease, diabetes)

1.15 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.

History of policy development

UK recommendations on feeding children aged 1 to 5 years

1.16 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.

1.17 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.

1.18 The recommendations from the COMA report (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/snacks per day)

1.19 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.

1.20 The main current UK government recommendations on feeding young children and the evidence informing these are detailed in Chapter 11, Table 11.1. Table 11.1 also summarises current UK government dietary recommendations, including those on free sugars, fish and the Eatwell guide, that are informed by evidence from other reports, such as the SACN report 'Carbohydrates and Health' (SACN,

2015) and the SACN/COT 'Advice on fish consumption: benefits and risks' (SACN/COT, 2004).

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

1.21 In addition to UK government dietary advice, there are several statutory schemes (see Annex 1, Table A1.1) that aim to improve the dietary intakes of young children in the UK, including the Healthy Start scheme and Best Start Foods scheme.

1.22 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 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.

1.23 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 fruit or vegetables; and dried, fresh, frozen or tinned pulses. Entitlement to vitamins was not included in the BSF scheme and instead this was replaced by universal access to vitamin D provision for children under 3 years and breastfeeding mothers.

World Health Organization (WHO) recommendations

1.24 The WHO has published several reports which provide recommendations for infant and 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).

1.25 Since 2001, the WHO has recommended that mothers worldwide exclusively breastfeed 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 guidelines also covered:

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

1.26 The WHO/UNICEF (2003) guidelines are further summarised in Annex 1 Table A1.2. 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

1.27 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 and 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 have been summarised in Annex 1 Table A1.2.

Current context

1.28 Food consumption, nutrient intakes and nutritional status in children in the UK are captured in 2 large national surveys, the Diet and Nutrition Survey of Infants and Young Children (DNSIYC) (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 children aged 4 to 18 months carried out over 8 months in 2011, while 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 methodology used in DNSIYC and NDNS see Annex 2.

1.29 The DNSIYC and the latest NDNS (collection years 2016/17 to 2018/19, or years 9 to 11 of the Rolling Programme) indicate that children in the UK are exceeding current UK government recommendations for dietary energy, protein, saturated fats and free sugars while not meeting recommendations for dietary fibre. The NDNS also suggests that there are proportions of children (>5 to 10%) in some age groups under 5 years that may have inadequate intakes of iron, zinc, vitamin A and vitamin D.

1.30 As a consequence of poor diets, the prevalence of overweight and obesity in young children has been on the rise, and oral health remains poor.

1.31 The most recent National Child Measurement Programme (NCMP), a nationally mandated public health programme in England (see Chapters 3 and 7 for details), showed that the prevalence of overweight and obesity in children who entered reception year of primary school (aged 4 to 5 years) in England was nearly 28% in the collection year 2020/21 (NHS Digital, 2021). This was a marked increase on the prevalence of overweight and obesity before the COVID-19 (coronavirus) pandemic (at 23%).

1.32 Dental caries in children remains a major public health problem. Latest available survey data indicate that 11% of children aged 3 years (PHE, 2021c) and 23% of children aged 5 years (PHE, 2020) 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. Just under 50,000 children aged 0 to 19 years were admitted to hospital to have teeth removed under general anaesthesia in 2019/2020 (PHE, 2021b).

Determinants of dietary behaviours and lifelong health and disease

1.33 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).

1.34 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 6 for details). The food preferences and eating habits of infants and young children are strongly shaped by their parents and caregivers' attitudes, beliefs and behaviours about food and feeding (Schwartz et al, 2011) and their parenting style. Wide cultural and regional differences in complementary feeding practices (Mennella et al, 2006) may also contribute to strong preferences for regional cuisines observed in infants and children (Mennella & Beauchamp, 2005). Children's food and meal environments can also be influenced by parents and carers through factors affecting feeding behaviours such as food accessibility and availability and the timing and frequency of meals (Silventoinen et al, 2010; Vollmer & Mobley, 2013).

2 Methodology

2.1 This draft report is based primarily on evidence provided by systematic reviews (SRs) and/or meta-analyses (MAs) of prospective cohort studies (PCS) and randomised controlled trials (RCTs). 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.

2.2 SACN's Framework for the Evaluation of Evidence (SACN, 2012) was used as the basis for assessing the evidence. The Framework is based on an evidence hierarchy which ranks the certainty of the evidence according to study design. More weight is placed on evidence from RCTs since well-conducted RCTs minimise the potential for selection bias and confounding. Less weight is placed on observational studies because such studies are potentially subject to bias, confounding and reverse causality. However, in the absence of RCTs, evidence from non-randomised intervention studies (NRSI) and prospective studies is considered stronger than that from other study designs (case-control, cross-sectional and case reports).

Evidence review process

Inclusion criteria

2.3 The following types of studies were included: SRs and MAs of RCTs, NRSI and PCS.

2.4 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 (HIC). Evidence from studies conducted in low and middle income countries (LIC and MIC) (using the World Bank classification) that was potentially relevant to the UK context was also considered.

Exclusion criteria

2.5 The following types of studies were excluded: primary studies, reviews that included only case-control studies, and narrative reviews.

2.6 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, unless they had 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

2.7 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 1.13 and 1.14).

2.8 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 2.3 and 2.4).

2.9 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).

2.10 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.

2.11 A supplementary online database search was performed for oral health in October 2019.

2.12 The agreed cut-off date for consideration of newly published eligible evidence was 22 May 2019. It was agreed that publications identified after 22 May 2019 would be considered as part of the draft report consultation, and that the draft report would be amended if any evidence identified after 22 May 2019 or through the consultation process was judged to have an important bearing on the conclusions.

Selection of studies

2.13 After removing duplicates, titles and abstracts of the identified publications were screened for eligibility.

2.14 The screening steps for eligibility (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.

2.15 Six thousand and ninety seven records were identified from 5 online databases (see paragraph 2.8). 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.

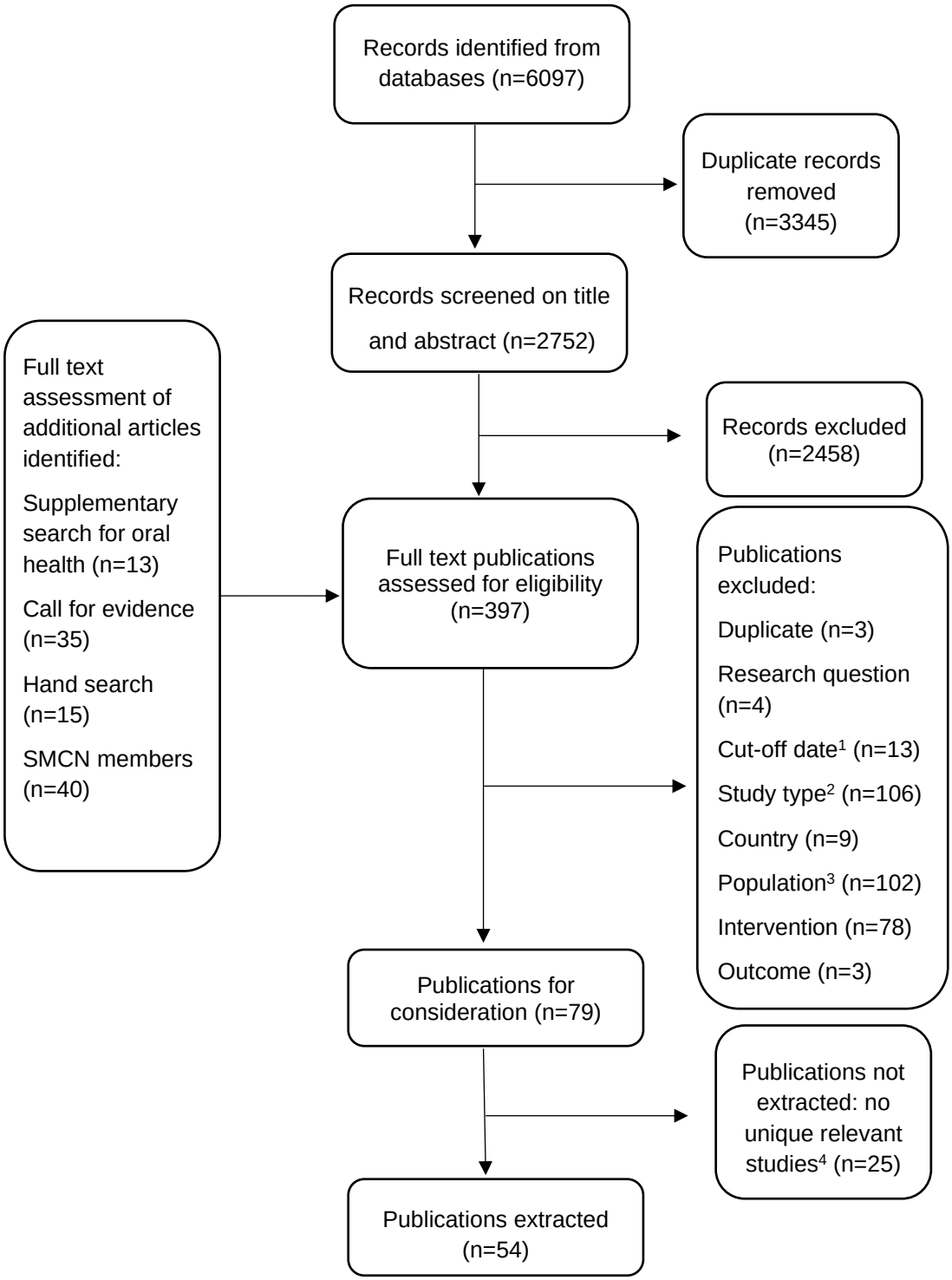
2.16 The supplementary online database search on oral health returned 2701 records, and full texts of 13 were retrieved and screened.

2.17 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 studies (RCT or NRSI or PCS) in the 1 to 5 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.

2.18 The process for study selection and inclusion is shown in Figure 2.1. Details of the excluded references and reasons for their exclusion are presented in Annex 4, Tables A4.1 to A4.3.

Figure 2.1 Flow diagram showing the literature selection process (described in paragraphs 2.15 to 2.19)



[1] Published before the cut-off dates for consideration of evidence for previous SACN reports (SACN, 2010; SACN, 2016).
[2] Excluded for not being a SR or for not including studies (RCT or NRSI or PCS) in the 1 to 5 age group.
[3] Excluded for not including studies or findings in the 1 to 5 age group or in children in health.
[4] All relevant studies included in more recent or comprehensive SRs considered in the draft report.

Data extraction

2.19 Of the 79 eligible SRs, evidence from 25 was included in more recent or comprehensive reviews and was not extracted into evidence tables (see Annex 4, Table A4.3 for details of these SRs).

2.20 Details of the remaining 54 SRs are presented in evidence tables in Annex 5 (Tables A5.1 to A5.6). The following data were extracted: first author, year of publication, study design, funding, declaration of interest, research question, total number of participants, demographics, outcome measures, confounders, study findings, the assessment of study quality and limitations identified by the SR authors.

2.21 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 (see Annex 8, Tables A8.1 to A8.44). 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 if these were from:

- primary studies that 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
- RCTs, NRSI or PCS; if the SR reported results from cross-sectional analyses from a RCT or PCS, these results were not extracted.

2.22 For SRs with MAs, summary estimates from the MAs were extracted (rather than the individual findings of primary studies) except in the following instances:

- if the summary estimate pooled estimates from cross-sectional or case control studies
- if studies in children aged 1 to 5 contributed less than 50% to the summary estimate
- if a subgroup analysis in children aged 1 to 5 years was performed, then only the estimate from that subgroup was extracted.

2.23 Many of the SRs described the age of participants in months rather than years. If the data were available, age was described in months.

Prioritisation of evidence and reporting of results

2.24 Chapters 3 to 8 have been divided into:

- a background section summarising current UK dietary recommendations and findings from national survey data of dietary intakes and nutritional status in children aged 1 to 5 years in the UK (Diet and Nutrition Survey in Infants and Young Children (DNSIYC) and the National Diet and Nutrition Survey (NDNS) (see section on ‘Other evidence considered’, p.37)
- an evidence section describing the findings from SRs identified on each topic area and the assessment of that evidence

2.25 In this draft report, ‘body composition’ was used to denote measures such as body mass index (BMI), body fat or adiposity; while ‘weight status’ was used to collectively denote underweight and excess weight (overweight or obesity). Overweight and obesity are defined in Chapter 7.

2.26 Where appropriate, evidence that related dietary or nutritional intakes to measures of body composition or weight status were grouped together and presented ahead of evidence that related dietary or nutritional intakes to other health outcomes (see Table 2.1).

2.27 If the evidence informing a topic area came from ≥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 while, for the smaller SRs, only findings from primary studies that were uniquely identified by and included in those SRs were reported.

2.28 If the evidence informing a topic area came from ≥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 section on ‘Grading of the evidence’ p.33).

2.29 The overlap between eligible SRs is shown in Annex 6, Tables A6.1 to A6.7.

2.30 Results were reported as they were reported in the SRs, and if available, full statistical findings (effect estimates, confidence intervals, p values, and statistical heterogeneity) were reported. If any of these were not reported, this is indicated by ‘NR’ (not reported).

Health outcomes for which evidence was identified

2.31 This draft report sought to identify evidence on a number of outcomes related to childhood health and future health. Table 2.1 and Table 2.2 details the health outcomes for which evidence was identified (also stated in Chapter 1, paragraphs 1.13 and 1.14) and where in the draft report evidence for these are located. Evidence was also sought but not identified on paediatric cancer (such as childhood leukaemia), child allergic and autoimmune disease, adult neurological and bone/skeletal health.

Table 2.1. Health outcomes for which evidence was identified

Outcome – childhood health	Location in the draft report
Growth, body composition or weight status[1]	Chapter 3 – energy and macronutrients Chapter 4 – micronutrients Chapter 5 – foods, dietary components, and dietary patterns Chapter 6 – eating and feeding behaviours Chapter 7 – obesity
Neurological outcomes, including cognitive outcomes	Chapter 3 – energy and macronutrients Chapter 4 – micronutrients Chapter 5 – foods, dietary components, and dietary patterns
Bone/skeletal health outcomes	Chapter 3 – energy and macronutrients Chapter 4 – foods, dietary components, and dietary patterns
Oral health	Chapter 8 – oral health
Development of eating habits and feeding behaviours	Chapter 6 – eating and feeding behaviours
Morbidities, including respiratory diseases	Chapter 4 – micronutrients

[1] 'Body composition' denotes outcome measures including BMI, BMI z-scores, weight-for-height z-scores. 'Weight status' denotes underweight or excess weight (overweight or obesity), which are defined in Chapter 7.

Table 2.2. Health outcomes for which evidence was identified

Outcome – future health	Location in the draft report
Overweight or obesity	Chapter 7 – obesity
Cardiovascular outcomes (coronary heart disease, diabetes)	Chapter 7 – obesity
Adult cancer	Chapter 7 – obesity

Evaluation of the quality of identified evidence

2.32 The quality of evidence was assessed by:

- the SACN Framework for the Evaluation of Evidence (SACN, 2012)
- the 'A Measurement Tool to Assess Systematic Reviews 2' (AMSTAR 2) tool (AMSTAR, 2021)

SACN Framework

2.33 The following criteria were considered:

- 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 sub-group and sensitivity analyses.

2.34 The word ‘effect’ was used to describe the evidence from RCTs and the word ‘association’ was used when referring to evidence from PCS. An effect/association was deemed to be statistically significant using the $p<0.05$ criterion.

AMSTAR 2 assessment

2.35 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.

2.36 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/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?

2.37 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[1]: 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.
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.

[1] 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.

2.38 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.

2.39 As many of the SRs identified for this risk assessment included all study designs, item 3 was not considered applicable.

2.40 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 7 (Table A7.1 to A7.8).

Approach to considering statistical methods

2.41 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).

2.42 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 below).

Grading of the evidence

2.43 The methods for grading evidence that were outlined in the SACN reports 'Carbohydrates and Health' (SACN, 2015), 'Saturated Fats and Health' (SACN, 2019) were modified for use in this draft report.

2.44 The certainty of the evidence for each exposure-outcome relationship was graded *adequate*, *moderate*, *limited*, *inconsistent* or *insufficient*. 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 2.3, 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).

2.45 Summary tables of the evidence grading process for each exposure-outcome relationship are presented in Annex 9 (Tables A9.1 to A9.28).

2.46 Exposure-outcome relationships for which there were fewer than 3 intervention or prospective cohort studies were automatically graded *insufficient*. The exposure-outcome relationships for which evidence was graded *insufficient* are listed in Annex 9 (Table A9.29).

2.47 Evidence identified from SRs that was graded *adequate* or *moderate* were used to inform conclusions and recommendations of this draft report, alongside findings from national diet and nutrition surveys (see paragraphs 2.48 to 2.52).

Table 2.4 Criteria for grading evidence (SACN, 2019)

Certainty of evidence	Explanatory notes
Adequate	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. Taking into account overlap of primary studies included in the identified publications, evidence from meta-analyses goes in the same direction. 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/associations are also consistent when major population subgroups or other relevant factors are considered in additional analyses. The identified publications are considered to be of good quality based on the key factors listed above. The inclusion and exclusion criteria of the identified publications are well defined and appropriate. 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. 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 ≥ 3 RCTs or ≥ 5 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 ≥ 4 RCTs or ≥ 5 PCS studies, of *adequate* size and considered to be of good quality, consistently went in the same direction.

Certainty of evidence	Explanatory notes
Moderate	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. Taking into account overlap of primary studies included in the identified publications, the majority of the evidence from MAs goes in the same direction. 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. Effects or associations may be less consistent when major population subgroups or other relevant factors are considered in additional analyses. The identified publications are considered to be of moderate to good quality based on the key factors listed above. The inclusion and exclusion criteria of the identified publications are reasonably well defined and generally appropriate. 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. 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 ≥3 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 ≥ 3 RCTs or 5 PCS, of moderate size and considered to be of moderate quality, consistently went in the same direction.

Certainty of evidence	Explanatory notes
Limited	There is *limited* evidence (therefore, even less conclusive) to make a decision about the effect or association of a factor(s)/intervention(s) in relation to a specific outcome. 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. 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. Effects or associations may be inconsistent when major population subgroups or other relevant factors are considered in additional analyses. The identified publications are considered to be of poor to moderate quality based on the key factors listed above. The inclusion and exclusion criteria of the identified publications are not well defined and may not be appropriate. 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. 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.
Inconsistent	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.
Insufficient	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, <3 to 4 eligible RCTs or PCS were identified. Therefore, it is not possible to draw conclusions.

Other evidence considered

2.48 Two large national surveys informed the sections describing current feeding practices of young children in the UK. These were the Diet and Nutrition Survey of Infants and Young Children (DNSIYC) 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. The majority of NDNS data and analyses presented in this draft report is based on the 3 most recent collection years available (years 2016/17 to 2018/19). Some analyses are based on all 11 years of data available (years 2008/09 to 2018/19) where larger cell sizes were required. For a summary of the methodology used in the DNSIYC and the NDNS see Annex 2. Full details of the methodology and findings from the 2 surveys can be found elsewhere (Bates et al, 2020; Lennox et al, 2013).

2.49 The National Child Measurement Programme (NCMP), a nationally mandated public health programme in England, informed sections on the prevalence of overweight and obesity in young children (in Chapters 3 and 7).

2.50 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 9.

2.51 In addition to the literature searches outlined (paragraphs 2.7 and 2.8), previously published SACN reports[5] of relevance to this draft report were considered and searches were undertaken to update evidence that might have accrued since their publication.

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

[5] 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/RCPCH report on: Application of WHO growth standards in the UK (2007).

Process for assessment of the evidence

2.53 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.

2.54 This draft report has been made available for public consultation and the comments received from interested parties will be taken into consideration before the report is finalised.

General limitations of the evidence

2.55 This section describes a number of general limitations that were identified in the SR evidence included in this draft report. Limitations specific to each topic area are summarised in their respective chapters. For limitations of the evidence from the NDNS, refer to Chapter 4 (‘Micronutrients’) and Annex 2.

2.56 There was no or insufficient SR evidence for a large number of exposure-outcome relationships of interest for this risk assessment (see Annex 9, Table A9.29).

2.57 SRs (with and without MAs) were included in the draft report if they searched for evidence in children aged 1 to 5 years. However, many of these SRs had a broader search strategy that included population groups outside the target age (that is, they searched for studies in children aged under 12 months and/or above the age of 5 years). It was therefore difficult to determine whether the search strategy for the target age was comprehensive, thereby minimising the risk of publication bias.

2.58 Publication bias was not formally (statistically) assessed by many of the SRs as the majority did not perform a MA or any other method of quantitative synthesis.

2.59 SRs without MAs reported findings from primary studies with varying levels 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.

2.60 Most of the evidence from SRs was from observational or NRSI. Non-randomised studies are at high risk of confounding and selection 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. Not all non-randomised studies (including non-randomised trials, prospective cohort studies, quasi-experimental studies) adjusted for all such confounding factors either in their study design or in their analyses. Even if they did, it is 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).

2.61 Among the studies that did consider the impact from potential confounding, a lack of consistency in the covariates considered made comparisons between study findings difficult. Risk of selection bias was also not always accounted for by the SRs. For example, if there are systematic differences between participants lost to follow up and participants who complete the study, this could lead to attrition bias, a form of selection bias.

2.62 Most SRs that included RCTs or other randomised study designs did not include information on the type of analysis (intention-to-treat or per protocol) carried out by the studies. Intention-to-treat (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). Per protocol (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).

2.63 Many primary studies either did not perform power calculations or did not report doing so. Findings of null associations with wide confidence intervals should therefore be interpreted with caution.

2.64 The evidence base on most 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 pooled analyses.

2.65 Primary studies included in the SRs used different dietary assessment methods (for example, food frequency questionnaires, 24-hour recalls, food diaries). In many 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, changes to normal intakes during the recording period and general underreporting (by failing to report foods or drinks consumed and/or underestimating quantities) 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.

2.66 The types of outcome data reported included dichotomous (risk ratios, odds ratios) and continuous (mean difference) measures, making it difficult or inappropriate to synthesise findings. 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 (≥10%) (Ranganathan et al, 2015).

2.67 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, increasing the unreliability of the measurements.

2.68 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 covered in this draft report did not allow conclusions to be drawn about the longer term health effects of nutrient or dietary intake, or sustained effects of increasing children's acceptance or intake of certain foods (for example, vegetables).

2.69 Primary studies that were conducted in high income countries (HIC) did not always account for ethnicity or included only white participants. It is therefore unclear whether the effects of dietary factors on health outcomes differ between individuals from different ethnic minority groups.

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 deteriorate between the ages of 11 and 15 months (Brugailleres et al, 2019). Experimental research has shown that by the time children 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 Section on portion sizes, paragraphs 3.26 to 3.29).

3.3 An impaired ability to self-regulate energy intake may tip the balance between energy intake 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).

3.4 Overweight and obesity occurs when TDEI is greater than the energy that is expended over a prolonged period (SACN, 2015). As presented later in this chapter, national dietary surveys in the UK indicate that children aged 1 to 5 years have TDEI that exceed government recommendations for this age group. The proportion of children entering school (aged 4 to 5 years) in England with overweight or obesity (indicated by the 85th and 95th centiles, respectively) in the

year 2020/21 was 28%, up from 23% the year before the COVID-19 pandemic. At the same time, there were large increases in children aged 4 to 5 years who were categorised as obese (including severely obese) (4.5 percentage points), severely obese (2.2 percentage points), and overweight or obese (4.7 percentage points) (NHS Digital, 2021).

Current recommendations for energy intake in the UK

3.5 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

3.6 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.

3.7 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.

3.8 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)[1]		SACN (2011)		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

[1] Source: (DH, 1991).

TDEI and children's weight status in the UK

3.9 TDEI and body weight data for children in the UK 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 2008/09 to 2018/19) are shown in Table 3.2.

Table 3.2. TDEI and body weight of children aged 12 to 60 months in the UK (DNSIYC and NDNS years 2008/09 to 2018/19)[1]

Age	EAR (MJ per day)		Energy intake (MJ per day)[2] mean (SD)		% participants above EAR		Body weight (kg)[3] mean (SD)	
	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls
12 to 18 months	3.2	3.0	4.2 (0.9)	4.0 (0.9)	88	88	11.2 (1.3)	10.6 (1.3)
18 to 23 months	3.2	3.0	4.5 (0.9)	4.1 (0.8)	96	87	12.6 (1.6)	11.7 (1.6)
24 to 35 months	4.2	3.9	4.7 (1.0)	4.4 (0.9)	69	69	14.7 (3.1)	13.5 (1.5)
36 to 47 months	4.9	4.5	4.9 (1.0)	4.8 (1.2)	47	58	16.6 (2.3)	16.1 (2.5)
48 to 60 months	5.8	5.4	5.7 (1.1)	5.1 (1.1)	43	37	18.7 (2.4)	18.0 (3.1)

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

[1] Data source: DNSIYC 2011 (Lennox et al, 2013) for children aged 12 to 18 months and from NDNS years 2008/09 to 2018/19 for children aged 18 to 60 months.

[2] Number of participants in each age category 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] Number of participants in each age category for body weight: 619 boys and 609 girls (12 to 18 months); 123 boys and 110 girls (18 to 23 months); 256 boys and 232 girls (24 to 35 months); 242 boys and 250 girls (36 to 47 months); 225 boys and 210 girls (48 to 60 months).

3.10 The surveys indicated that 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 2.65).

3.11 Time trend analysis of NDNS data (years 2008/09 to 2018/19) for children aged 18 to 47 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 age groups 12 to 18 months or 48 to 60 months.

3.12 At the same time, the most recent findings from the National Child Measurement Programme (NCMP) show that while the combined prevalence of overweight and obesity (see Chapter 7, paragraph 7.6) for children in England entering reception year of primary school (at ages 4 to 5 years) was fairly stable at approximately 22 to 23% from the collection years 2006/2007 to the year preceding the COVID-19 pandemic (2019/2020), this increased significantly to 28% in the collection year 2020/21 (NHS Digital, 2021). In addition, there were large increases in reception year children categorised as obese (including severely obese) (from 10% to 14%) and severely obese (from 3% to 5%). For both collection years 2019/20 and 2020/21, the prevalence of overweight and obesity was higher for boys than girls, and the increase in prevalence from 2019/20 to 2020/21 was also higher for boys than girls (see Chapter 7 for details).

TDEI, body weight and deprivation

3.13 TDEI by index of multiple deprivation (IMD), a broad measure of deprivation (see Annex 13, Glossary), for children aged 18 to 60 months from NDNS (years 2008/09 to 2018/19) is shown in Table 3.3. Body weight by IMD for the same age group is shown in Table 3.4.

Table 3.3. TDEI by IMD quintile in children aged 18 to 60 months in England[1]

Energy (MJ/day)	IMD quintile 1 (least deprived)	IMD quintile 2	IMD quintile 3	IMD quintile 4	IMD quintile 5 (most deprived)
Mean (90%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

[1] Data from NDNS years 2008/09 to 2018/19.

Table 3.4. Body weight by IMD quintile in children aged 18 to 60 months in England[1]

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 (90%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)
	Number of participants	136	148	120	148	178
48 to 60 months	Mean (90%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)
	Number of participants	55	47	46	64	64

[1] Data from NDNS years 2008/09 to 2018/19.

3.14 There appears to be 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, and risk of underreporting of TDEI, which may be greater in participants from lower socioeconomic status households (see Chapter 4).

3.15 In contrast, NCMP (year 2020/21) indicated a strong and increasing relationship between prevalence of obesity (including severe obesity) and IMD (see Chapter 7, Figure 7.5, Figure 7.6 and Figure 7.7). Obesity prevalence increased with each IMD decile, from the least deprived (7.8%, in decile 10) to the most deprived decile (20.3%, in decile 1). For severe obesity, prevalence was 4 times higher in children living in the most deprived areas (7.6%) than those in the least deprived areas (1.9%).

Main dietary sources of energy

3.16 Main dietary sources of energy in children aged 12 to 60 months are presented in Table 3.5. Milk and cream were the largest source of energy at age 12 to 18 months, providing 19% of energy intake, while a further 10% came from infant formula. Milk and cream still provided 10% of energy at ages 48 to 60 months.

3.17 Cereals and cereal products were also an important source of energy: bread provided 7% of energy in the 12 to 18 month age group and 10% in the older groups. Biscuits, buns, cakes, pastries and puddings provided 6% of energy in the youngest age group, and 11% and 13% in children aged 18 to 47 months and 48 to 60 months, respectively.

3.18 Meat, meat products and dishes, vegetables and potatoes, fruit, and commercial toddler foods and drinks each provided 6 to 8% of energy intake at ages 12 to 18 months. By ages 18 to 47 months, meat, meat products and dishes provided 11% of energy intake while 3 to 8% came from each of vegetables and potatoes, fruit, and sugars, preserves and confectionery. For children aged 48 to 60 months, meat, meat products and dishes provided 13%, vegetables and potatoes provided 8% and fruit and sugars, preserves and confectionery each provided 5-6%.

Table 3.5. Contribution (% TDEI) of food groups (food sources) to average daily total dietary energy intake (MJ per day) for children aged 12 to 60 months (DNSIYC and NDNS years 2016/17 to 2018/19)[1]

Food groups[2,3,4]	12 to 18 months[1]		18 to 47 months[2]		48 to 60 months[2]	
	%	kcal	%	kcal	%	kcal
Milk and cream[5]	19.0	187	15.0	159	10.1	125
Infant formula[6]	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
Commercial toddlers foods and drinks	6.2	58	1.0	11	0.7	8
Biscuits, buns, cakes, pastries, 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
Yogurt, fromage, frais and dairy desserts[5]	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, products and dishes	2.7	26	2.8	29	3.2	38
Fat spreads	2.5	25	2.9	32	3.3	41
Cheese[5]	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 soft drinks	0.4	4	0.4	4	0.5	7.7
Ice cream[5]	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		354		114	

1 Data for children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013) otherwise data from NDNS years 2016/17 to 2018/19.

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 energy 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 dairy alternatives.

6 Infant formula consumed by children aged 18 months upwards are mainly follow on formula and growing up milks (see Annex 13, Glossary).

Evidence identified on dietary energy intake and body composition

3.19 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.

3.20 Details of the 6 SRs included in this section can be found in Annex 5 (Tables A5.1, A5.3 and A5.4). Quality assessment of the SRs using the AMSTAR 2 tool can be found in Annex 7 (Tables A7.2, A7.4, A7.6 and A7.7). Additional data extracted on the primary studies can be found in Annex 8 (Table A8.1).

3.21 The criteria used to grade the evidence are provided in Chapter 2 (Table 2.4, paragraphs 2.43 to 2.47). Summary tables of the evidence grading process for this section are provided in Annex 9 (Tables A9.1, A9.2 and A9.29).

Limitations of the evidence on dietary energy intake

3.22 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) directly 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 in this topic area may not have been comprehensive, which is a potential source of bias.

3.23 Most of the 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.

3.24 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).

3.25 Many of the studies were conducted in the 1990s indicating a possible need for more current research in this area.

Effect of portion sizes on food or energy intake

3.26 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 (interventions lasting for up to 3 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.

3.27 Ward et al (2015) (AMSTAR confidence rating: moderate) included a pre-post study (see Annex 13, 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; $p<0.01$).

3.28 Osei-Assibey et al (2012) (AMSTAR confidence rating: low) included 2 within-subject crossover studies (see Annex 13, 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% (± SEM 7%; $p<0.001$) and energy intake (kcal) by 15% (± 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]; $p<0.002$).

3.29 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 a choice to select from a number of smaller portions (statistics 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 decreasing the energy density of a dish (macaroni and cheese) served as part of a school lunch by 30% decreased 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 (statistics NR).

Dietary energy intake and BMI

3.30 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 sugars-sweetened beverages (SSBs), 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 (statistics NR). The study adjusted for children's exact age at 2 years and maternal age, pre-pregnancy BMI and education.

3.31 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.

3.32 One PCS (in 146 participants) reported no association between TDEI at ages 3 to 5 years and change in BMI 2 years later (statistics NR). The study adjusted for sex, age, baseline BMI, physical activity and parental weight status.

3.33 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 increases in 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.

3.34 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 did not adjust for potential confounding factors.

Dietary energy intake and body fat

3.35 Dougkas et al (2019) (AMSTAR 2 confidence rating: low) included 1 PCS that examined the relationship between energy intake from milk in children aged 1 to 5 years and body fat in later childhood. 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.

3.36 Parsons et al (1999) (AMSTAR 2 confidence rating: critically low) included an additional PCS (in 37 participants) in children aged 1 to 5 years, that 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 did not adjust for potential confounding factors.

Summary: dietary energy intake and body composition

3.37 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[1]	Certainty of evidence
Portion sizes	Food and energy intake	↑	Moderate
Dietary energy intake or energy density of the whole diet	BMI	N/A	Insufficient
Dietary energy intake or energy density of the whole diet	Body fat	N/A	Insufficient

Abbreviations: BMI, body mass index; N/A, not applicable.

[1] Direction of effect/association for reported outcomes: ↑increase; ↓inverse; N/A: not enough evidence to draw conclusions and recommendations.

3.38 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.

3.39 Evidence from 6 intervention studies included in 3 SRs by Ward et al (2015),Mikkelsen et al (2014) and Osei-Assibey et al (2012) Osei-Assibey et al (2011) suggests that increasing portion sizes of snacks and meals in preschool settings increases children's food and energy intake (in grams or energy) in the short term (interventions lasting for up to 3 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.

3.40 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 later childhood 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 have been comprehensive for dietary energy intake as an exposure, which is a potential source of bias.

3.41 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

3.42 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.

3.43 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).

3.44 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.

3.45 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 TDEI may mediate the effects of sugars-sweetened beverages (SSBs) 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 sugars-sweetened beverages (SSBs) by reducing energy consumption from foods (SACN, 2015).

3.46 Therefore, this draft report considered findings that were adjusted for TDEI separately from findings not adjusted for TDEI, when data was available.

Limitations of the evidence on macronutrients

3.47 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' dietary fat, protein or carbohydrate intakes.

3.48 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 Chapter 3, paragraphs 3.44 and 3.45).

3.49 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.

3.50 Other potential confounding factors that were not always accounted for by the SRs or primary studies when outcomes related to energy balance were under investigation included physical activity levels, parental weight status and SES.

3.51 Many primary studies either did not perform power calculations or did not report doing so. Findings of null associations with wide confidence intervals should therefore be interpreted with caution.

Carbohydrates

Classification of carbohydrates

3.52 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 (α or β) (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).

3.53 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.

3.54 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.

3.55 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.

3.56 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.

3.57 The following terms are used in this draft report to describe carbohydrates:

- Free sugars — all added sugars in any form; all sugars naturally present in fruit and vegetable juices, purées and pastes 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. The sugars naturally present in milk and dairy products, fresh and most types of processed fruit and vegetables and in cereal grains, nuts and seeds are excluded from the definition (Swan et al, 2018).

- Starch — polymer of glucose, found in foods such as rice, bread, pasta and potatoes
- Dietary fibre — defined in paragraph 3.54

3.58 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

3.59 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).

3.60 For children aged 2 years and older, it is 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
- sugars-sweetened beverages (SSBs) should be minimised
- the average population intake of dietary fibre for children aged 2 to 5 years should approximate 15g per day.

3.61 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 6 months of age, gradual diversification of the diet to provide increasing amounts of whole grains, pulses, vegetables and fruit was encouraged (SACN, 2015).

3.62 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 are not available.

Table 3.7. 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)[1] [at least]	145	134	198	184
Free sugars (grams per day)[2] [less than]	15	13	20	18
Dietary fibre (grams per day)	15	15	15[3] 20[4]	15[3] 20[4]

Source: (PHE, 2016a).

[1] Calculated using the energy figures from SACN (2011). The % for which to calculate grams of total carbohydrate per day (50% TDEI) was obtained from SACN (2015).

[2] 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).

[3] Applies at age 4 years.

[4] Applies at age 5 to 6 years.

Carbohydrate intakes in the UK

3.63 Total carbohydrate intake for children in the UK aged 12 to 60 months from DNSIYC and NDNS (years 2016/17 to 2018/19) are shown 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 2016/17 to 2018/19)[1]

Age	Grams per day Mean (SD)	% TDEI[2] 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.

[1] Data for children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013) otherwise data from NDNS years 2016/17 to 2018/19 (Bates et al, 2020).

[2] TDEI, total dietary energy intake. Total energy is equivalent to food energy as no alcohol is consumed by children of this age.

3.64 Time trend analysis for the age group 18 to 47 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 (2008/09 to 2018/19) (Bates et al, 2020). No time trend data was available for the other 2 age groups.

3.65 Sugars intake for children aged 12 to 60 months is shown in Table 3.9. Mean intake of free sugars (see paragraph 3.57) are presented for children aged 18 to 60 months but for children aged 12 to 18 months, intakes of non-milk extrinsic sugars (NMES) are presented because the survey data for this age group predated the definition of 'free sugars' (SACN, 2015). Although the definitions of 'free sugars' and 'NMES' are similar, the main difference between the 2 terms is that 'NMES' included 50% of the sugar from canned, stewed, dried or preserved fruit, while 'free sugars' excludes fruit other than that which is pureed, juiced or extruded (Roberts et al, 2018; Swan et al, 2018).

3.66 Mean intake of free sugars was almost double the maximum recommendation of 5% TDEI for children aged 18 to 47 months (9.7%) and more than double the maximum recommendation for children aged 48 to 60 months (11.7%). Eighty-five percent and 97% of children in both these age groups, respectively, had intakes above the 5% recommendation.

3.67 Time trend analysis of NDNS data (years 2008/09 to 2018/19) for children aged 18 to 47 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 2 age groups.

Table 3.9. Sugars intake in children aged 12 to 60 months in the UK (DNSIYC and NDNS years 2016/17 to 2018/19)[1]

Age	NMES[2] Grams per day[4]	NMES[2] % TDEI[3,4]	Free sugars[2] Grams per day[4]	Free sugars[2] % TDEI[3,4]	% participants exceeding DRV[5]	Number of participants
12 to 18 months	19.8 (12.1)	7.7 (4.5)	N/A	N/A	72	1275
18 to 47 months	N/A	N/A	27.9 (15.8)	9.7 (4.6)	85	306
48 to 60 months	N/A	N/A	38.9 (19.3)	11.7 (4.6)	97	102

Abbreviations: DRV, dietary reference value; NMES, non-milk extrinsic sugars; N/A, not applicable; SD, standard deviation; TDEI, total dietary energy intake.

[1] Data for children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013) otherwise data from NDNS years 2016/17 to 2018/19 (Bates et al, 2020).

[2] Sugar intakes for the age group 12 to 18 months were reported as non-milk extrinsic sugars (NMES). Sugar intakes for the 2 older age groups (18 to 47 months; 48 to 60 months) were reported as free sugars. [3] TDEI is equivalent to food energy as no alcohol is consumed by children of this age.

[4] Mean (SD).

[5] DRV: ≤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 NMES intake in the 12 to 18 month age group for illustrative purposes.

3.68 Dietary fibre intake for children aged 12 to 60 months is shown in Table 3.10. NDNS (years 2016/17 to 2018/19) used the definition of dietary fibre recommended by SACN (2015) (see paragraph 3.54) that is chemically determined using prevailing Association of Official Agricultural Chemists (AOAC) methods. DNSIYC used a narrower definition of dietary fibre, as non-starch polysaccharides (NSP), that predated SACN (2015) (DH, 1991; DH, 1994b).

3.69 Mean intake of AOAC fibre was 10.4 grams per day for children aged 18 to 47 months, and 12.6 grams per day for 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.

3.70 Time trend analysis of NDNS data (years 2008/09 to 2018/19) for children aged 18 to 48 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 2 age groups.

Table 3.10. Dietary fibre intake in children aged 12 to 60 months in the UK (DNSIYC and NDNS 2016/17 to 2018/19)[1]

Age	NSP[2,3] grams per day	AOAC fibre[2,3] grams per day	% participants below DRV[4]	Number of participants
12 to 18 months	7.3 (2.7)	N/A	N/A	1275
18 to 47 months	N/A	10.4 (3.5)	88	306
48 to 60 months	N/A	12.6 (4.7)	72	102

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

[1] Data for children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013) otherwise data from NDNS years 2016/17 to 2018/19 (Bates et al, 2020).

[2] 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.

[3] Mean (SD).

[4] 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

3.71 Intake of carbohydrates (by type) by IMD (See Annex 13, Glossary) for children aged 18 to 60 months from NDNS (years 2008/09 to 2018/19) are shown 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).

3.72 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 quintile 1 than in quintile 5, and dietary fibre intakes in quintiles 2 and 3 were also significantly higher than in quintile 5.

Table 3.11. Carbohydrate intakes by IMD quintile in children aged 18 to 60 months in England (NDNS years 2008/09 to 2018/19)

Intakes Mean (90% CI)	IMD quintile 1 (least deprived)	IMD quintile 2	IMD quintile 3	IMD quintile 4	IMD quintile 5 (most deprived)
Total carbohydrate % TDEI[2]	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[2]	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.

[1] Data from NDNS years 2008/09 to 2018/19.

[2] TDEI, total dietary energy intake. Total energy is equivalent to food energy as no alcohol is consumed by children of this age.

Main dietary sources of carbohydrates

Total carbohydrates

3.73 The main dietary sources of total carbohydrates for children aged 12 to 60 months are presented in Table 3.12. Milk and cream, bread and fruit were the largest contributors to carbohydrate intake for children aged 12 to 18 months, while bread, and baked goods (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 total carbohydrate intake for children aged 12 to 60 months (DNSIYC and NDNS years 2016/17 to 2018/19)[1]

Contribution of food groups[2,3,4] to total carbohydrate intake	12 to 18 months		18 to 47 months		48 to 60 months	
	%	grams per day	%	grams per day	%	grams per day
Milk and cream[5]	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
Infant formula[6]	9.1	11.4	1.1	1.4	0.0	0.0
Commercial toddlers foods and drinks	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, pies puddings	7.3	9.3	12.2	17.5	13.7	23.2
Yogurt, fromage frais and dairy desserts[5]	5.5	6.9	3.8	5.3	3.0	5.1
Potatoes, potato products 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, products 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 soft drinks	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[5]	0.4	0.5	1.1	1.6	2.2	3.7
Low calorie soft drinks	0.2	0.2	0.5	0.7	0.4	0.6
Cheese[5]	0.1	0.1	0.5	0.6	0.4	0.5
Number of participants	1275	1275	306	306	102	102

[1] Data for children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013) otherwise data from NDNS years 2016/17 to 2018/19 (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 total carbohydrate 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 dairy alternatives.
[6] Infant formula consumed by children aged 18 months upwards are mainly follow on formula and growing up milks (see Annex 13, Glossary).

Free sugars (and NMES)

3.74 The main dietary sources of free sugars for children aged 18 to 60 months and NMES for children aged 12 to 18 months are presented in Table 3.13. Yogurt, fromage frais and dairy desserts followed by baked goods (biscuits, buns, cakes, pastries, fruit pies) and puddings were the largest contributors to NMES intake for children aged 12 to 18 months. For older children, baked goods (biscuits, buns, cakes, pastries, fruit pies) and puddings, and sugar preserves and confectionery were the largest contributors, providing approximately 40% of free sugars intake for the 18 to 47 months age group and 48% for the 48 to 60 months age group.

3.75 In children aged 18 to 47 months, fruit juice and smoothies contributed 11% to free sugars intake while sugars-sweetened beverages contributed less than 3% to free sugars intake.

Table 3.13. Food group contributors to free sugars (and NMES) for children aged 12 to 60 months (DNSIYC and NDNS years 2016/17 to 2018/19)[1]

Contribution of food groups[2,3,4] to free sugars and NMES intake	12 to 18 months NMES[5]		18 to 47 months free sugars		48 to 60 months free sugars	
	%	grams per day	%	grams per day	%	grams per day
Yogurt fromage frais and dairy desserts[7]	24.6	4.2	11.8	2.9	7.5	2.9
Biscuits, buns, cakes, pastries, fruit pies puddings	16.5	3.2	22.7	6.3	25.5	9.2
Commercial toddlers foods and drinks	13.0	2.5	2.4	0.7	1.5	0.7
Sugar preserves and confectionery	12.3	2.6	17.7	5.1	22.6	9.4
Fruit	6.9	1.3	0.9	0.3	0.1	0.1
Breakfast cereals	5.5	1.0	8.0	1.8	6.9	2.4
Fruit juice and smoothies	5.1	1.2	10.5	3.6	6.6	3.1
Sugars-sweetened beverages	3.7	1.1	2.8	1.1	3.8	2.0
Vegetables, products and dishes	2.9	0.5	1.6	0.3	1.7	0.5
Infant formula[6]	2.1	0.6	2.8	0.8	0.0	0.0

Contribution of food groups[2,3,4] to free sugars and NMES intake	12 to 18 months NMES[5]		18 to 47 months free sugars		48 to 60 months free sugars	
	%	grams per day	%	grams per day	%	grams per day
Ice cream[7]	1.6	0.4	3.9	1.1	6.8	2.5
Low calorie soft drinks	1.3	0.2	3.0	0.6	2.3	0.6
Pizza, pasta, rice, products and dishes	1.2	0.2	2.0	0.5	1.3	0.4
Meat, meat products and dishes	0.7	0.1	2.5	0.6	3.0	0.8
Soup	0.6	0.1	0.3	0.1	0.3	0.2
Beverages dry weight	0.6	0.2	1.4	0.5	3.0	1.5
Savoury sauces pickles gravies and condiments	0.6	0.1	1.4	0.4	2.2	0.8
Milk and cream[6]	0.5	0.1	1.2	0.4	2.6	0.9
Bread	0.1	0.0	1.7	0.4	1.3	0.4
Crisps and savoury snacks	0.1	0.0	0.5	0.1	0.4	0.2
Number of participants	1275	1275	306	306	102	102

Abbreviations: NMES, non-milk extrinsic sugars.

[1] Data for children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013) otherwise data from NDNS years 2016/17 to 2018/19 (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] The definition of free sugars includes: all added sugars in any form; all sugars naturally present in fruit and vegetable juices, purées and pastes 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. Intakes for children aged 12 to 18 months are presented as NMES as DNSIYC pre-dated the definition of free sugars. The definition of NMES is similar to that of free sugars except that for NMES 50% of sugars in canned, stewed dried or preserved fruits was defined as extrinsic and 50% intrinsic.

[6] Infant formula consumed by children aged 18 months upwards are mainly follow on formula and growing up milks (see Annex 13, Glossary).

[7] Includes dairy alternatives.

Dietary fibre

3.76 The main dietary sources of dietary fibre for 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 categories.

Table 3.14. Food group contributors to dietary fibre intake for children aged 12 to 60 months (DNSIYC and NDNS years 2016/17 to 2018/19)[1]

Contribution of food groups[2,3,4] to dietary fibre[5] intake	12 to 18 months NSP		18 to 47 months AOAC fibre		48 to 60 months AOAC fibre	
	%	grams per day	%	grams per day	%	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
Commercial toddlers foods and drinks	9.8	0.7	1.6	0.2	1.1	0.1
Infant formula[6]	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 dishes	1.3	0.1	1.3	0.1	0.9	0.1
Yogurt, fromage frais and dairy desserts[7]	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

Contribution of food groups[2,3,4] to dietary fibre[5] intake	12 to 18 months NSP		18 to 47 months AOAC fibre		48 to 60 months AOAC fibre	
	%	grams per day	%	grams per day	%	grams per day
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 and cream[7]	0.1	0.0	0.4	0.1	0.4	0.1
Ice cream[7]	0.0	0.0	0.3	0.0	0.7	0.1
Cheese[7]	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 for children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013) otherwise data from NDNS years 2016/17 to 2018/19 (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] Infant formula consumed by children aged 18 months upwards are mainly follow on formula and growing up milk (see Annex 13, Glossary).

[7] Includes dairy alternatives.

Evidence identified on carbohydrate intake and health outcomes

3.77 Four SRs (Frantsve-Hawley et al, 2017; Gibson, 2008; Luger et al, 2017; Malik et al, 2006; Perez-Morales et al, 2013; Te Morenga et al, 2012), 1 with MA, were identified that examined the health impact of carbohydrate intake in childhood. An additional 3 SRs without MAs (Hörnell et al, 2013; Parsons et al, 1999; Tandon et al, 2016) included primary studies that examined the health impact of carbohydrates. 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.

3.78 Details of the SRs can be found in Annex 5 (Tables A5.1 and A5.3). Quality assessment of the SRs using the AMSTAR 2 tool can be found in Annex 7 (Tables A7.2 and A7.5). Additional data extracted on the primary studies can be found in Annex 8 (Tables A8.2 to A8.3). The criteria used to grade the evidence are provided in Chapter 2 (Table 2.4, paragraphs 2.43 to 2.47). Summary tables of the evidence grading process for this section are provided in Annex 9 (Tables A9.3, A9.4 and Table A9.29).

3.79 Key exposures were total carbohydrates (Hörnell et al, 2013), dietary sugars (Te Morenga et al, 2012) and sugars-sweetened beverages (SSBs) (Frantsve-Hawley et al, 2017; Luger et al, 2017; Perez-Morales et al, 2013; Tandon et al, 2016; Te Morenga et al, 2012). To be consistent with the SACN report 'Carbohydrates and Health' (SACN, 2015), the evidence identified on SSBs is presented in this chapter while the evidence on fruit juice (if analysed separately from SSBs by the SRs, see paragraph 3.84) is presented in Chapter 5 ('Foods, dietary components and dietary Patterns').

3.80 No evidence from SRs was identified on the health impact of dietary fibre in children aged 1 to 5 years.

3.81 Key outcomes examined 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.

3.82 The majority of primary studies included in the SRs were from high income countries (HIC).

Limitations of the evidence on carbohydrates

3.83 The evidence identified on the health impact of total carbohydrate intake in young children is from SRs that did not actively consider this as part of their research question. Therefore, the literature searches conducted by these SRs would not have been comprehensive for total carbohydrate intake as an exposure, which is a potential source of bias.

3.84 'Sugars-sweetened beverage' was defined differently in each SR that included this as an exposure. In Frantsve-Hawley et al (2017), SSBs included all sugars-sweetened (non-dairy) beverages and 100% fruit juice. In Te Morenga et al (2012), SSB was not defined but was identified separately from fruit juice (also not defined). In Luger et al (2017), SSBs included soft drinks, fruit juice drinks (not defined), syrup-based drinks, flavoured water with sugar and sports drinks. In Perez-Morales et al (2013), SSBs included soft drinks, soda, fruit drinks (not defined), sports drinks, sweetened iced tea and lemonade. In Tandon et al (2016), SSBs included soft drinks, cordial and fruit drinks (not defined).

3.85 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 3.45).

Carbohydrates and body composition or weight status

Total carbohydrate intake and body composition

3.86 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).

3.87 For this draft 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 in later childhood (Hörnell et al, 2013; Parsons et al, 1999).

3.88 Overall, the PCS included in the SRs that adjusted their findings for TDEI reported a null association between total carbohydrate intake and BMI, whereas those that did adjust for TDEI reported an inverse association between total carbohydrate intake and BMI.

3.89 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 (statistics NR), unadjusted for TDEI. The study adjusted for multiple key confounding factors (sex, baseline child BMI, parental BMI and a measure of sedentary behaviour).

3.90 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

3.91 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 (statistics NR). The study adjusted for TDEI, baseline child BMI, parental BMI and SES.

Summary: total carbohydrate intake and body composition

3.92 The evidence identified from SRs on total carbohydrate intake and body composition is summarised in Table 3.15.

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

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Total carbohydrate intake	BMI	N/A	Insufficient
Total carbohydrate intake	Body fat	N/A	Insufficient

Abbreviations: BMI, body mass index; N/A, not applicable.
[1] N/A: not enough evidence to draw conclusions and recommendations.

3.93 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.

3.94 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 conflicting. As carbohydrate intake was neither an exposure nor included in the search terms of either SR, their literature searches would not have been 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 Chapter 3, paragraphs 3.44 and 3.45), the evidence was graded **insufficient**.

3.95 There was also **insufficient** evidence 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.

Sugars-sweetened beverages and body composition or weight status

3.96 The SACN report 'Carbohydrates and Health' 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).

3.97 For this draft report, 1 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) or risk (RR) of overweight or obesity, and changes in body composition (BMI, BMI z-score, weight-for-height z-score) over time.

Sugars-sweetened beverages and odds or risk of overweight or obesity in later childhood

3.98 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 in later childhood.

3.99 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 an increased 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.

3.100 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 >65ml per day of SSBs had an increased odds of being overweight 30 months later compared with children who consumed <65ml 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.

3.101 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.

Sugars-sweetened beverages and change in BMI or WHZ

3.102 Two SRs without Mas (Frantsve-Hawley et al, 2017; Luger et al, 2017) examined the relationship between SSB consumption and change in BMI/BMI z-score or weight-for-height z-score (WHZ) in children.

3.103 Overall, the PCS included in the 2 SRs that reported an association between higher SSB consumption and greater change in BMI (or BMI z-score) or WHZ did not adjust for TDEI, while most of the PCS that reported no association did adjust for TDEI.

3.104 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). The other 2 PCS (in a total of 1381 participants) reported no association.

3.105 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.

3.106 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.

3.107 The PCS that reported an association did not adjust for baseline BMI and therefore reverse causality cannot be ruled out (see paragraph 3.49).

3.108 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.

Sugars-sweetened beverages and body fat

3.109 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 in later childhood. 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: sugars-sweetened beverage consumption and body composition or weight status

3.110 The evidence on SSB consumption and body composition or weight status is summarised in Table 3.16.

Table 3.16. Summary of the evidence on SSB consumption and body composition or weight status

Exposure	Outcome	Direction of association[1]	Certainty of evidence
SSB consumption	Odds of overweight or obesity (mostly adjusted for TDEI)	↑	Adequate
SSB consumption	Change in weight status[2] (unadjusted for TDEI)	↑	Moderate
SSB consumption	Body fat	N/A	Insufficient

Abbreviations: N/A, not applicable; SSB, sugars-sweetened beverage; TDEI, total energy intake.
[1] Direction of association for reported outcomes: ↑increase; ↓inverse; N/A: not enough evidence to draw conclusions and recommendations.
[2] Change in BMI, BMI z-score, weight-for-height z-score.

3.111 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.

3.112 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 an increased odds of overweight or obesity in later 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.

3.113 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 later childhood, unadjusted for TDEI. 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).

3.114 Together with the findings from Te Morenga et al (2012) (paragraph 3.112), 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 3.45) and partially independent of its contribution to increasing TDEI.

3.115 There was **insufficient** evidence 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.

Carbohydrates and other health outcomes

Sugars-sweetened beverages and cognitive development

3.117 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 in later childhood. 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 (statistics 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: sugars-sweetened beverages and cognitive development

3.118 The evidence on SSB intake and cognitive development is summarised in Table 3.17.

Table 3.17. Summary of the evidence on carbohydrate intake and cognitive development

Exposure	Outcome	Direction of association[1]	Certainty of evidence
SSB consumption	Cognitive development	N/A	Insufficient

Abbreviations: N/A, not applicable; SSB, sugars-sweetened beverage.
[1] N/A: not enough evidence to draw conclusions and recommendations.

3.119 The available evidence from SRs on the relationship between SSB consumption and cognitive development is from 1 SR without MA, given a critically low confidence rating using the AMSTAR 2 tool.

3.120 There was **insufficient** evidence 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.

3.121 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.

3.122 No evidence from SRs was identified on the relationship between carbohydrate intake and any other health outcomes in children aged 1 to 5 years.

Dietary fat

3.124 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).

3.125 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

3.126 The DRVs for dietary fat are shown in Table 3.18. These currently do not apply before age 2 years and apply in full from age 5 years (DH, 1994a). A flexible approach is 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).

3.127 For the purposes of assessing the nutritional intake of young children in the UK, the recommendations for children aged 5 years and older have been applied to children aged under 5 years. Table 3.19 shows the DRVs for dietary fats (DH, 1991; DH, 1994b; SACN, 2019) for 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 for children aged 1 to 3 years are not available.

Table 3.18. UK government dietary recommendations for dietary fat[1] for adults and children aged 5 years and older

Dietary fat	DRV (population average)
Total fats[4]	33% total dietary energy
Saturated fatty acids (saturated fats)[3]	10% of total dietary energy
Monounsaturated fatty acids (MUFA)[4]	No specific recommendations for MUFA[5]
n-6 polyunsaturated fatty acids (n-6 PUFA)[4]	6% total dietary energy[6]
Linoleic acid[2]	Provide at least 1% of total energy

Dietary fat	DRV (population average)
Long chain n-3 PUFA[7]	Increase from 0.2 grams per day to 0.45 grams per day[8]
Alpha linolenic acid (ALA)[2]	Provide at least 0.2% of total energy
Trans fats[4]	Provide no more than about 2% of dietary energy

[1] Values are expressed as proportions of either total (dietary) energy or dietary energy, depending on the source report.
[2] From DH (1991).
[3] From SACN (2019).
[4] From (DH, 1994a)
[5] 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.
[6] 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.
[7] 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.
[8] 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 3.19. DRVs for dietary fat for children aged 4 to 6 years in grams per day[1]

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

[1] Source from PHE (2016b), 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% food 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

3.128 Total fat intake for children aged 12 to 60 months in the UK from DNSIYC and NDNS (years 2016/17 to 2018/19) is shown in Table 3.20. Mean intake as % TDEI was highest (35%) in the youngest age group (12 to 18 months) and lowest (34%) in the oldest age group (48 to 60 months). Although the DRV for total fat applies from age 5 years onwards, it is notable that 45% of children aged 4 to 5 years (48 to 60 months) had intakes above it.

3.129 Time trend analysis of NDNS data (years 2008/09 to 2016/17) for children aged 18 to 47 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 2 age groups.

Table 3.20. Total fat intake in children aged 12 to 60 months in the UK (DNSIYC and NDNS years 2016/17 to 2018/19)[1]

Age	Grams per day[2]	% TDEI[2,3]	% participants above DRV[4]	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.

[1] Data for children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013), otherwise data from NDNS years 2016/17 to 2018/19

[2] Mean (SD).

[3] TDEI is equivalent to food energy as no alcohol is consumed in children of this age.

[4] 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

3.130 Saturated fatty acids (saturated fat) intake for children aged 12 to 60 months is shown in Table 3.21. Mean saturated fat intake was 16% TDEI for children aged 12 to 18 months, 15% for children aged 18 to 47 months and 14% for children aged 48 to 60 months. For the oldest age group (48 to 60 months), 91% of children had intakes above the DRV. It should be noted that the DRV of no more than 10% TDEI from saturated fats applies in full to children from 5 years of age.

3.131 Time trend analysis of NDNS data (years 2008/09 to 2018/19) for children aged 18 to 47 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 2 age groups.

Table 3.21. Saturated fat intakes in children aged 12 to 60 months in the UK (DNSIYC and NDNS 2016/17 to 2018/19)[1]

Age	Grams per day[2]	% TDEI[2,3]	% participants exceeding DRV[4]	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

[1] Data for children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013), otherwise data from NDNS years 2016/17 to 2018/19 (Bates et al, 2020).

[2] Mean (SD).

[3] TDEI is equivalent to food energy as no alcohol is consumed in children of this age.

[3] 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

3.132 Mean intake of cis monounsaturated fatty acids (cis MUFA) for children aged 12 to 60 months was approximately 12% TDEI (see Table 3.22). There is no specific UK recommendation for cis MUFAs.

3.133 Time trend analysis of NDNS data (years 2008/09 to 2018/19) for children aged 18 to 47 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 2 age groups.

Table 3.22. Cis monounsaturated fatty acids (cis MUFA) intakes in children aged 12 to 60 months in the UK (DNSIYC and NDNS 2016/17 to 2018/19)[1]

Age	Grams per day[2]	% TDEI[2,3]	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.

[1] Data for children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013), otherwise data from NDNS 2016/17 to 2018/19 (Bates et al, 2020).
[2] Mean (SD).
[3] TDEI is equivalent to food energy as no alcohol is consumed in children of this age.

Polyunsaturated fatty acids

3.134 Mean intake of cis n-3 polyunsaturated fatty acids (cis n-3 PUFA) for 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 5% TDEI (see Table 3.23).

3.135 Time trend analysis of NDNS data (years 2008/09 to 2018/19) for children aged 18 to 47 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 2 age groups.

Table 3.23. 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 2016/17 to 2018/19)[1]

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

Abbreviations: TDEI, total dietary energy intake.
[1] Data for children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013) otherwise data from NDNS 2016/17 to 2018/19 (Bates et al, 2020).
[2] Mean (SD).
[3] TDEI is equivalent to food energy as no alcohol is consumed in children of this age.

Trans fatty acids

3.136 Mean intake of trans fatty acids for children aged 12 to 60 months was 0.5% TDEI in each age group (Table 3.24). No children in any age group exceeded the recommendation of no more than 2% TDEI from trans fatty acids.

3.137 Time trend analysis of NDNS data (years 2008/09 to 2016/17) for children aged 18 to 47 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 2 age groups.

Table 3.24. Trans fat intakes in children aged 12 to 60 months in the UK (DNSIYC and NDNS years 2016/17 to 2018/19)[1]

Age	Grams per day[2]	% TDEI[2,3]	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.

[1] Data for children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013) otherwise data from NDNS 2016/17 to 2018/19 (Bates et al, 2020).

[2] Mean (SD).

[3] TDEI is equivalent to food energy as no alcohol is consumed in children of this age.

Dietary fat intakes and deprivation

3.138 Dietary fat intakes by IMD (See Annex 13, Glossary) for children aged 18 to 60 months are shown in Table 3.25. Data from NDNS years 2016/17 to 2018/19 indicated that there was no relationship between total fat and saturated fat intakes and IMD quintile. For cis MUFA and cis PUFA, the data suggested slightly higher intakes with increasing deprivation but any differences in intakes between quintiles were not statistically significant (as indicated by overlapping confidence intervals).

Table 3.25. Dietary fat intakes by IMD quintile in children aged 18 to 60 months in England (NDNS years 2008/09 to 2018/19)[1]

Intakes % TDEI[2]	IMD quintile 1 (least deprived)	IMD quintile 2	IMD quintile 3	IMD quintile 4	IMD quintile 5 (most deprived)
Total fat Mean (90% 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 (90% 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 (90% 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 (90% 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 (90% 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: TDEI, total dietary energy intake.

[1] Data from NDNS 2008/09 to 2018/19 (Bates et al, 2020).

[2] TDEI is equivalent to food energy as no alcohol is consumed by children of this age.

Main dietary sources of dietary fat

Total fat

3.139 The main dietary sources of total dietary fat for children aged 12 to 60 months are presented in Table 3.26. Milk and cream followed by infant formula were the largest contributors to total fat intake for the youngest age group (age 12 to 18 months) while for the 2 older age groups, meat (including meat products and dishes) and baked goods (biscuits, buns, cakes, pastries, fruit pies) and puddings made substantial contributions.

Table 3.26. Food group contributors to total dietary fat intake for children aged 12 to 60 months (DNSIYC and NDNS years 2016/17 to 2018/19)[1]

Contribution of food groups[2,3,4] to total fat intake	12 to 18 months		18 to 47 months		48 to 60 months	
	%	Grams per day	%	Grams per day	%	Grams per day
Milk and cream[5]	27.0	10.7	20.7	8.9	12.4	5.9
Infant formula[6]	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, pies and puddings	6.0	2.3	11.0	4.5	14.4	6.5
Cheese[5]	5.2	2.1	6.2	2.5	4.3	2.1
Yogurt, fromage frais and dairy desserts[5]	3.9	1.4	2.8	1.2	2.4	1.1
Commercial toddlers foods and drinks	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, 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[5]	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	306

[1] Data for children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013) otherwise data from NDNS years 2016/17 to 2018/19 (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 dairy alternatives.

[6] Infant formula consumed by children aged 18 months upwards are mainly follow on formula and growing up milks (see Annex 13, Glossary).

Saturated fatty acids

3.140 The main dietary sources of saturated fatty acids (saturated fats) for children aged 12 to 60 months are presented in Table 3.27. Milk and cream and infant formula contributed 45% to saturated fat intake for children aged 12 to 18 months. For the 2 older age groups, milk and cream remained the highest contributor to saturated fat intake, followed by meat (including meat products and dishes) and baked goods (biscuits, buns, cakes, pastries, fruit pies) and puddings.

Table 3.27 Food group contributors to saturated fat intake for children aged 12 to 60 months (DNSIYC and NDNS 2016/17 to 2018/19)[1]

Contribution of food groups[2,3,4] to saturated fat intake	12 to 18 months		18 to 47 months		48 to 60 months	
	%	Grams per day	%	Grams per day	%	Grams per day
Milk and cream[5]	34.3	6.6	28.5	5.4	18.6	3.6
Infant formula[6]	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[5]	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, pies and puddings	6.0	1.0	11.8	2.0	16.6	2.9
Yogurt, fromage frais and dairy desserts[5]	5.6	0.9	4.2	0.7	3.6	0.7
Commercial toddlers foods and drinks	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

Contribution of food groups[2,3,4] to saturated fat intake	12 to 18 months		18 to 47 months		48 to 60 months	
	%	Grams per day	%	Grams per day	%	Grams per day
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[5]	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

[1] Data for children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013) otherwise data from NDNS years 2016/17 to 2018/19 (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 dairy alternatives.
[6] Infant formula consumed by children aged 18 months upwards are mainly follow on formula and growing up milks (see Annex 13, Glossary).

Monounsaturated fatty acids

3.141 The main dietary sources of cis MUFA for children aged 12 to 60 months are presented in Annex 10, Table A10.3. Milk and cream and infant formula contributed over 45% to cis MUFA intake in children aged 12 to 18 months. For children aged 18 to 47 months, meat (including meat products and dishes) was the highest contributor to cis MUFA intake, followed by milk and cream and baked goods (biscuits, buns, cakes, pastries, fruit pies) and puddings. For 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

3.142 The main dietary sources of cis n-3 PUFA and cis n-6 PUFA for children aged 12 to 60 months are presented in Annex 10, Tables A10.4 and A10.5, respectively. For children aged 12 to 18 months, infant formula, butter and fat spreads, meat (including meat products and dishes) and milk and cream were the largest contributors to n-3 PUFA intake. For 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), baked goods (biscuits, buns, cakes, pastries, pies) and puddings was also a large contributor (>10%).

3.143 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. For the 2 older age groups, meat (including meat products and dishes) followed by baked goods (biscuits, buns, cakes, pastries, pies) and puddings were major contributors to n-6 PUFA intake.

Trans fatty acids

3.144 The main dietary sources of trans fatty acids for children aged 12 to 60 months are presented in Annex 10, Table A10.6. Milk and cream, meat (including meat products and dishes) and cheese were the largest contributors to intakes of trans fatty acids. For the oldest age group (age 48 to 60 months), butter and fat spreads as well as baked goods (biscuits, buns, cakes, pastries, pies) and puddings were also major contributors to intake of trans fatty acids.

Evidence identified on dietary fat intake and health outcomes

3.146 Two SRs without MAs (Naude et al, 2018; Voortman et al, 2015a) were identified that examined the health impact of total fat intake 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.

3.147 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 7 (Table A7.2). The criteria used to grade the evidence are provided in Chapter 2 (Table 2.4, paragraphs 2.43 to 2.47). Additional data extracted from the primary studies can be found in Annex 8 (Table A8.4 to A8.7). The criteria used to grade the evidence are provided in chapter 2 (Table 2.4, paragraphs 2.43 to 2.46). Summary tables of the evidence grading process for this section are provided in Annex 9 (Table A9.5 and Table A9.29).

3.148 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 draft report (May 2019). Evidence related to saturated fat intake in children included in SACN's 2019 report (2019) 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.

3.149 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.

3.150 Key outcomes examined were measures of body composition (BMI, body weight and body fat), blood lipids, blood pressure and linear growth.

3.151 The majority of primary studies included in the identified SRs were from high income countries (HIC).

Dietary fat intake and body composition or weight status

Total fat intake and BMI or body weight

3.152 Two SRs without MAs were identified that examined the relationship between total fat intake and body weight or BMI in children (Naude et al, 2018; Parsons et al, 1999).

3.153 Naude et al (2018) (AMSTAR 2 confidence rating: low) 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

3.154 The outcomes examined in the 4 shorter term PCS were body weight (1 study), BMI (2 studies) or both (1 study).

3.155 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 (≤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 0.66kg 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.

3.156 Of the 3 PCS that examined change in BMI as an outcome, 1 PCS (in 146 participants) reported that for every 1% increase in dietary energy intake from total fat at age 3 to 5 years, BMI increased by 0.034kg/m^2 (95% CI NR) when measured 2 years later (p=0.05). The study adjusted for TDEI, and several key confounding factors (sex, age, baseline BMI and physical activity and parental BMI).

3.157 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 0.02kg/m^2 per year (95% CI -0.26 to 0.30; p>0.05) between children with lower fat intakes (≤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), statistics were NR. The study adjusted for TDEI, and key confounding factors (sex, ethnicity, baseline BMI, physical activity, and parental weight status).

Longer-term studies

3.158 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^2 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.

3.159 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

3.160 Two SRs without MAs examined the relationship between total fat intake and body fat (Naude et al, 2018; Parsons et al, 1999).

3.161 Naude et al (2018) (AMSTAR 2 confidence rating: low) 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 of 0.62% (SE 0.26; p=0.02) and total body fat of 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).

3.162 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

3.163 The evidence identified from SRs on total fat intake or body composition and weight status is summarised in Table 3.28.

Table 3.28. Summary of the evidence on the relationship between total fat intake and body composition or weight status

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Total fat intake	Change in BMI or body weight (shorter-term)	Null	Limited
Total fat intake	BMI or change in BMI (longer-term)	N/A	Insufficient
Total fat intake	Body fat	N/A	Insufficient

Abbreviations: BMI, body mass index; N/A, not applicable; TDEI, total dietary energy intake.
[1] Direction of association for reported outcomes: ↑increase; ↓inverse; N/A: not enough evidence to draw conclusions and recommendations.

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

3.165 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.

3.166 There was **insufficient** evidence 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.

3.167 There was also **insufficient** evidence 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

3.168 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, 1 RCT was conducted in children aged 1 to 5 years (% weighting of the MA NR).

3.169 No additional SRs were identified on saturated fats and obesity outcomes in children.

PUFA intake and body composition or weight status

3.170 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

3.171 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 becoming 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.

3.172 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 90th centile (defined as overweight in the study) at age 5 years ($p=0.06$) in unadjusted analyses.

PUFA intake and body fat

3.173 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 in later childhood. 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

3.174 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.

3.175 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) (statistics NR).

3.176 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 (statistics NR). The study adjusted for birth weight, breastfeeding duration and maternal BMI.

n-3 PUFA and body fat

3.177 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

3.178 The evidence identified from SRs on PUFA intake and body composition or weight status is summarised in Table 3.29.

Table 3.29. Summary of the evidence on PUFA intake and body composition or weight status

Exposure	Outcome	Direction of association[1]	Certainty of evidence
PUFA intake	Overweight	N/A	Insufficient
PUFA intake	Body fat	N/A	Insufficient
n-3 PUFA intake	BMI or BMI z-score	N/A	Insufficient
n-3 PUFA intake	Body fat	N/A	No evidence identified

Abbreviations: BMI, body mass index; N/A, not applicable; PUFA, polyunsaturated fatty acids.
[1] N/A: not enough evidence to draw conclusions and recommendations.

3.179 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.

3.180 There was **insufficient** evidence 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

3.181 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

3.182 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

3.183 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.

3.184 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).

3.185 Reduced intake of saturated fats lowered serum LDL-C using a random-effects model (statistics 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.

3.186 There was no effect of reduced intake of saturated fats on serum HDL-C (statistics 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).

3.187 There was no effect of reduced intake of saturated fats on triacylglycerol (statistics 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).

3.188 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).

3.189 No additional SRs were identified on saturated fats and blood lipids in children.

PUFA intake and blood lipids

3.190 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

3.191 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 (statistics 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 (statistics 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

3.192 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 (statistics NR) in univariate regression analyses.

Serum HDL cholesterol

3.193 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 (-0.15mmol/l; 95% CI -0.29 to -0.01mmol/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).

3.194 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

3.195 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

3.196 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

3.197 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 (statistics NR).

Serum triacylglycerol

3.198 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 (statistics NR).

Summary: dietary fat intake and blood lipids

3.199 The evidence identified from SRs on dietary fat intake and blood lipids is summarised in Table 3.30.

Table 3.30. Summary of the evidence on dietary fat intake and blood lipids

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Total fat	Blood lipids (all)	N/A	No evidence identified
Saturated fats	Blood lipids (all)	N/A	No additional evidence identified[2]
PUFA intake	TC	N/A	Insufficient
PUFA intake	LDL-C	N/A	Insufficient
PUFA intake	HDL-C	N/A	Insufficient
PUFA intake	Triacylglycerol	N/A	No evidence identified
n-3 PUFA intake	TC	N/A	No evidence identified
n-3 PUFA intake	LDL-C	N/A	No evidence identified
n-3 PUFA intake	HDL-C	N/A	Insufficient
n-3 PUFA intake	Triacylglycerol	N/A	Insufficient

Abbreviations: HDL-C, high-density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; N/A, not applicable; PUFA, polyunsaturated fatty acids; TC, total cholesterol.

[1] N/A: not enough evidence to draw conclusions and recommendations.

[2] Since the SACN report 'Saturated fats and health' (SACN, 2019).

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

3.201 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.

3.202 There was **insufficient** evidence 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

3.203 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 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

3.204 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

3.205 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.

3.206 There was no effect of reducing saturated fats on systolic blood pressure (SBP) (statistics 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).

3.207 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).

3.208 No additional SRs were identified on saturated fats and blood lipids in children.

PUFA intake and blood pressure

3.209 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).

3.210 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

3.211 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

3.212 The evidence identified from SRs on dietary fat intake and obesity outcomes is summarised in Table 3.31.

Table 3.31. Summary of the evidence on dietary fat intake and blood pressure

Exposure	Outcome	Direction of effect or association[1]	Certainty of evidence
Total fat intake	Blood pressure	N/A	No evidence identified
Saturated fat intake	Blood pressure	N/A	No additional evidence identified[2]
PUFA intake	Blood pressure	N/A	Insufficient
n-3 PUFA intake	Blood pressure	N/A	Insufficient

Abbreviations: N/A, not applicable; PUFA, polyunsaturated fatty acids.
[1] N/A: not enough evidence to draw conclusions and recommendations.
[2] Since the SACN report 'Saturated fats and health' (SACN, 2019).

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

3.214 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.

3.215 There was **insufficient** evidence 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

3.216 In this draft 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

3.217 Two SRs without MAs (Hörnell et al, 2013; Naude et al, 2018) examined the relationship between total fat intake and linear growth.

3.218 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.

3.219 Naude et al (2018) (AMSTAR 2 confidence rating: low) 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.

Saturated fat intake and linear growth

3.220 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

3.221 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

3.222 The evidence identified from SRs on dietary fat intake and linear growth is summarised in Table 3.32.

Table 3.32. Summary of the evidence on dietary fat intake and linear growth

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Total fat intake	Age at PGV	N/A	Insufficient
Total fat intake	Linear growth	N/A	Insufficient
Saturated fat intake	Linear growth	N/A	No evidence identified[2]
PUFA intake	Linear growth	N/A	No evidence identified
n-3 PUFA intake	Linear growth	N/A	No evidence identified

Abbreviations: N/A; not applicable; PGV, peak growth velocity; PUFA, polyunsaturated fatty acids.
[1] N/A: not enough evidence to draw conclusions and recommendations.
[2] Since the SACN report 'Saturated fats and health' (SACN, 2019).

3.223 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 moderate confidence rating using the AMSTAR 2 tool, the other given a low confidence rating.

3.224 There was **insufficient** evidence 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.

3.225 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

3.226 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

3.227 Dietary proteins are necessary for tissue growth and maintenance (EFSA, 2015a).

3.228 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).

3.229 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, 2007). 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.

3.230 The DRVs set by DH (1991) and EFSA (2015a) are presented in Table 3.33, while the values from which the DRVs set by each body were derived are presented in Table 3.34. Table 3.34 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 3.33. 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)[1]	EFSA (2012) PRI (grams per day)[2] Boys	EFSA (2012) PRI (grams per day)[2] 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.

[1] 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.

[2] Data from EFSA (2015a).

Table 3.34. Safe level of protein intake[1] for children aged 1 to 5 years in the WHO 1985 and 2007 reports

Age (years)	WHO 1985[2]	WHO 2007[3]
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

[1] 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, 2007).

[2] Data from (WHO, 1985) on which the COMA DRVs for protein (DH, 1991) were derived.

[3] Data from (WHO, 2007) on which the EFSA DRVs for protein (EFSA, 2015a) were derived.

Protein intakes in the UK

3.231 Protein intake for children aged 12 to 60 months in the UK from DNSIYC and NDNS (years 2016/17 to 2018/19) is shown in Table 3.35.

Table 3.35. Protein intakes in children aged 12 to 60 months in the UK (DNSIYC and NDNS 2016/17 to 2018/19)[1]

Age	Grams per day[2]	% TDEI[2,3]	% 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.

[1] Data for children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013), otherwise data from NDNS 2016/17 to 2018/19 (Bates et al, 2020).

[2] Mean (SD).

[3] TDEI is equivalent to food energy as no alcohol is consumed in children of this age.

3.232 Mean protein intake in children aged 12 to 18 months was approximately 38 grams per day, more than 2.5 times the RNI (14.5 grams per day) and more than 3 times the PRI (Table 3.33). In children aged 18 to 48 months, mean protein intake was 41 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.

3.233 Time trend analysis of NDNS data (years 2008/09 to 2016/17) for children aged 18 to 47 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 (PHE, 2019). No time trend data was available for the other 2 age groups.

Protein intake and deprivation

3.234 Protein intake by IMD (see Annex 13, Glossary) is shown in Table 3.36. 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 3.36. Protein intakes by IMD quintile in children aged 18 to 60 months in England (NDNS years 2008/09 to 2018/19)[1]

Protein	IMD quintile 1 (least deprived)	IMD quintile 2	IMD quintile 3	IMD quintile 4	IMD quintile 5 (most deprived)
Grams per day Mean (90% 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[2] Mean (90% 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: RNI, reference nutrient intake; TDEI, total dietary energy intake.
[1] Data from NDNS years 2008/09 to 2018/19 (Bates et al, 2020).
[2] TDEI is equivalent to food energy as no alcohol is consumed by children of this age.

Main dietary sources of protein

3.235 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).

3.236 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 bread and cereals, legumes and nuts. 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).

3.237 The main dietary sources of protein for children aged 12 to 60 months in the UK are presented in Table 3.37. Milk and cream followed by meat (and meat products and dishes) were the largest contributors to protein intake for children aged 12 to 18 months. In children aged 48 to 60 months, meat (including meat products and dishes) was the largest contributor to protein intake followed by milk and cream.

Table 3.37. Food group contributors to protein intake for children aged 12 to 60 months (DNSIYC and NDNS years 2016/17 to 2018/19)[1]

Contribution of food groups to protein intake[2,3,4]	12 to 18 months		18 to 47 months		48 to 60 months	
	%	Grams per day	%	Grams per day	%	Grams per day
Milk and cream[5]	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
Yogurt, fromage frais and dairy desserts[5]	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
Infant formula[6]	6.6	2.0	0.8	0.3	0.0	0.0
Commercial toddlers foods and drinks	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, 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[5]	3.9	1.5	5.0	2.0	3.6	1.7
Vegetables, 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[5]	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

[1] Data for children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013), otherwise data from NDNS years 2016/17 to 2018/19 (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 dairy alternatives.
[6] Infant formula consumed by children aged 18 months upwards are mainly follow on formula and growing up milks (see Annex 13, Glossary).

Evidence identified on protein intake and health outcomes

3.238 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.

3.239 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 A7.2). Additional data extracted on the primary studies can be found in Annex 8 (Table A8.8 to A8.11). The criteria used to grade the evidence are provided in chapter 2 (Table 2.4, paragraphs 2.43 to 2.47). Summary tables of the evidence grading process for this section are provided in Annex 9 (Tables A9.6, A9.7 and A9.29).

3.240 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).

3.241 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

Protein intake and body composition or weight status

3.242 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).

3.243 Some researchers consider the first 5 years of life to be a ‘critical period’ for protein intake and later adiposity (Gunther et al, 2007).

3.244 This draft report examined the evidence from SRs on protein intake in children aged 1 to 5 years and obesity outcomes in later childhood.

Total protein intake and later BMI and overweight

3.245 Two SRs without MAs examined the relationship between total protein intake and BMI (Hörnell et al, 2013; Parsons et al, 1999).

3.246 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.

3.247 Of the 4 PCS, 2 PCS also reported an association between higher total protein intake in early childhood and later overweight.

3.248 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 >75th percentile of German reference curves. The analysis adjusted for TDEI, sex, baseline BMI SDS and SES.

3.249 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 1 year, other macronutrients (% TDEI), parental age and weight status. Overweight was defined as having a BMI >90th percentile of age- and sex-adjusted curves created by Rolland-Cachera et al (1982).

3.250 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

3.251 Two SRs without MAs examined the relationship between total protein intake and body fat (Hörnell et al, 2013; Parsons et al, 1999).

3.252 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 75th 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.

3.253 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

3.254 Two SRs without MAs examined the relationship between animal protein intake and BMI or body weight (Hörnell et al, 2013; Parsons et al, 1999).

3.255 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.

3.256 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 (statistics NR).

Animal protein intake and body fat

3.257 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

3.258 Hörnell et al (2013) included 1 PCS (described in paragraph 3.257) that reported no association between vegetable protein intake (as % TDEI) at age 1 year and BMI at 7 years (statistics NR) in adjusted analyses.

Vegetable protein intake and body fat

3.259 Hörnell et al (2013) included 1 PCS (described in paragraph 3.257) that reported no association between vegetable protein intake (as % TDEI) at age 1 year and % body fat at 7 years (statistics NR) in adjusted analyses.

Summary: protein intake and body composition or weight status

3.260 The evidence identified from SRs on protein intake and body composition or weight status is summarised in Table 3.38.

Table 3.38. Summary of the evidence on protein intake and body composition or weight status

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Total protein intake	BMI	↑	Moderate
Total protein intake	Overweight	N/A	Insufficient
Total protein intake	Body fat	N/A	Insufficient
Animal protein intake	BMI	N/A	Insufficient
Animal protein intake	Body fat	N/A	Insufficient
Vegetable protein intake	BMI	N/A	Insufficient
Vegetable protein intake	Body fat	N/A	Insufficient

Abbreviations: BMI, body mass index; N/A, not applicable.
[1] Direction of association for reported outcomes: ↑increase; ↓inverse; N/A: not enough evidence to draw conclusions and recommendations.

3.261 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.

3.262 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 increased BMI in later childhood. However, the role of TDEI in this relationship is unclear. The evidence was graded **moderate**. There was **insufficient** evidence 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.

3.263 There was **insufficient** evidence 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

3.264 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

3.265 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).

3.266 One SR without MA (Hörnell et al, 2013) was identified that examined the relationship between total protein intake in children and timing of AR.

3.267 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 (statistics NR). One PCS adjusted for maternal BMI, gestational age and breastfeeding duration, as well as TDEI. The other study adjusted for sex only.

3.268 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 (statistics NR). The study adjusted for TDEI, maternal BMI, child gestational age and breastfeeding duration.

Protein intake and peak linear growth velocity (PLGV)

3.269 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

3.270 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 (statistics 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

3.271 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

3.272 The evidence identified from SRs on protein intake and growth outcomes is summarised in Table 3.39.

Table 3.39. Summary of the evidence on protein intake and growth

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Total protein intake	Age at AR	N/A	Insufficient
	BMI at AR	N/A	Insufficient
Total protein intake	PLGV	N/A	No evidence identified
Animal protein intake	PLGV	N/A	Insufficient
Vegetable protein intake	PLGV	N/A	No evidence identified

Abbreviations: AR, adiposity rebound; BMI, body mass index; N/A, not applicable; PLGV, peak linear growth velocity.

[1] N/A: not enough evidence to draw conclusions and recommendations.

3.273 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.

3.274 There was **insufficient** evidence 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.

3.275 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

3.277 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 later childhood, is unclear.

3.278 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.

3.279 For this draft 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

3.280 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 (statistics NR). However, it is unclear whether the analyses adjusted for confounding factors.

Animal protein intake and age of menarche or voice break

3.281 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.

3.282 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 statistics were not reported for a second PCS.

3.283 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.

3.284 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.

3.285 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 an increased odds of menarche by age 12 years and 8 months (statistics NR).

Vegetable protein intake and age of menarche or voice break

3.286 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. Statistics 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 3.283.

Protein intake and age of onset of pubertal growth spurt

3.287 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

3.288 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

3.289 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

3.290 Hörnell et al (2013) included 1 PCS (described in paragraph 3.289) 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

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

Animal protein intake and age at PLGV

3.292 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

3.293 Hörnell et al (2013) included 1 PCS (described in paragraph 3.292) 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

3.294 The evidence identified from SRs on protein intake and timing of puberty is summarised in Table 3.40.

Table 3.40. Summary of the evidence on protein intake and timing of puberty

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Total protein intake	Age of menarche	N/A	Insufficient
Animal protein intake	Age of menarche or voice break	↓	Limited
Vegetable protein intake	Age of menarche or voice break	N/A	Insufficient
Total protein intake	Age of onset of pubertal growth	N/A	No evidence identified
Animal protein intake	Age of onset of pubertal growth	N/A	Insufficient
Vegetable protein intake	Age of onset of pubertal growth	N/A	Insufficient
Total protein intake	Age at PLGV	N/A	No evidence identified
Animal protein intake	Age at PLGV	N/A	Insufficient
Vegetable protein intake	Age at PLGV	N/A	Insufficient

Abbreviations: N/A, not applicable; PLGV, peak linear growth velocity.

[1] Direction of association for reported outcomes: ↑increase; ↓inverse; N/A: not enough evidence to draw conclusions and recommendations.

3.295 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.

3.296 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.

3.297 There was **insufficient** evidence 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

3.298 One SR without MA (Voortman et al, 2015b) was identified that examined the relationship between total protein intake in childhood and blood lipids.

3.299 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).

3.300 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

3.301 The evidence identified from SRs on protein intake and blood lipids is summarised in Table 3.41.

Table 3.41. Summary of the evidence on protein intake and blood lipids

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Total protein intake	Blood lipids	N/A	Insufficient
Animal protein intake	Blood lipids	N/A	No evidence identified
Vegetable protein intake	Blood lipids	N/A	No evidence identified

Abbreviations: N/A, not applicable.

[1] N/A: not enough evidence to draw conclusions and recommendations.

3.302 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.

3.303 There was **insufficient** evidence 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.

3.304 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

3.305 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).

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

3.307 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≤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).

3.308 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

3.309 The evidence identified from SRs on protein intake and bone health is summarised in Table 3.42.

Table 3.42. Summary of the evidence on protein intake and bone health

Exposure	Outcome	Direction association[1]	Certainty of evidence
Protein intake	Bone health	N/A	Insufficient
Animal protein intake	Bone health	N/A	No evidence identified
Vegetable protein intake	Bone health	N/A	No evidence identified

Abbreviations: N/A, not applicable.

[1] N/A: not enough evidence to draw conclusions and recommendations.

3.310 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.

3.311 There was **insufficient** evidence 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.

3.312 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

3.313 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).

3.314 For this draft 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

3.315 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 (statistics NR). Analyses were stratified by sex but were not adjusted for other potential confounding factors, such as socioeconomic status.

Summary: protein intake and neurodevelopment

3.316 The evidence identified from SRs on protein intake and neurodevelopment is summarised in Table 3.43.

Table 3.43. Summary of the evidence on protein intake and neurodevelopment

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Total protein intake	Neurodevelopment	N/A	Insufficient

Abbreviations: N/A, not applicable.

[1] N/A: not enough evidence to draw conclusions and recommendations.

3.317 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.

3.318 There was **insufficient** evidence 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 Regarding 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 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 Annex 13, 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 milk (see Annex 13, Glossary). Vitamin A is also included in Healthy Start vitamins for children under 4 years (see Annex 1, Table A1.1 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%) that may be at risk of inadequate intakes of these micronutrients (for DNSIYC and NDNS data on all nutrients that were surveyed, see Annex 10, Tables A10.26). 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 indicate that children from lower socioeconomic backgrounds and certain ethnic minority groups may be more at risk of micronutrient deficiency.

4.7 This chapter provides an assessment of intake levels, dietary contributors to intake and status measures for iron, zinc, vitamin A and vitamin D in children aged 1 to 5 years followed by the evidence identified from systematic reviews (SRs) on the health impact of each micronutrient for this age group.

4.8 In addition to the above micronutrients, the committee noted that a brief 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 (Chapter 11, Table 11.1). Healthy Start vitamin drops also provide these vitamins.

4.9 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 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.

4.10 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 through the diet alone (for example, vitamin D and folate), risk management strategies should be identified.

Limitations of the evidence on micronutrients

4.11 The limitations described in paragraphs 4.12 to 4.19 relate to the NDNS while those described in paragraph 4.20 relate to the evidence identified from systematic reviews (SRs) with or without meta-analyses (MAs) on micronutrients.

4.12 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.

4.13 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 10, Tables A10.8 to A10.11 for details).

4.14 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).

4.15 For children aged 48 to 60 months, the proportion of children who gave a blood sample based on their age, sex and ethnic minority group roughly matched the age, sex and ethnic minority breakdown of the whole sample.

4.16 Children from all age groups 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).

4.17 Misreporting of food consumption and therefore 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 10, Table A10.12). The analysis showed 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 Annex 13, Glossary on 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 vitamins and minerals is not known.

4.18 Body weight z-scores (see Annex 13, Glossary) of children with intakes of iron, zinc or vitamin A below, at or above the LRNI for these micronutrients were compared 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 10, Table A10.12). 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.

4.19 The LRNI is set at the lowest 2.5th 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.

4.20 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, HIC). 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 or middle income countries (LIC or MIC). While findings from these trials can be useful in understanding health inequalities in HIC, 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

4.21 Due to the limitations stated in paragraph 4.20, the committee decided that only evidence that was most relevant to the UK context should be graded (see Chapter 2 on evidence grading) 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 Chapter 11, Table 11.1) and fortification trials for iron, vitamin A, vitamin D (given mandatory or voluntary fortification of foods with these micronutrients in the UK)
- Outcomes: status measures, growth, cognitive development, morbidities (including diarrhoea, fever, vomiting, respiratory infection)

4.22 Evidence was graded if findings from SRs were stratified by intervention type and baseline nutritional status (paragraph 4.21). 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 deficiency may improve health

outcomes, supplementing children with adequate status may lead to adverse health outcomes (paragraph 4.127).

4.23 Evidence that was not graded has been summarised in this chapter as it can offer insights into the physiological basis underpinning deficiency and deficiency-related health outcomes.

4.24 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 7 (Table A7.3). Additional data extracted on the primary studies can be found in Annex 8 (Table A8.12 to A8.23). The criteria used to grade the evidence are provided in chapter 2 (Table 2.4, paragraphs 2.43 to 2.46). Summary tables of the evidence grading process for this section are provided in Annex 9 (Tables A9.8, A9.10 and A9.29).

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

4.25 This section considers NDNS data on the dietary intake of children aged 18 to 60 months in the UK who meet the UK government 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.

4.26 As vitamin D requirements cannot be met through the diet alone, there is no entry for vitamin D in this section.

Iron

4.27 Dietary contributors to mean daily iron intake for 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 shown in Table 4.1 and Table 4.2, respectively.

4.28 In both age groups, almost a quarter of mean iron intake came from breakfast cereals (see paragraphs 4.77 and 4.78 for details on fortification in the UK). In the younger age group (age 18 to 47 months), infant formula contributed nearly 11% to mean iron intake. In both age groups, sources of haem iron (see paragraph 4.54), 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 for children aged 18 to 47 months with intakes at or above the RNI[1] for iron, zinc and vitamin A (NDNS years 2008/09 to 2018/19)

Food group	% contribution[2,3]	mg per day
Breakfast cereals	22.2	2.0
Infant formula[4]	10.7	1.1
Bread	10.3	0.9
Meat, meat products and dishes	9.3	0.8
Vegetables, products and dishes	7.1	0.7
Dietary supplements	6.5	0.7
Biscuits, buns, cakes, pastries, puddings	6.5	0.6
Fruit	5.1	0.5
Pizza, pasta, rice, products and dishes	4.5	0.4
Potatoes, products and dishes	2.7	0.2
Eggs, egg products and dishes	2.4	0.2
Commercial toddlers foods and drinks	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 2008/09 to 2018/19.
[1] 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).
[2] Food groups that contributed less than 1% to iron, zinc and vitamin A intakes are not presented.
[3] Average % contribution for each food group has been calculated from the % contribution for each individual. Non-consumers are included in the average.
[4] Infant formula consumed by children aged 18 months upwards are mainly follow on formula and growing up milks (see Annex 13, Glossary).

Table 4.2. Contributors to mean iron intake for children aged 48 to 60 months with intakes at or above the RNI[1] for iron, zinc and vitamin A (NDNS years 2008/09 to 2018/19)

Food group	% contribution[2,3]	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, 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
Commercial toddler foods and drinks	2.1	0.2
Potatoes, 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
Yogurt, fromage frais, dairy desserts[4]	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 2008/09 to 2018/19.

[1] 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).

[2] Food groups that contributed less than 1% to iron, zinc and vitamin A intakes are not presented.

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

[4] Includes dairy alternatives.

Zinc

4.29 Dietary contributors to mean zinc intake for 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% to zinc intake while in the older age group (age 48 to 60 months), zinc-containing dietary supplements contributed nearly 12%.

Table 4.3. Contributors to mean zinc intake for children aged 18 to 47 months with intakes at or above the RNI[1] for zinc, iron and vitamin A (NDNS years 2008/09 to 2018/19)

Food group	% contribution[2,3]	mg per day
Meat, meat products and dishes	17.6	1.21
Milk and cream[4]	15.5	1.04
Infant formula[5]	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[3]	5.0	0.33
Vegetables, products and dishes	4.4	0.30
Biscuits, buns, cakes, pastries, puddings	4.1	0.27
Yogurt, fromage frais and dairy desserts[4]	3.8	0.26
Fruit	2.6	0.18
Eggs, egg products and dishes	2.3	0.16
Potatoes, products and dishes	1.9	0.12
Fish, fish products and dishes	1.7	0.11
Commercial toddlers foods and drinks	1.6	0.12
Number of participants	254	254

Abbreviations: RNI, reference nutrient intake

Data from NDNS years 2008/09 to 2018/19.

[1] 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).

[2] Food groups that contributed less than 1% to zinc, iron and vitamin A intake are not presented.

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

[4] Includes dairy alternatives.

[5] Infant formula consumed by children aged 18 months upwards are mainly follow on formula and growing up milks (see Annex 13, Glossary).

Table 4.4. Contributors to zinc intake for children aged 48 to 60 months with intakes at or above the RNI[1] for zinc, iron, and vitamin A (NDNS years 2008/09 to 2018/19)

Food Group	% contribution[2,3]	mg per day
Meat, meat products and dishes	19.3	1.59
Milk and cream[4]	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[4]	6.1	0.49
Breakfast cereals	5.1	0.42
Vegetables, products and dishes	4.5	0,38
Biscuits, buns, cakes, pastries, puddings	4.3	0.36
Yogurt, fromage frais, dairy desserts[4]	3.7	0.30
Commercial toddlers foods and drinks	2.2	0.16
Fruit	2.1	0.18
Eggs, egg products and dishes	1.9	0.15
Potatoes, 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 2008/09 to 2018/19.

[1] 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).

[2] Food groups that contributed less than 1% to zinc, iron and vitamin A intake are not presented.

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

[4] Includes dairy alternatives.

Vitamin A

4.30 Dietary contributors to mean vitamin A intake for 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.

4.31 Carrots and milk (plus infant formula in younger children) and dietary supplements were the main contributors to vitamin A intake for both age groups.

Table 4.5. Contributors to vitamin A intake for children aged 18 to 47 months with intakes at or above the RNI[1] for vitamin A, iron, and zinc (NDNS years 2008/09 to 2018/19)

Food Group	% contribution[2,3]	µg per day
Carrots raw and cooked	15.5	155
Milk and cream[4]	11.5	79
Dietary supplements	11.3	124
Meat, meat products and dishes	8.9	92
Infant formula[5]	8.5	66
Fat spreads	7.6	51
Vegetables, products and dishes (excl carrots)	6.8	50
Cheese[4]	4.8	33
Soup	3.5	33
Yogurt, fromage frais and dairy desserts[4]	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
Commercial toddlers foods and drinks	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 2008/09 to 2018/19.

[1] 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).

[2] Food groups that contributed less than 1% to vitamin A, zinc and iron intake are not presented.

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

[4] Includes dairy alternatives.
[5] Infant formula consumed by children aged 18 months upwards are mainly follow on formula and growing up milks (see Annex 13, Glossary).

Table 4.6. Contributors to vitamin A intake for children aged 48 to 60 months with intakes at or above the RNI[1] for vitamin A, iron, and zinc (NDNS years 2008/09 to 2018/19)

Food group	% contribution[2,3]	µg per day
Carrots raw and cooked	19.9	226
Dietary supplements	17.1	202
Milk and cream[4]	12.3	102
Vegetables, products and dishes (excluding carrots)	8.5	72
Fat spreads	7.0	60
Cheese[4]	5.8	49
Meat, meat products and dishes	5.4	65
Biscuits, buns, cakes, pastries, puddings	4.5	35
Pizza, pasta, rice, products and dishes	3.5	30
Yogurt, fromage frais, dairy desserts[4]	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[4]	1.2	9
Commercial toddlers foods and drinks	1.1	12
Number of participants	71	71

Abbreviations: RNI, reference nutrient intake
Data from NDNS years 2008/09 to 2018/19.
[1] 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).
[2] Food groups that contributed less than 1% to vitamin A, zinc and iron intake are not presented.
[3] Average % contribution for each food group has been calculated from the % contribution for each individual. Non consumers are included in the average.
[4] Includes dairy alternatives.

4.32 Tables 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).

4.33 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.

4.34 Children in the younger age group (18 to 47 months) were meeting the RNIs for all 3 micronutrients with infant formula and dietary supplements. 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 per day, as formula (see Annex 13, Glossary) is fortified with vitamins A, C and D and other nutrients. Children who consume both infant formula and dietary supplements who meet the RNI for all 3 micronutrients may be at risk of excess intakes of these micronutrients.

4.35 Dietary supplements also contributed substantially to intakes of the 3 micronutrients in the older age group (9 to 17%).

Iron

Physiological requirements

4.37 If born with adequate iron stores, healthy term infants of appropriate weight have sufficient iron for growth and development for the first 6 months of life (SACN, 2018).

4.38 After 6 months of age, dietary requirements for iron are increased as iron stores diminish (SACN, 2010). A diverse complementary diet, alongside breast milk as the main drink throughout the first year of life, is needed to meet the increasing iron requirements of older infants (SACN, 2018).

4.39 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

4.40 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).

4.41 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).

4.42 Serum ferritin and haemoglobin concentrations are commonly cited markers of iron status, but the thresholds to indicate deficiency have been much debated (see SACN, 2018 for details). 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).

4.43 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

4.44 Although no single marker of iron metabolism is considered ideal for the assessment of iron deficiency (or excess), in this draft 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.

4.45 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, 2001b). 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).

4.46 For this draft 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

4.47 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.

4.48 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.

4.49 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 2008/09 to 2018/19)

Age	Haemoglobin (g/dl)[1,2]	Plasma ferritin(µg/l) [1,3]	% ID (plasma ferritin below 12µg/l)	% anaemia (Hb below 110g/l)	% IDA (% below thresholds for ferritin and Hb)
12 to 18 months[4]	11.7 (1.0)	28.3 (18.8)	11	15	2
18 to 47 months [5]	12.0 (8.2)	24.5 (18.7)	23.9	9.0	3.3
48 to 60 months [5]	12.3 (8.0)	29.1 (22.6)	20.0	7.2	[0.0][6]

Abbreviations: Hb, haemoglobin; ID, iron deficiency; IDA, iron deficiency anaemia; SD, standard deviation
[1] Mean (SD).
[2] Number of participants: 325 (12 to 18 months), 140 (18 to 47 months), 58 (48 to 60 months).
[3] Number of participants: 298 (12 to 18 months), 117 (18 to 47 months), 53 (48 to 60 months).
[4] Data from DNSIYC 2011 (DH, 2013).
[5] Data from NDNS years 2008/09 to 2018/19.
[6] Data for a variable with a cell size between 30 to 49 are presented in square brackets.

Non-dietary determinants of iron status

4.50 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).

4.51 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).

4.52 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).

4.53 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

4.54 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).

4.55 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).

4.56 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).

4.57 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[1]

Age	LRNI mg per day (µmol per day)	EAR mg per day (µmol per day)	RNI mg per day (µmol 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

[1] Source: DH (1991) and SACN (2010).

Iron intake in the UK

4.58 Iron intake data for children in the UK aged 12 to 60 months from DNSIYC and NDNS (years 2016/17 to 2018/19) are shown in Table 4.9.

Table 4.9. Iron intake in children aged 12 to 60 months in the UK (DNSIYC and NDNS years 2016/17 to 2018/19)

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[1]	93	92	13	Data not available	1275
18 to 47 months[2]	88	84	11	11	306
48 to 60 months[2]	187	186	1	1	102

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

[1] Data from DNSIYC 2011 (DH, 2013).

[2] Data from NDNS years 2016/17 to 2018/19

4.59 Table 4.9 indicates 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.

4.60 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 4.17), and uncertainties in the iron DRVs (SACN, 2011b).

4.61 Secondary analysis of NDNS data (years 2008/09 to 2018/19) 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 10, Tables A10.14 to A10.17). Characteristics that were considered were age, sex, ethnicity and household socioeconomic status.

4.62 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 category (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 has 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 needs to be taken when interpreting the findings because the total number of children with intakes below the LRNI was small (n=118).

4.63 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).

4.64 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.

4.65 Time trend analysis of NDNS data (years 2008/09 to 2018/19) for children aged 18 to 47 months indicated a significant average annual reduction in daily iron intake (from food sources only) of -0.07mg (95%CI -0.11 to -0.03mg), equivalent to a reduction of 0.8mg 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.

4.66 No time trend data was available for the other 2 age groups.

Iron intake and deprivation

4.67 Iron intake by index of multiple deprivation (IMD) for children aged 18 to 60 months are shown 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 2008/09 to 2018/19)

Iron mg/day	IMD quintile 1 (least deprived)	IMD quintile 2	IMD quintile 3	IMD quintile 4	IMD quintile 5 (most deprived)
Mean (90% 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
Data from NDNS years 2008/09 to 2018/19.

4.68 The IMD analysis does 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 2012/13 to 2016/17) in children aged 18 to 36 months that used a narrower measure of household socioeconomic status (equivalised household income, see Annex 13, Glossary) suggested 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

4.69 Dietary iron exists in 2 main forms: haem iron and non-haem iron (paragraph 4.54).

4.70 The main dietary contributors (including from dietary supplements) to mean iron intake for children in the UK with intakes below the LRNI for iron were compared with those for children with intakes above the LRNI. Detailed results of this analysis of NDNS data (years 2008/09 to 2018/19) are presented in Annex 10, Tables A10.18 and A10.19 for 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.

4.71 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 10, Table A10.15). 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) for children with an iron intake below the LRNI.

4.72 While children with iron intakes below the LRNI obtained a higher proportion of their iron intake from bread, meat and meat products, their absolute intake of iron from these foods was lower than that in children with a mean intake at or above the LRNI. This may be accounted for by their lower TDEI, smaller body size or the greater tendency to underreport TDEI (see paragraph 4.17), or a combination of these factors.

4.73 For children aged 48 to 60 months, there were insufficient numbers of children to present results in those with intakes below the LRNI.

Evidence identified on iron

Interventions to improve iron status

4.74 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.

4.75 Only 1 randomised controlled trial (RCT), included in 2 SRs identified for this draft 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.

4.76 Observational evidence from Ireland suggests that cows' milk intake ≥400ml 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

4.77 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).

4.78 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).

4.79 The composition of infant formula and follow-on formula (see Annex 13, 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).

4.80 For this draft 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.

4.81 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 (≤12 months) and conducted in upper-middle income or lower-middle income countries (UMIC/LMIC) or LIC.

4.82 Following the methodological approach outlined in paragraphs 4.21 and 4.22, 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.

4.83 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 2.43 to 2.47). Summary tables of the evidence

grading process for this section are provided in Annex 9 (Table A9.8, A9.9 and A9.29).

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

4.84 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.

4.85 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 (statistics 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)

4.86 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.

4.87 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.

4.88 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

4.89 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.

4.90 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 (statistics 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 (statistics 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

4.91 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.

4.92 Matsuyama et al (2017)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. Statistics were NR for either study. According to the SR, both studies had either a low or unclear risk of bias from their funding sources.

4.93 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 (statistics 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

4.94 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

4.95 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.

4.96 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).

4.97 All 3 trials reported a reduction in the prevalence of anaemia after 2 to 12 months' intervention using PP analyses (none performed ITT analyses).

4.98 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.

4.99 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) led to a reduction in 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.

4.100 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)

4.101 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.

4.102 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

4.103 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

Population subgroup	Outcome (population subgroup)	Direction of effect[1]	Certainty of evidence
Iron fortification (with or without other micronutrients)	Hb concentration (in children without anaemia)	N/A	No evidence identified
	Hb concentration (in children with anaemia or high anaemia prevalence)	N/A	Insufficient
	Serum ferritin (in children without anaemia)	N/A	Insufficient
	Serum ferritin (in children with anaemia or high anaemia prevalence)	N/A	Insufficient
	Prevalence of ID (in children with anaemia or high anaemia prevalence)	N/A	Insufficient
	Anaemia prevalence (in children without anaemia)	N/A	No evidence identified
	Anaemia prevalence (in children with anaemia or high anaemia prevalence)	↓	Limited

Abbreviations: ID, iron deficiency; N/A, not applicable.
Definitions: ID (serum ferritin <12µg/l); anaemia (haemoglobin <110g/l).
[1] Direction of effect for reported outcomes: ↑increase; ↓decrease; N/A: not enough evidence to draw conclusions and recommendations.

4.104 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.

4.105 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 9, Table A9.9 for details for the grading process).

4.106 There was **insufficient** evidence 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 9, Table A9.8 for details for the grading process).

4.107 There was **insufficient** evidence for all other outcomes (Table 4.11) as fewer than 3 primary studies included in the SRs examined these relationships.

Iron supplementation

4.108 Much of the research examining interventions to prevent or reverse IDA in children aged under 5 years comes from supplementation trials conducted in LIC or LMIC 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 indicate 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, page 134).

4.109 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, see SACN, 2010 for details), and the possible adverse side effects on growth in young children who do not have anaemia (see section on Iron status and growth).

4.110 Nonetheless, supplementation trials conducted in lower income countries have been useful in informing the understanding of iron metabolism, deficiency and associated health outcomes.

4.111 SRs with MAs (De-Regil et al, 2011; Thompson et al, 2013) identified for this draft 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

4.112 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.

4.113 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).

4.114 One SR with MA (Domellöf et al, 2013) identified for this draft report examined the effects of interactions between iron and other micronutrients or food components on iron status.

4.115 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/or 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

4.116 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 neurological development

4.117 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 (2001) cut-off for IDA (SACN, 2010).

ID without anaemia and neurological development

4.118 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).

4.119 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).

4.120 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

4.121 For this draft report, 1 SR with MA of supplementation trials was identified that examined the effect of iron on cognitive outcomes in children aged under 5 years with ID from mostly MIC (Pasricha et al, 2013). Following the methodological approach outlined in paragraphs 4.21 123and 4.22, the evidence is described below but was not graded.

4.122 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

4.123 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

4.124 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).

4.125 For this draft 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.

4.126 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

4.127 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

4.128 For this draft 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 4.21 and 4.22, the evidence was not graded.

4.129 Thompson et al (2013) (AMSTAR 2 confidence rating: moderate) reported no difference in effect on either linear growth or weight gain between children supplemented with iron daily for up to 12 months and the control group (see Annex 8, Table A8.14 for details).

Iron status and immune function

4.130 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.

4.131 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

4.132 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 4.21 and 4.22, the evidence is described below but was not graded.

4.133 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.

4.134 Evidence on the effect of iron supplementation on fever was equivocal. Pasricha et al (2013) reported that iron supplementation may increase 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 (statistics NR).

4.135 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

4.136 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

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

Table 4.12. Dietary reference values for zinc for children aged 1 to 6 years[1]

Age	LRNI mg per day (µmol per day)	EAR mg per day (µmol per day)	RNI mg per day (µmol 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

[1] Source: (DH, 1991).

Zinc intake in the UK

4.138 Intake data for children in the UK aged 12 to 60 months from DNSIYC and NDNS years 2016/17 to 2018/19 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 2016/17 to 2018/19)

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[1]	200	200	4	4	1275
18 to 47 months[2]	101	96	8	8	306
48 to 60 months[2]	84	83	20	21	102

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

[1] Data from DNSIYC 2011 (DH, 2013).

[2] Data from NDNS years 2016/17 to 2018/19.

4.139 Table 4.13 shows that 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 4.17).

4.140 Secondary analysis of the data from NDNS (years 2008/09 to 2018/19) 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 10, Tables A10.14 to A10.17). Characteristics that were considered were age, sex, ethnicity and household socioeconomic status.

4.141 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 category (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 has 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 needs to 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).

4.142 Time trend analysis of NDNS data (years 2008/09 to 2016/17) for children aged 18 to 47 months indicated a significant average annual reduction in daily zinc intake (from food sources only) of -0.05 mg (95% CI -0.10 to -0.01mg) 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 (0.37 percentage point change per year; 95% CI -0.33 to 1.06 percentage points) (Bates et al, 2019). No time trend data was available for the other 2 age groups.

Zinc intake and deprivation

4.143 Zinc intake by IMD (see Annex 13, Glossary) for children aged 18 to 60 months is shown 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 2008/09 to 2018/19)[1]

Zinc intake mg/day	IMD quintile 1 (least deprived)	IMD quintile 2	IMD quintile 3	IMD quintile 4	IMD quintile 5 (most deprived)
Mean (90%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
Data from NDNS years 2008/09 to 2018/19.

4.144 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 2012/13 to 2016/17) in children aged 18 to 36 months that used a narrower measure of household socioeconomic status (equivalised household income, see Annex 13, 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 0.09mg per day (95% CI 0.01 to 0.18mg 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

4.145 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).

4.146 The main dietary contributors (including from supplements) to zinc intake for children in the UK with intakes below the LRNI for zinc were compared with those for children with intakes above the LRNI. Detailed results of this analysis of NDNS data (years 2008/09 to 2018/19) are presented in Annex 10, Tables A10.20 to A10.23 for children aged 18 to 47 months, and ages 48 to 60 months. The contribution of these food groups to TDEI is also shown.

4.147 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 10, Table A10.20). 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 the children with intakes at or above the LRNI (22.2%).

4.148 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

4.149 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).

4.150 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).

4.151 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).

4.152 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).

4.153 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 shows that mean zinc intake as a percentage of RNI decreases with increasing age (Table 4.13), likely reflecting the increase in the RNI for children aged 4 to 6 years Table 4.12.

Evidence identified on zinc

Interventions to improve zinc status

Zinc supplementation

4.154 Trials conducted in LIC and LMIC 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

4.155 Just as high iron intake may interfere with the metabolism of other similar metals (see Iron and interactions with other micronutrients and food components, p.148), 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.

4.156 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

4.158 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

4.159 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).

4.160 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.

4.161 For this draft 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 4.21 and 4.22, the evidence is described below but the certainty of the evidence was not graded.

Linear growth

4.162 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

4.163 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).

Vitamin A

Physiological requirements

4.164 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).

4.165 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

4.166 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, 2016b).

4.167 The current DRVs for vitamin A (Table 4.15) 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 (µg) of retinol equivalents (RE): 1µg RE = 1µg retinol = 6µg beta-carotene = 12 µg other carotenoids with provitamin A activity (WHO and FAO, 1967).

Table 4.15. DRVs for vitamin A for children aged 1 to 6 years[1]

Age	LRNI RE µg per day	EAR RE µg per day	RNI RE µg per day	TUL for retinol[2] 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

[1] Source: (DH, 1991).

[2] 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 4.170).

4.168 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.

4.169 In 2002, the European Scientific Committee on Food (SCF) established TUL of preformed vitamin A (retinol) for children as well as adults (Table 4.15). 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.

4.170 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 9).

Vitamin A intake in the UK

4.171 Intake data for children in the UK aged 12 to 60 months from the DNSIYC and NDNS years 2016/17 to 2018/19 are presented in Table 4.16.

Table 4.16. Vitamin A intake (RE) in children aged 12 to 60 months in the UK (DNSIYC and NDNS years 2016/17 to 2018/19)

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[3]	Intake from diet only % above TUL for retinol[3]	Number of participants
12 to 18 months[1]	175	169	2	2	1.9	0.9	1275
18 to 47 months[2]	136	115	8	9	4.2	0.4	306
48 to 60 months[2]	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

[1] Data from DNSIYC 2011 (DH, 2013).

[2] Data from NDNS years 2016/17 to 2018/19.

[3] 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 4.170).

4.172 Mean intake of vitamin A 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. 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 4.17).

4.173 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 4.19). However, given COT's concerns regarding the TUL set by the European SCF (see paragraph 4.170), the data should be interpreted with caution.

4.174 Secondary analysis of the data from NDNS (years 2008/09 to 2018/19) 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 10, Tables A10.14 to A10.17). Characteristics that were considered were age, sex, ethnicity and household socioeconomic status.

4.175 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 needs to be taken when interpreting the findings because the total number of children with intakes below the LRNI was small (n=95).

4.176 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.

4.177 Time trend analysis of NDNS data (years 2008/09 to 2018/19) for children aged 18 to 47 months indicated an average annual reduction in vitamin A intake (from food sources only) of -2.35% (95% CI -3.54 to -1.16%), 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 (0.46 percentage point average change per year; 95% CI -0.19 to 1.11 percentage points). No time trend data was available for the other 2 age groups.

4.178 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).

4.179 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

4.180 Vitamin A intake by IMD (see Annex 13, Glossary) for children aged 18 to 60 months is shown in Table 4.17. Mean vitamin A intake was highest in quintile 1 (least deprived) (562µg RE per day) and lowest in quintile 5 (most deprived) (421µg RE per day). The confidence intervals indicate that mean intake in quintile 1 was significantly higher than in quintile 4 and 5, and that mean intake in quintile 2 was significantly higher than in quintile 5.

Table 4.17. Vitamin A intake (from diet only) by IMD quintile in children aged 18 to 60 months in England (NDNS years 2008/09 to 2018/19)

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 (90%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 2008/09 to 2018/19.

4.181 Evidence that vitamin A intake follow a social gradient in the UK is supported by an analysis on NDNS data (years 2012/13 to 2016/17) in children aged 18 to 36 months that used a narrower measure of household socioeconomic status (equivalised household income, see Annex 13, 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

4.182 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).

4.183 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).

4.184 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).

4.185 To take account of the contribution from provitamin A carotenoids, the total vitamin A content of the diet is usually expressed as micrograms (µg) of RE (see Annex 13, Glossary).

4.186 Table 4.18 presents 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.

Table 4.18. Sources of vitamin A intake (retinol and total carotene) in children aged 12 to 60 months in the UK (DNSIYC and NDNS 2016/17 to 2018/19)

Age	Retinol (µg/day)	Retinol (µg/day)	Total carotene (µg/day)	Total carotene (µg/day)	Number of participants
	Intake from diet and supplements Mean (SD)	Intake from diet only Mean (SD)	Intake from diet and supplements Mean (SD)	Intake from diet only Mean (SD)	
12 to 18 months[1]	341 (183)	319 (147)	2144 (1871)	2141 (1870)	1275
18 to 47 months[2]	319 (221)	236 (121)	1347 (1214)	1345 (1215)	306
48 to 60 months[2]	306 (223)	225 (134)	1827 (2047)	1826 (2047)	102

[1] Data from DNSIYC 2011.
[2] Data from NDNS years 2016/17 to 2018/19.

4.187 Intake of retinol from food decreased with age, potentially reflecting the drop in milk or formula milk intake in the older age groups (Table 4.18).

4.188 The main dietary contributors (including from supplements) to vitamin A intake for children with intakes below the LRNI for vitamin A were compared with those for children with intakes above the LRNI. Detailed results of this analysis of NDNS data (years 2008/09 to 2018/19) are presented in Annex 10, Tables A10.24 to A10.25 for 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.

4.189 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 that met or exceeded the LRNI compared with those with intakes below the LRNI was most pronounced for carrots and dietary supplements (Annex 10, Table A10.24). 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.

4.190 While children with intakes below the LRNI obtained a higher proportion of their vitamin A intake from milk and cream, cheese and yogurt, 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 10, Table A10.24). This may be accounted for by their lower TDEI, smaller body size, the greater tendency to underreport energy intakes (see paragraph 4.17) or a combination of these factors.

4.191 Only 46 children aged 18 to 47 months had high vitamin A intakes (Table 4.19). 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 4.19. Contributors to retinol intake for children aged 18 to 47 months who exceeded the TUL[2] for vitamin A (including from supplements)[1]

Food group	% contribution[3,4]	µg per day
Retinol containing dietary supplements	[58.5]	[648]
Meat, meat products and dishes	[16.8]	[259]
Milk and cream[5]	[7.2]	[80]
Infant formula	[3.1]	[42]
Butter and fat spreads	[2.8]	[31]
Cheese[5]	[2.5]	[25]
Biscuits, buns, cakes, pastries, puddings	[2.2]	[23]
Eggs, products and dishes	[1.7]	[16]
Yogurt, fromage frais, dairy desserts[5]	[1.2]	[12]
Pizza, pasta, rice products and dishes	[0.9]	[10]
Commercial toddlers foods and drinks	[0.9]	[10]
Ice cream[5]	[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
[1] Data from NDNS years 2008/09 to 2018/19.
[2] 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 4.170).
[3] Food groups that contributed less than 0.5% to retinol intake 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 dairy alternatives.

4.192 Dietary supplements were the principal source of high intake of vitamin A (Table 4.19) 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 4.170). 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 Chapter 9). However, if effects did occur it would be in a small proportion of consumers.

4.193 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 A1.1), an analysis undertaken by the COT (COT, 2017) of data from DNSIYC and NDNS (years 2008/09 to 2011/12) indicates that among children eligible for the scheme, uptake of these vitamins was unlikely to result in intakes above the TUL.

Assessment of vitamin A status

4.194 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).

4.195 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).

4.196 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).

4.197 Vitamin A status (plasma retinol concentrations) for children aged 12 to 60 months from the DNSIYC and NDNS (years 2008/09 to 2018/19) are shown in Table 4.20. 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 4.20. Vitamin A status (plasma retinol) in children aged 12 to 60 months in the UK (DNSIYC and NDNS years 2008/09 to 2018/19)

Age	Plasma retinol (µmol/l) Mean (SD)	% below 0.35µmol/l[1]	% at 0.35 to 0.70µmol/l[1]	Number of Participants
12 to 18 months[2]	Not available	Not available	Not available	N/A
18 to 47 months[3]	1.03 (0.26)	0	7	103
48 to 60 months[3]	[1.12 (0.30)][4]	[0][e]	[10][e]	41

[1] Thresholds confined mainly to non-elderly adults (Bates et al, 1997).
[2] Plasma retinol was not measured in this age group.
[3] Data from NDNS years 2008/09 to 2018/19.
[4] [ ] data presented in square brackets denotes that the estimates are based on a cell size between 30 and 49.

4.198 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.

Evidence identified on vitamin A

Interventions to improve vitamin A status

Vitamin A supplementation

4.199 For this draft 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 LIC, LMIC and UMIC. 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 4.21 and 4.22).

4.200 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=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

4.201 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 4.21 and 4.22).

4.202 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).

4.203 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 3.7µg/dl; 95% CI 1.3 to 6.1µg/dl; p-value NR; I^2=37%; 4 RCTs, participants NR).

Vitamin A and interactions with other nutrients

4.204 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).

4.205 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).

4.206 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)2D to exert genomic and non-genomic effects (Bouillon et al, 2019).

4.207 See Section on Vitamin A deficiency and anaemia (p.172) for a discussion on interactions between vitamin A and iron.

Vitamin A and health

4.208 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

4.209 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

4.210 One SR with MA (Imdad et al, 2017) was identified that examined the effect of vitamin A supplementation on opthalmological outcomes in children mostly aged up to 5 years. Most interventions lasted beyond 1 year and were formed in LIC, LMIC and UMIC. 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 4.21 and 4.22).

4.211 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=NR; I^2=0%; fixed-effects model; 2 RCTs, 22,972 participants) compared with the control group.

4.212 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=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=NR; I^2=49%; fixed effects model), incident xerophthalmia (RR 0.85; 95% CI 0.70 to 1.03; p=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 immunity

4.213 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.

4.214 Pre-existing VAD may worsen infection in young children (WHO, 2009). In LMIC, VAD in infants and young children has been associated with increased mortality from infection, and increased infectious morbidity (Imdad et al, 2017).

4.215 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

4.216 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 LMIC. 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 4.21 and 4.22).

4.217 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=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

4.218 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).

4.219 One SR with MA (Das et al, 2013) was identified that examined the effect of vitamin A fortification on iron status in children from LMIC. 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 4.21 and 4.22).

4.220 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^2=93%; random-effects model; 2 RCTs, 1538 participants).

Vitamin D

Physiological requirements

4.221 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

4.222 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).

4.223 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).

4.224 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

4.225 The UK government 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 intake in the UK

4.226 Mean vitamin D intake (% RNI) for vitamin D in children not breastfed and children breastfed (excluding breast milk) from DNSIYC and NDNS years 2016/17 to 2018/19 are presented in Table 4.21.

4.227 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 4.21. Vitamin D intake in children aged 12 to 60 months in the UK (DNSIYC and NDNS 2016/17 to 2018/19)

Age	Intake from diet and supplements	Intake from diet only	Intake from diet and supplements	Intake from diet only	Number of participants
	Mean intake as % of RNI Non-breastfed[1]		Mean intake as % of RNI Breastfed excluding breast milk[2]		
12 to 18 months[3]	55	50	37	26	1275
18 to 47 months[4]	40	24	N/A	N/A	306
48 to 60 months[4]	39	25	N/A	N/A	102

Abbreviations: LRNI, Lower Reference Nutrient Intake; RNI, Reference Nutrient Intake; N/A not applicable.
[1] 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).
[2] 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).
[3] Data from DNSIYC 2011 (DH, 2013).
[4] Data from NDNS years 2016/17 to 2018/19).

4.228 Time trend analysis of NDNS data (years 2008/09 to 2016/17) for children aged 18 to 47 months showed a non-significant average annual change in vitamin D intake of 1.21% (95% CI -1.05 to 3.52%) for the 9-year period (Bates et al, 2019). No time trend data was available for the other 2 age groups.

Vitamin D intake and deprivation

4.229 Vitamin D intake by IMD (See Annex 13, Glossary) for children aged 18 to 60 months is shown in Table 4.22..

Table 4.22. Vitamin D intake (from diet only) by IMD quintile in children aged 18 to 60 months in England (NDNS years 2008/09 to 2018/19)

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 (90%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 2008/09 to 2018/19.

4.230 There appears to be no relationship linking vitamin D intake and IMD, a broad measure of deprivation (indicated by overlapping confidence intervals). However, another analysis of NDNS data (years 2012/13 to 2016/17) in children aged 18 to 36 months that used a narrower measure of household socioeconomic status (equivalised household income, see Annex 13, Glossary) suggested 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 minority group in the UK

4.231 Vitamin D intake by ethnic minority group the DNSIYC and the NDNS (years 2016/17 to 2018/19) are presented in Table 4.23. Sample numbers were insufficient to analyse data from specific ethnic minority groups.

Table 4.23. Vitamin D intake (µg per day) by ethnic minority group for young children in the UK (DNSIYC and NDNS years 2016/17 to 2018/19)

Age	Other ethnic minority groups[3]		White[4]	
	Intake from diet and supplements Mean (SD)	Intake from diet only Mean (SD)	Intake from diet and supplements Mean (SD)	Intake from diet only Mean (SD)
12 to 18 months[1]	4.7 (4.5)	3.8 (4.0)	3.6 (3.6)	3.3 (3.2)
18 to 60 months[2]	3.1 (3.1)	2.9 (3.4)	2.9 (2.8)	2.3 (2.1)

[1] Data from DNSIYC 2011 (DH, 2013).
[2] Data from NDNS 2016/17 to 2018/19.
[3] 90 participants in the 12 to 18 months age category; 63 participants in the 18 to 60 months age category. Sample sizes were insufficient to analyse data from specific ethnic minority groups.
[4] 1085 participants in the 12 to 18 months age category. 343 participants in the 18 to 60 months age category.

Assessment of vitamin D status

4.232 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).

4.233 Vitamin D status in children aged 12 to 60 months from the DNSIYC and the NDNS (years 2008/09 to 2018/19) is shown in Table 4.24.

Table 4.24. Vitamin D status (serum 25(OH)D) in children aged 12 to 60 months in the UK (DNSIYC and NDNS 2008/09 to 2018/19)

Age	25(OH)D nmol/l Mean (SD)	% below 25nmol/l	Number of participants
12 to 18 months[1]	64.3 (24.3)	2	300
18 to 47 months[2]	58.3 (23.2)	9	116
48 to 60 months[2]	[47.7 (21.3)][3]	[28][3]	49

Abbreviations: 25(OH)D, 25-hydroxy vitamin D; SD, standard deviation.
[1] Data from DNSIYC 2011 (DH, 2013).
[2] Data from NDNS years 2008/09 to 2018/19.
[3] [ ] data presented in square brackets denotes that the estimates are based on a cell size between 30 and 49.

4.234 Time trend analysis of NDNS data (years 2008/09 to 2018/19) for children aged 18 to 47 months showed no significant change in serum 25(OH)D (0.08 nmol/l; 95%

CI -1.36 to 1.52 nmol/l) for the 11-year period, and no significant change in the percentage of children below the 25 (OH) vitamin D threshold of 25nmol/l (-0.32 percentage point average change per year; 95% CI -1.54 to 0.90 percentage points) (Bates et al, 2020). No time trend data was available for the other 2 age groups.

Vitamin D status and deprivation

4.235 An analysis of NDNS data (years 2012/13 to 2016/17) suggested that every £10,000 increase in equivalised household income (Annex 13, 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

4.236 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).

4.237 Table 4.25 compares the vitamin D status of children aged 12 to 18 months by ethnic minority group (Lennox et al, 2013). Although the sample size is too small to draw firm conclusions, the data do indicate that, compared with white children, children from other ethnic minority groups are at higher risk of vitamin D deficiency. Recent data in the UK also suggests that they are at higher risk of clinical manifestations of vitamin D deficiency (see Section on nutritional rickets, p.181). Data from the NDNS were insufficient to perform a similar analysis on children aged 18 to 60 months.

Table 4.25. Vitamin D status (25(OH)D) by ethnic minority group in children aged 12 to 18 months in the UK (DNSIYC)[1]

Age	25(OH)D nmol/l[2] Mean (SD)		% below 25µmol/l	
	Other ethnic minority groups[3]	White[4]	Other ethnic minority groups[3]	White[4]
12 to 18 months	[61.0 (25.7)][2]	66.1 (24.4)	[4][2]	1

Abbreviations: 25(OH)D, 25-hydroxy vitamin D; SD, standard deviation.

[1] Data from DNSIYC 2011 (DH, 2013). Note that blood samples were not collected over a full calendar year
[2] [ ] data presented in square brackets denotes that the estimates are based on a cell size between 30 and 49.
[3] 40 participants in the 12 to 18 months age category. Sample sizes were insufficient to analyse data from specific ethnic minority groups.
[4] 191 participants in the 12 to 18 months age category.

Evidence identified on vitamin D

Interventions to improve vitamin D status

4.238 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 draft report (May 2019).

4.239 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 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) cannot be said to be comprehensive for identifying studies on vitamin D fortification.

4.240 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 7 (Table A7.3). The criteria used to grade the evidence are provided in Chapter 2 (Table 2.4, paragraphs 2.43 to 2.47). Additional data extracted from the primary studies can be found in Annex 8 (Table A8.23). The criteria used to grade the evidence are provided in chapter 2 (Table 2.4, paragraphs 2.43 to 2.46). Summary tables of the evidence grading process for this section are provided in Annex 9 (Table A9.10). Following the methodological approach described in paragraphs 4.21 and 4.22, the certainty of the evidence was graded.

4.241 Hojsak et al (2018) (AMSTAR 2 confidence rating: 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 (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 <50nmol/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 70nmol/l. Two of the 3 studies were funded by manufacturers of formula milk.

Summary: vitamin D fortification and vitamin D status

4.242 The evidence identified from SRs on vitamin D fortification (of milk or formula) and vitamin D status is summarised in Table 4.26.

Table 4.26. Summary of the evidence on the relationship between vitamin D fortification and vitamin D status

Exposure	Outcome	Direction of effect[1]	Certainty of evidence
Vitamin D fortification (of milk or formula)	Vitamin D status	↑	Limited

[1] Direction of effect for reported outcomes: ↑increase; ↓inverse.

4.243 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.

4.244 Evidence from 3 RCTs included in the SR by Hojsak et al (2018) suggests that vitamin D fortification of milk or formula 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 9, Table A9.10 for details for the grading process).

Vitamin D and health

Nutritional rickets and osteomalacia

4.245 The re-emergence of nutritional rickets in children in many countries, including the UK, has become a public health concern. A recent 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 minority groups, and at diagnosis, 78% of cases were not reportedly receiving any vitamin supplements (Julies et al, 2020).

4.246 For this draft report, no new evidence from SRs was identified on the relationship between vitamin D status/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 draft report (May 2019).

4.247 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.

4.248 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 draft report.

Bone health indices (bone mineral content, bone mineral density, biochemical markers of bone turnover)

4.249 For this draft 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 draft report (May 2019).

4.250 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

4.252 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).

4.253 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

4.254 The UK government recommends that children aged from 6 months up to 5 years are 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, 2016b). Vitamin C is also included in the vitamin drops provided under the Healthy Start scheme in England, Wales and Northern Ireland (see Chapter 1, Table 1 for details on the scheme).

4.255 The current 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

4.256 Intake data for children in the UK aged 12 to 60 months from DNSIYC and NDNS (years 2016/17 to 2018/19) are presented in Table 4.27.

Table 4.27 Vitamin C intake in children aged 12 to 60 months in the UK (DNSIYC and NDNS years 2016/17 to 2018/19)

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[1]	208	202	0	0	1275
18 to 47 months[2]	248	214	0	0	306
48 to 60 months[2]	270	230	0	0	102

Abbreviations: LRNI, Lower Reference Nutrient Intake; RNI, Reference Nutrient Intake.
[1] Data from DNSIYC 2011 (DH, 2013).
[2] Data from NDNS 2016/17 to 2018/19.

4.257 No time trend data from NDNS is available for vitamin C.

Vitamin C intake and deprivation

4.258 Vitamin C intake by Index of Multiple Deprivation (IMD) for children aged 18 to 60 months is shown in Table 4.28. Mean vitamin C intake (from food sources only) ranged from 66.7 mg per day in quintile 5 (most deprived) to 73.9 mg per day in quintile 3. The confidence intervals do not indicate any significant differences in mean intake between IMD quintiles.

Table 4.28 Vitamin C intake (from diet only) by IMD quintile in children aged 18 to 60 months in England (NDNS years 2008/09 to 2018/19)

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 (90%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 2008/09 to 2018/19.

Dietary sources of vitamin C

4.259 The main dietary contributors to vitamin C intake in children aged 18 to 47 months, and 48 to 60 months from NDNS (years 2016/17 to 2018/19) are presented in Table 4.29 and Table 4.30.

Table 4.29. Contributors to vitamin C intake[1] in children aged 18 to 47 months

Food Group	% contribution[2,3]	mg per day
Fruit	24.1	18.0
Soft drinks[3]	16.8	12.3
Fruit juice and smoothies	12.9	12.2
Milk and cream[4]	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
Infant formula	3.0	3.2
Breakfast cereals	1.5	0.9
Commercial toddlers foods and drinks	1.2	1.0
Sugar and chocolate confectionery	1.0	0.9
Number of participants	306	306

[1] Data from NDNS years 2016/17 to 2018/19.
[2] Food groups that contribute less than 1% of intake are not presented.
[3] Average % contribution for each food group has been calculated from the % contribution for each individual. Non consumers are included in the average.
[4] Fizzy drinks, squashes and ready-to-drink still drinks, both those with added sugar and diet types
[5] Includes dairy alternatives.

Table 4.30. Contributors to vitamin C intake[1] in children aged 48 to 60 months

Food Group	% contribution[2,3]	mg/day
Fruit	22.6	19.7
Soft drinks[4]	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 and cream[5]	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

[1] Data from NDNS years 2016/17 to 2018/19.
[2] Food groups that contribute less than 1% of intake are not presented.
[3] Average % contribution for each food group has been calculated from the % contribution for each individual. Non consumers are included in the average.
[4] Fizzy drinks, squashes and ready-to-drink still drinks, both those with added sugar and diet types.
[5] Includes dairy alternatives.

4.260 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).

4.261 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 components, and dietary patterns

Background

5.1 Dietary or nutritional exposures can be examined using different approaches. These include investigating intakes of single nutrients and non-nutrients, consumption of individual foods and food groups, and dietary pattern analysis, which considers the whole diet. Dietary pattern analysis, which has gained in popularity since the early 2000s as a promising alternative in nutrition research, can take account of 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 sections: foods, dietary patterns and dietary (non-nutrient) components. Each section includes a short introduction to the topic area and a description of the identified evidence.

5.3 The limitations of the evidence base specific to each topic area are described in their corresponding section. However, a limitation that is applicable to the whole chapter is that the prospective cohort studies (PCS) included in the identified systematic reviews (SRs) did not always adjust for key potential confounding factors or key mediators and effect modifiers.

5.4 Key confounding factors are age, sex and socioeconomic status (SES). An additional confounding factor for outcomes that relate to or result from effects on energy balance (for example, body mass index [BMI]) is child baseline weight status (BMI or other anthropometric measurements). Primary studies (included in the SRs) that examined the health impact of specific foods did not always adjust for total dietary energy intake (TDEI) (see Chapter 3, paragraphs 3.44 and 3.45) or intake of other foods (Jacobs & Tapsell, 2007). Primary studies that examined cognition-related outcomes did not always adjust for child baseline cognition and parental cognition. A key mediator is maternal education, which is associated with healthier dietary patterns and has a key influence on children's diets (Emmett et al, 2015).

Foods

5.5 This section is divided into the following food groups which form the basis of current UK government dietary recommendations for the whole population:

- vegetables and fruit
- milk and dairy products
- foods rich in starchy carbohydrates
- non-dairy sources of protein
- foods high in fat, salt or sugars (HFSS)

Evidence identified on breastfeeding beyond the first year of life is also presented in this section.

Limitations of the evidence identified on 'foods'

5.6 There was a paucity of evidence from SRs on the health impact of different foods or food groups for children aged 1 to 5 years. Evidence was only identified on vegetables and fruit, milk and dairy, sugars-sweetened beverages (SSBs) (see Chapter 3) and breastfeeding beyond 12 months of age.

5.7 No evidence from SRs was identified on the health impact of sources of starchy carbohydrates or non-dairy protein, or HFSS intake in children 1 to 5 years old.

Vegetables and fruit

5.8 Vegetables and fruit are a good source of vitamins, minerals and dietary fibre (NHS, 2019a). The UK government advises 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) (see Chapter 11, Table 11.1). Fruit juice also counts towards the government recommendation but should be limited to 1 portion of 150ml a day because of high levels of free sugars (see Chapter 3, Carbohydrates section).

Consumption of vegetables and fruit in the UK

5.9 Data from the Diet and Nutrition Survey of Infants and Young Children (DNSIYC) and the National Diet and Nutrition Survey (NDNS years 2016/17 to 2018/19) on vegetables and fruit consumption in children in the UK aged 18 to 60 months are shown in Table 5.1. For total vegetables (excluding potatoes) and fruit (excluding fruit juice), children aged 18 to 47 months consumed, on average, 178 grams per day (18% TDEI) while children aged 48 to 60 months consumed, on average, 217

grams per day (21% TDEI). In both age groups, fruit made a greater contribution to total vegetables and fruit consumption than vegetables.

Table 5.1. Vegetables (excluding potatoes), fruit (excluding fruit juice) and fruit juice consumption[1] (grams per day) and their contribution to TDEI for children aged 12 to 60 months in the UK (DNSYIC and NDNS years 2016/17 to 2018/19)[1]

Food Group	12 to 18 months		18 to 47 months		48 to 60 months	
	Grams per day Mean (SD)	% Contribution to TDEI	Grams per day Mean (SD)	% Contribution to TDEI	Grams per day Mean (SD)	% Contribution to TDEI
Total Vegetables[2]	74 (46)	8.1	70 (42)	8.1	88 (55)	9.0
Total Fruit[3]	96 (69)	14.8	112 (74)	11.3	131 (93)	12.2
Total Vegetables and fruit[4]	170 (92)	22.3	178 (98)	18.3	217 (117)	20.6
Fruit juice[5]	13 (36)	0.6	81 (100)	2.8	71 (67)	2.2
Number of participants	1275	1275	306	306	102	102

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

[1] Data for children aged 12 to 18 months from DNSIYC 2011 (Lennox et al, 2013) otherwise data from NDNS 2016/17 to 2018/19.

[2] Includes vegetables in composite dishes and manufactured products.

[3] Includes fruit in composite dishes and manufactured products.

[4] Includes vegetables and fruit in composite dishes and manufactured products.

[5] Fruit juice covers 100% fruit juice and smoothies.

5.10 Time trend analysis of NDNS data (years 2008/09 to 2016/17) for children aged 18 to 47 months indicated no significant change in mean consumption of vegetables and fruit (not including fruit juice) (average change per year -2g, 95%CI -5.1 to 0.3g) for the 9 year period (Bates et al, 2019). No time trend data was available for the other 2 age groups.

Vegetables and fruit consumption by deprivation

5.11 Vegetables and fruit consumption by index of multiple deprivation (IMD) (see Annex 13, Glossary) for children aged 18 to 60 months are shown in Table 5.2. Table 5.3

Table 5.2. Vegetables and fruit consumption by IMD quintile in children aged 18 to 60 months in England[2] (NDNS years 2008/09 to 2018/19)

Food group	IMD quintile 1 (least deprived)	IMD quintile 2	IMD quintile 3	IMD quintile 4	IMD quintile 5 (most deprived)
Vegetables[1] mean (90%CI) (grams per day)	82 (75 to 88)	80 (73 to 86)	73 (67 to 79)	72 (66 to 79)	64 (59 to 69)
Fruit[2] mean (90%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

Data from NDNS years 2008/09 to 2018/19.

[1] Vegetables consumption excludes potatoes.

[2] Fruit consumption excludes fruit juice.

5.12 Vegetable consumption was lowest (64 grams per day) in quintile 5 (most deprived) and highest (82 grams per day) in quintile 1 (least deprived). The confidence intervals indicate that vegetable consumption was significantly higher in quintiles 1 and 2 than in quintile 5.

5.13 Fruit consumption was lowest (93 grams per day) in quintile 5 (most deprived) and highest (133 grams per day) in quintile 1 (least deprived). The confidence intervals indicate that fruit consumption was significantly higher in quintile 1 than in quintiles 4 and 5.

Evidence identified on vegetables and fruit

5.14 Two SRs without meta-analyses (MAs) (Frantsve-Hawley et al, 2017; Ledoux et al, 2011) were identified that included studies that examined the health impact of vegetables and fruit or fruit juice. Key outcomes were measures of body composition or weight status (BMI, body weight, body fat).

5.15 Details of the 2 SRs included in this section can be found in Annex 5 (Tables A5.1 and A5.3). Quality assessment of the SRs using the AMSTAR 2 tool can be found in Annex 7 (Tables A7.2 and A7.4). Additional data extracted on the primary studies can be found in Annex 8 (Table A8.2 and A8.24).

5.16 The criteria used to grade the evidence are provided in Chapter 2 (Table 2.4, paragraphs 2.43 to 2.47). Summary tables of the evidence grading process for this section are provided in Annex 9 (Tables A9.11 and A9.29).

5.17 All primary studies included in the SRs were from high-income countries (HICs).

Whole vegetables and fruit consumption and BMI or body weight

5.18 One SR without MA (Ledoux et al, 2011) (AMSTAR 2 confidence rating: critically low) was identified that examined the relationship between vegetable and fruit consumption and obesity outcomes. The SR included 2 PCS that examined this relationship in children aged 1 to 5 years.

5.19 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 (statistics NR). It should also be noted that, while 18% of girls and 23% of boys had overweight or obesity at baseline, the PCS did not adjust for baseline weight. This PCS also did not report the vegetable and fruit classification used in its analysis.

5.20 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 (statistics NR). The exposure did not include juice, carrots, potatoes and salads.

Whole vegetables and fruit consumption and body fat

5.21 No evidence was identified from SRs on the relationship between whole vegetables and fruit consumption and body fat in children aged 1 to 5 years.

Fruit juice consumption and BMI

5.22 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 reported below.

5.23 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.

5.24 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^2 (95% CI -0.05 to 0.20 kg/m^2) for a

consumption of 1 to 7 ounces of juice per day to 0.36 kg/m^2 (95% CI 0.08 to 0.64 kg/m^2) for consumption over 16 ounces per day after 6 years' follow-up.

5.25 The 3 PCS that reported an association tended to have longer follow-up durations than the 3 PCS that reported no association (2 to 6 years versus 6 months to 4 years).

5.26 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 3.44 and 3.45). 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.

5.27 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 8, Table A8.24 for details).

5.28 Most of the 7 PCS adjusted for multiple potential confounding factors including sex, a measure of baseline body size, ethnicity or SES.

Fruit juice consumption and body fat

5.29 No evidence was identified from SRs on the relationship between fruit juice consumption and body fat in children aged 1 to 5 years.

Summary: Fruit and vegetable consumption and body composition

5.30 The evidence identified from SRs on fruit and vegetable consumption and body composition is summarised in Table 5.3.

Table 5.3. Summary of the evidence on vegetables and fruit consumption and obesity outcomes

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Whole vegetables and fruit	BMI or body weight	N/A	Insufficient
Whole vegetables and fruit	Body fat	N/A	No evidence identified
Fruit Juice	Change in BMI (or BMI z-score)	↑ (non-TDEI adjusted) Null (TDEI-adjusted)	Limited[2]
Fruit Juice	Body fat	N/A	No evidence identified

Abbreviations: BMI, body mass index; N/A, not applicable; TDEI, total dietary energy intake.

[1] Direction of association for reported outcomes: ↑increase; ↓inverse; N/A: not enough evidence to draw conclusions and recommendations.

[2] Findings both unadjusted and adjusted for TDEI were graded separately as limited.

5.31 The available evidence on the relationship between consumption of whole vegetables and fruit and body composition is from 1 SR without MA given a critically low confidence rating using the AMSTAR 2 tool.

5.32 There was **insufficient** evidence to enable conclusions to be drawn on any relationship between whole vegetable and fruit 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.

5.33 The available evidence from SRs on the relationship between fruit juice consumption in children aged 1 to 5 years and body composition is from 1 SR without MA given a moderate confidence rating using the AMSTAR 2 tool.

5.34 Evidence from 3 PCS included in the SR by Frantsve-Hawley et al (2017) suggests that higher fruit juice consumption in children aged 1 to 5 years is associated with increased BMI in later childhood, unadjusted for TDEI. 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 suggests that there is no association between fruit juice consumption and later BMI 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 3.44 and 3.45). 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**.

5.35 There is **insufficient** evidence from SRs to enable conclusions to be drawn on any relationship between consumption of fruit juice in children aged 1 to 5 years and body fat in later childhood as fewer than 3 primary studies included in the SRs examined this relationship.

5.36 No evidence from SRs was identified on the impact of fruit and vegetable consumption on other health outcomes in children aged 1 to 5 years.

Milk and dairy

5.37 According to European Union regulations, the definition of milk is a mammary secretion of animals obtained from milking with the most common type being cows' milk (Dougkas et al, 2019). Throughout this draft report, the term 'milk' is used to refer to cows' milk. Milk and other dairy products such as cream, butter, cheese and yogurt are protected terms (Dougkas et al, 2019). Milk and dairy products are good sources of protein, calcium, vitamin A, riboflavin, iodine, phosphorus, potassium, zinc and vitamin B12, although the nutrient content is dependent on the type of product, geographical location, season, diet of the animals and husbandry practices (Dougkas et al, 2019; Haug et al, 2007; NHS, 2019a). Plain milk contains lactose, which is a sugar naturally present in milk and dairy products, and no free sugars (SACN, 2015). Milk is also an important source of energy for young children (NHS, 2021).

5.38 Current UK government recommendations are that children from the age of 1 year can be given whole cows' milk as a main drink and that from the age of 2 years, semi-skimmed milk can be gradually introduced to children who are growing well (see Chapter 11, Table 11.1).

Consumption of milk and dairy in the UK

5.39 Data from NDNS (years 2016/17 to 2018/19) on milk and dairy consumption for children aged 18 to 60 months in the UK are shown in Table 5.4. Mean total dairy consumption was 322 grams per day for children aged 18 to 47 months (contributing 22.3% TDEI) and 281 grams per day for children aged 48 to 60 months (contributing 15.2% TDEI). Total milk consumption contributed 16.1% to TDEI in the younger age group and decreased to 10.4% of TDEI in the older age group.

Table 5.4. Milk and dairy consumption (grams per day) and contribution to TDEI for children aged 18 to 60 months in the UK (NDNS years 2016/17 to 2018/19)

Food group	18 to 47 months		48 to 60 months	
	Grams per day (Mean SD)[5]	% Contribution to TDEI[5]	Grams per day (Mean SD)[5]	% Contribution to TDEI[5]
Total milk[1]	283 (197)	16.1	247 (212)	10.4
Cream and other milk products[2]	4 (4)	0.9	N/A[6]	N/A[6]
Cheese	14 (13)	4.4	13 (11)	3.5
Yogurt and fromage frais[3]	54 (45)	4.5	51 (36)	3.6
Total dairy[4]	322 (208)	22.3	281 (214)	15.2
Number of participants	306	306	102	102

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake.
Data from NDNS years 2016/17 to 2018/19.
[1] Total milk - all types of cows' milk and other dairy milk.
[2] Cream and other milk products – all types of dairy cream, dairy toppings, crème fraiche. For the 48 to 60 month age group, the number of consumers was too small for data to be presented.
[3] Yogurt and fromage frais – includes unsweetened and sugar-sweetened products.
[4] Total dairy: Total of milk, cream, cheese, yogurt and fromage frais.
[5] Excluding non-consumers.
[6] Too few consumers (fewer than 30) to present data.

Milk substitution analyses

5.40 Intake 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 (full fat) 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 yogurt, and dried milk were excluded. 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 take account of summer and winter values (Annex 11, Table A11.1).

5.41 Detailed results are presented in Annex 11 (Tables A11.2 to A11.4). 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. Tables A11.5 to A11.7 use the 5th and 95th percentiles of overall TDEI to present results for children with the highest and lowest TDEI.

5.42 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 11 (Tables A11.2 to A11.4).

5.43 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, for children with high and low TDEI (Tables A11.5 to A11.7).

5.44 The milk substitution analysis indicates 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. 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.

5.45 In contrast, the milk substitution analysis indicates that a move from whole milk to skimmed or 1% milk would result in a greater proportion of participants below the LRNI for vitamin A in all groups of TDEI with the greatest impact in children with the highest milk consumption (Table A11.11) and lowest TDEI (Table A11.12).

Plant-based drinks

5.46 Plant-based drinks, such as soya, oat and almond drinks, are becoming increasingly popular in the UK for a variety of reasons including cultural, environmental and ethical beliefs and values, personal preference, and as an alternative to cows' milk for people with cows' milk protein allergy or those who are lactose intolerant. There has also been an increase in the range of these drinks available on the market in the UK.

5.47 Evidence on plant-based drinks consumption in children will be summarised in the Joint SACN/COT risk assessment on plant-based drinks.

Evidence identified on milk and dairy

5.48 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 relationship between milk and dairy, and growth, obesity and other health outcomes.

5.49 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 7 (Tables A7.4 and A7.5). Additional data extracted on the primary studies can be found in Annex 8 (Table A8.25).

5.50 The criteria used to grade the evidence are provided in Chapter 2 (Table 2.4, paragraphs 2.43 to 2.47). Summary tables of the evidence grading process for this section are provided in Annex 9 (Tables A9.12, A9.13 and A9.29).

5.51 It should be noted that only 1 study (RCT) from the SR with MA (de Beer, 2012) met the inclusion criteria for this draft report.

5.52 Key outcomes examined were measures of body composition (BMI, weight, body fat) and weight status (overweight), linear growth (height), as well as bone health, blood pressure and cognitive development, which are presented under ‘other health outcomes’.

5.53 All primary studies included in the SRs were from HIC.

5.54 Some SRs did not specify whether the milk and dairy was only of bovine origin. However, all studies referred to milk as dairy not as dairy alternatives. All mammalian milk is considered dairy and cows’ milk is the most common type used (Dougkas et al, 2019).

Total dairy consumption and body composition or linear growth

BMI and body fat

5.55 One SR without MA (Dougkas et al, 2019) (AMSTAR 2 confidence rating: low) examined the relationship between dairy consumption and BMI or body fat in later childhood 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.

5.56 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 907.06g; SE 284.06; p=0.003) after 6 years of 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.

5.57 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.

5.58 One PCS (in 362 participants) reported that greater consumption of dairy products (as % TDEI) (compared with protein, meat and fruit consumption) at age 18 months was associated with a decrease in BMI (beta coefficient -0.21 kg/m^2; 95% CI 0.41 to 0.01 kg/m^2; p=0.04) at age 8 years. The analysis was adjusted for sex, birth weight, parental obesity status, ethnicity, smoking in pregnancy, paternal education.

5.59 Another PCS (in 335 participants) in the same cohort of children as the study described in paragraph 5.58 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

5.60 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 (statistics NR). The analysis was adjusted for child's age, sex, measures of SES, baseline weight status and TDEI.

Summary: total dairy consumption and growth and body composition

5.61 The evidence identified from SRs on total dairy consumption and body composition or linear growth is summarised in Table 5.5.

Table 5.5. Summary of the evidence on total dairy consumption and growth and obesity outcomes

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Total dairy consumption	BMI	N/A	Insufficient
Total dairy consumption	Body fat	N/A	Insufficient
Total dairy consumption	Linear growth	N/A	Insufficient

Abbreviations: BMI, body mass index; N/A, not applicable
[1] N/A: not enough evidence to draw conclusions and recommendations

5.62 The available evidence from SRs on the relationship between total dairy consumption and linear growth and body composition is from 1 SR given a critically low confidence rating using the AMSTAR 2 tool.

5.63 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**.

5.64 There was **insufficient** evidence to enable conclusions to be drawn on any relationship between total dairy consumption and linear growth or body fat in children 1 to 5 years as fewer than 3 primary studies included in the SR examined these relationships.

Total dairy and other health outcomes

Bone health

5.65 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^2) (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 [DXA]) at the time of the bone scan.

Summary: total dairy consumption and bone health

5.66 The evidence identified from SRs on total dairy consumption and bone health is summarised in Table 5.6.

Table 5.6. Summary of the evidence on dairy consumption and bone health

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Total dairy consumption	Bone mineral content (grams)	N/A	Insufficient
	Bone area (cm)	N/A	Insufficient

Abbreviations: N/A, not applicable
[1] N/A: not enough evidence to draw conclusions and recommendations

5.67 The available evidence on the relationship between total dairy consumption and bone health is from 1 SR given a critically low confidence rating using the AMSTAR 2 tool.

5.68 There was **insufficient** evidence 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

5.69 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 early childhood was associated with lower blood pressure in later childhood.

5.70 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.

5.71 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/day of dairy (beta coefficient 2.90 (SE 0.18) vs 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 (statistics 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

5.72 The evidence identified from SRs on dairy consumption and blood pressure is summarised in Table 5.7.

Table 5.7. Summary of the evidence on total dairy consumption and blood pressure

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Total dairy consumption	SBP	N/A	Insufficient
Total dairy consumption	DBP	N/A	Insufficient

Abbreviations: DBP, diastolic blood pressure; SBP, systolic blood pressure; N/A, not applicable
[1] N/A: not enough evidence to draw conclusions and recommendations.

5.73 The available evidence on the relationship between dairy consumption and blood pressure is from 1 SR given a critically low confidence rating using the AMSTAR 2 tool. There was **insufficient** evidence 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

5.74 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 (statistics 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

5.75 The evidence identified from SRs on total dairy consumption and cognitive ability is summarised in Table 5.8.

Table 5.8. Summary of the evidence on total dairy consumption and cognitive outcomes

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Total dairy consumption	Verbal cognitive outcomes	N/A	Insufficient

Abbreviations: N/A, not applicable.

[1] N/A: not enough evidence to draw conclusions and recommendations.

5.76 The available evidence on the relationship between dairy consumption and cognitive outcomes is from 1 SR given a critically low confidence rating using the AMSTAR 2 tool. There was **insufficient** evidence 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.

Milk consumption and body composition or weight status

5.77 One SR without MA (Dougkas et al, 2019) (AMSTAR 2 confidence rating: low) examined the relationship between milk consumption and body composition or weight status in childhood and included 6 PCS that examined this relationship in children aged 1 to 5 years.

5.78 Of the 6 PCS, 5 PCS (in total 20,418 participants) reported no association between milk consumption and BMI (or BMI z-score). The follow-up period in the 5 PCS ranged from 8 months to 4 years. All 5 PCS adjusted for sex and demographic factors (race or ethnicity); 4 studies adjusted for socioeconomic status; 3 studies adjusted for TDEI (see Chapter 3, paragraphs 3.44 and 3.45) and nutrient intake. For detailed results, see Annex 8, Table A8.25.

5.79 The sixth PCS (in 103 participants), which measured % body fat, reported that children in the highest tertile of milk consumption (411ml per day) between 3 to 5 years old had a lower % body fat compared with children in the lowest tertile of consumption (115ml per day) after 12 years of 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.

5.80 Two of the above PCS also examined the relationship between low-fat versus full-fat dairy product consumption in children aged 1 to 5 years and BMI in later childhood.

5.81 One PCS (in 852 participants) reported that full-fat milk consumption at age 2 years was associated with a decrease in BMI z-score at age 3 years (beta coefficient -0.09; 95% CI -0.16 to -0.01; p=0.02), adjusted for TDEI. There was no

association between reduced-fat milk consumption and BMI z-score (statistics NR). Both analyses were adjusted for age, sex, ethnicity, baseline BMI z-score, TDEI, and maternal education.

5.82 The second PCS (in 8300 participants) reported that there was no difference in change in BMI z-scores from ages 2 to 4 years between children who consumed full-fat 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 normal weight at baseline who consistently drank 1% fat or skimmed milk at ages 2 and 4 years had an increased odds of becoming overweight or obese during this time period (OR 1.57; 95% CI 1.03 to 2.42; p<0.05). The analyses were adjusted for sex, race or ethnicity and SES.

Summary: milk consumption and body composition or weight status

5.83 The evidence identified from SRs on milk consumption and body composition or weight status is summarised in Table 5.9.

Table 5.9 Summary of the evidence on milk consumption and body composition or weight status

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Total milk consumption	Body fat	N/A	Insufficient
Total milk consumption	BMI	Null	Moderate
Total milk consumption	Incident overweight	N/A	Insufficient
Skimmed/reduced-fat milk consumption	BMI	N/A	Insufficient
Full-fat milk consumption	BMI	N/A	Insufficient

Abbreviations: BMI, body mass index; N/A, not applicable.
[1] N/A: not enough evidence to draw conclusions and recommendations.

5.84 The available evidence on the relationship between milk consumption and growth and body composition or weight status is from 1 SR given a low confidence rating using the AMSTAR 2 tool.

5.85 There was **insufficient** evidence 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.

5.86 Evidence from 5 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 **moderate** rather than adequate due to the lack of confidence intervals and inconsistency in adjustment for confounders.

Other dairy products and weight status or linear growth

Overweight or obesity

5.87 One SR without MA (Dougkas et al, 2019) (AMSTAR 2 confidence rating: low) examined the relationship between consumption of other 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 (statistics NR). The analyses were adjusted for parental education and parental BMI.

Linear growth

5.88 One SR with MA (de Beer, 2012) (AMSTAR 2 confidence rating: critically low) examined the relationship between consumption of other dairy products 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.

5.89 The RCT (in 402 participants) reported that children (mean age 3.3 years at baseline) who received 125g of yogurt 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: other dairy products consumption and weight status or linear growth

5.90 The evidence identified from SRs on consumption of other dairy products and weight status or linear growth is summarised in Table 5.10.

Table 5.10 Summary of the evidence on other dairy products consumption and weight status or linear growth

Exposure	Outcome	Direction of effect or association[1]	Certainty of evidence
Cheese consumption	Overweight or obesity	N/A	Insufficient
Cream/crème fraiche consumption	Incident overweight	N/A	Insufficient

Yogurt consumption	Linear growth	N/A	Insufficient

Abbreviations: N/A, not applicable.
[1] N/A: not enough evidence to draw conclusions and recommendations.

5.91 The available evidence on the relationship between other dairy products and weight status and linear growth is from 1 SR given a critically low confidence rating using the AMSTAR 2 tool.

5.92 There was **insufficient** evidence to enable conclusions to be drawn on any relationship between other dairy products and obesity outcomes and linear growth in children aged 1 to 5 years as fewer than 3 primary studies included in the SR examined these relationships.

Foods rich in starchy carbohydrates

5.93 Starchy foods, such as potatoes, bread, rice, pasta and cereals, are the main sources of carbohydrates in the UK diet (see Chapter 3 on 'Carbohydrates'). These foods provide dietary energy and are good source of fibre, calcium, iron and B vitamins (NHS, 2020b).

5.94 Due a lack of absence 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. But from about age 6 months gradual diversification of the diet is encouraged, including increasing amounts of wholegrains (SACN, 2015). However, it is 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, 2019c). For recommendations on carbohydrates (total, free sugars and dietary fibre), see Chapter 3 and Chapter 11 (Table 11.1).

Consumption of foods rich in starchy carbohydrates consumption in the UK

5.95 Data from NDNS (years 2016/17 to 2018/19) on consumption of foods rich in starchy carbohydrates for children in the UK aged 18 to 60 months are shown in Table 5.11. Mean total consumption of foods rich in starchy carbohydrates was 117g per day for children aged 18 to 47 months (20.7% TDEI), and 126 grams per day for children aged 48 to 60 months (10.3% TDEI). Of the main sources of starchy carbohydrates examined, bread (wheat and non-wheat) made the largest contribution to TDEI (10.5%) for both age groups and breakfast cereals (with a

total sugars content less than 22.5 grams per 100g) made the second highest contribution to TDEI, at under 6% for both age groups.

Table 5.11. Consumption (grams per day) of foods rich in starchy carbohydrates consumption and their contribution to TDEI for children aged 18 to 60 months in the UK (NDNS years 2016/17 to 2018/19)

Food group	18 to 47 months		48 to 60 months	
	Grams per day Mean (SD)[7]	% Contribution to TDEI[7]	Grams per day Mean (SD)[7]	% Contribution to TDEI[7]
Bread[1]	43 (28)	10.5	49 (28)	10.5
Pasta[2]	38 (34)	4.5	34 (27)	3.8
Rice[3]	28 (32)	3.8	34 (32)	4.1
Potatoes[4]	33 (27)	2.6	39 (30)	2.6
Breakfast cereals sugar <22.5g per 100g[5]	20 (25)	5.9	19 (14)	5.5
All foods rich in starchy carbohydrates[6]	117 (57)	20.7	126 (65)	20.3
Number of participants	306	306	102	102

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake
Data from NDNS years 2016/17 to 2018/19.
[1] All types of wheat and non-wheat bread and rolls.
[2] Plain and filled pasta. Homemade pasta dishes and pasta based products and ready meals. Excludes meat based dishes including pasta.
[3] Plain rice, homemade rice based dishes and rice based products.
[4] Boiled, mashed, baked potatoes; homemade potato based dishes, instant, canned potatoes. Excludes chips, fried potatoes and fried potato products.
[5] All types of breakfast cereals with total sugars <22.5g per 100g (green or amber).
[6] Starchy carbohydrates: sum of bread, pasta, rice, potatoes, breakfast cereals green or amber for sugar
[7] Excluding non-consumers.

5.96 No evidence from SRs was identified on the health impact of consumption of foods rich in starchy carbohydrates in children aged 1 to 5 years. The evidence on carbohydrates in general can be found in Chapter 3.

Foods as non-dairy sources of protein

5.97 Non-dairy sources of protein include beans, pulses, fish, eggs, meat and other foods rich in protein. These foods are also rich in vitamins and minerals, but the nutritional profile varies by food category (for example, meat is a good source of iron and pulses are rich in fibre). See Chapter 3 for more details on protein.

5.98 The current UK government 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. See Chapter 3 and Chapter 11, Table 11.1 for details on protein recommendations.

Consumption of foods that are non-dairy sources of protein in the UK

5.99 Data from NDNS (years 2016/17 to 2018/19) on consumption of foods that are non-dairy sources of protein for children in the UK aged 18 to 60 months are shown in Table 5.12. Mean total consumption of foods that are non-dairy sources of protein was 61 grams per day for children aged 18 to 47 months (9% TDEI) and 74 grams per day for children aged 48 to 60 months (approximately 9% TDEI). Of the main non-dairy sources of protein examined, unprocessed meat made the highest contribution to TDEI in both age groups (approximately 8%). Children in the younger age group (18 to 47 months) consumed, on average, 27 grams per day of unprocessed meat while children in the older age group consumed, on average, 33 grams per day.

Table 5.12 Consumption (grams per day) of foods that are non-dairy sources of protein and their energy contribution to TDEI for children aged 18 to 60 months in the UK (NDNS years 2016/17 to 2018/19)

Food group	18 to 47 months		48 to 60 months	
	Grams per day Mean (SD)[7]	% Contribution to TDEI[7]	Grams per day Mean (SD)[7]	% Contribution to TDEI[7]
Unprocessed meat[1]	27 (19)	7.8	33 (23)	8.2
Fish[2]	14 (9)	4.0	15 (11)	3.1
Eggs[3]	19 (15)	2.7	20 (15)	2.5
Beans and pulses[4]	16 (16)	3.0	14 (15)	2.7
Plain nuts[5]	3 (4)	4.4	4 (7)	4.5

All non-dairy protein sources[6]	61 (36)	9.2	74 (48)	8.8
Number of participants	306	306	102	102

Abbreviations: SD, standard deviation; TDEI, total dietary energy intake.
Data from NDNS years 2016/17 to 2018/19.
[1] Beef, pork, lamb, chicken, turkey, offal. Includes meat components of manufactured products and dishes but excludes non-meat components.
[2] All types of fish and shellfish including in manufactured products and homemade dishes. Excludes non fish components of products and dishes.
[3] Boiled, fried, poached, scrambled eggs; omelettes. Includes manufactured products and dishes based on egg. Excludes quiches, scotch egg, meringues, souffles.
[4] All types of beans and pulses including baked beans. Includes components of manufactured products and homemade dishes.
[5] All types of unsalted uncoated nuts including nuts as components of manufactured products and homemade dishes, Excludes seeds.
[6] Non-dairy protein sources: sum of unprocessed meat, fish, eggs, beans and pulses, plain nuts
[7] Excluding non-consumers.

5.100 No evidence from SRs was identified on individual groups of foods that are non-dairy source of protein. See Chapter 3 for evidence on protein.

Breastfeeding beyond the first year of life

5.101 The composition of breast milk changes during lactation stages and can also vary between mothers. While the composition of breast milk during the first year postpartum has been extensively studied, there is limited evidence on the composition of breastmilk beyond the first and second years postpartum. However, findings from a study of breast milk composition suggested that protein and fat content were higher and carbohydrates content was lower in breast milk expressed by mothers who breast fed for longer than 12 months compared with those that breast fed for up to 12 months (Czosnykowska-Łukacka et al, 2018).

5.102 For recommendations on breastfeeding see the SACN report 'Feeding in the first year of life' (SACN, 2018) and UK government advice (NHS, 2019c; NHS, 2019d).

Evidence identified on breastfeeding beyond the first year of life

5.103 One SR with MA (Delgado & Matijasevich, 2013) was identified that included studies that examined the relationship between breastfeeding beyond the first year of life and growth, obesity or other health outcomes.

5.104 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 7

(Table A7.4). Additional data extracted on the primary studies can be found in Annex 8 (Table A8.26).

5.105 The criteria used to grade the evidence are provided in Chapter 2 (Table 2.4, paragraphs 2.43 to 2.47). Summary tables of the evidence grading process for this section are provided in Annex 9 (Tables A9.29).

5.106 Key outcomes examined were measures of growth (weight gain and linear growth) and cognitive development.

5.107 All primary studies included in the SRs were from lower middle income countries (LMICs) and low income countries (LICs), which may limit the generalisability of the findings to the UK population.

5.108 Further evidence on breastfeeding beyond the first year of life and oral health outcomes are in Chapter 8.

Breastfeeding beyond the first year of life and growth

5.109 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 measures. Both studies were conducted LMIC and LIC.

5.110 One PCS (in 28,753 participants) reported that children who were breastfed for 2 years or longer gained less weight between ages 24 to 36 months than those who were no longer receiving breastmilk at age 24 months (MD -205g; 95% CI -279 to -131g; p-value NR). The difference in weight gain was smaller in children from relatively affluent households (MD -38g; 95% CI -106 to 30g; p-value NR). Analyses were adjusted for sex and various baseline variables including child age, sex, dietary vitamin A intake, morbidity, household wealth, availability of water in the house, maternal literacy. It should be noted that the analyses combined children of normal and low nutritional status (wasting or stunting). The same PCS reported that the relationship between continued breastfeeding and nutritional status (wasting or stunting) was mediated by SES (household wealth) and maternal education.

5.111 One PCS (in 443 participants) reported that children who were breastfed between the ages of 21 to 26 months experienced greater linear growth over the 6-month period than children who had stopped breastfeeding by the beginning of the 6-month interval (MD 0.7cm; SD 0.3; $p<0.05$). Housing quality was a key modifier for the association between breastfeeding duration and linear growth. The SR stated that children living in poor housing who were breastfed during the 6-month period experienced greater linear growth than children living in poor housing who were no longer being breastfed by the beginning of the 6-month period. However, the opposite was true for children living in adequate housing. Analyses were adjusted for a season (wet or dry), quality of housing, initial age and weight.

Summary: breastfeeding beyond the first year of life and growth

5.112 The evidence identified from SRs on breastfeeding beyond the first year of life and growth is summarised in Table 5.13.

Table 5.13. Summary of the evidence on breastfeeding beyond 12 months of age and growth and obesity outcomes

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Breastfeeding beyond 12 months	Weight gain	N/A	Insufficient
Breastfeeding beyond 12 months	Linear growth	N/A	Insufficient

Abbreviations: N/A, not applicable.
[1] N/A: not enough evidence to draw conclusions and recommendations.

5.113 The available evidence 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.

5.114 There was **insufficient** evidence 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 outcomes

5.115 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 child development outcomes. The studies were conducted in a LMIC.

5.116 One PCS (in 1979 participants) examined the relationship between breastfeeding for 2 years and beyond compared with breastfeeding for 0 to less than 6 months and cognitive development measured by cognitive ability score. The study reported no association between breastfeeding duration and cognitive ability scores at ages 8.5 years and 11.5 years (statistics NR). The analyses were adjusted for sex, various measures of SES, maternal age, maternal alcohol use in pregnancy and preterm status of child.

5.117 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 beyond compared with children who were breastfed for 0 to 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 outcomes

5.118 The evidence identified from SRs on breastfeeding beyond the first year of life and cognitive outcomes is summarised in Table 5.14.

Table 5.14. Summary of the evidence on breastfeeding beyond 12 months of age and growth and obesity outcomes

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Breastfeeding beyond 12 months	Cognitive development	N/A	Insufficient
Breastfeeding beyond 12 months	Psychosocial development	N/A	Insufficient

Abbreviations: N/A, not applicable.
[1] N/A: not enough evidence to draw conclusions and recommendations.

5.119 The available evidence 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 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.

Foods high in fat, salt or free sugars

5.120 Foods that are high in saturated fat, salt or free sugars (HFSS) (such as sugars-sweetened beverages (SSBs), confectionery, cakes and biscuits, salty snacks such as crisps and sausage rolls, meat products such as sausages and salami, ice-cream and some breakfast cereals) are not essential in the human diet and should be eaten less often and in small amounts (NHS, 2019a; NHS, 2020a). In 2015, SACN recommended that the consumption of SSBs should be minimised in children (SACN, 2015). For current recommendations on free sugars, total dietary fat and saturated fats see Chapter 3.

5.121 Foods high in saturated fat, salt or sugar is a description that has been used in the UK since 2007 as part of the Nutrient Profiling Model, a tool (developed by the UK Foods Standards Agency) to help television broadcasters restrict advertisements of unhealthy foods to children. However, such foods have also been referred to as ‘processed foods’ or ‘ultra-processed foods’, for which there are no universally agreed definitions (DH, 2011).

Consumption of foods high in (total) fat, salt or free sugars (HFSS) in the UK

5.122 Data from NDNS (years 2016/17 to 2018/19) on the consumption of HFSS foods for children in the UK aged 18 to 60 months are shown in Table 5.15. Mean total consumption of HFSS foods was 114 grams per day for children aged 18 to 47 months (26.9% TDEI) and 179 grams per day for children aged 48 to 60 months (34.1% TDEI). Of the HFSS foods that were examined, baked goods (biscuits, buns, cakes and pastries) made the highest contribution to TDEI, contributing 9.3% and 10.7% to TDEI in the younger and older age groups, respectively.

Table 5.15. Consumption (grams per day) of HFSS foods and their contribution to TDEI for children aged 18 to 60 months in the UK (NDNS years 2016/17 to 2018/19)

Food group	18 to 47 months		48 to 60 months	
	Grams per day Mean (SD)[1]	% Contribution to TDEI[1]	Grams per day Mean (SD)[1]	% Contribution to TDEI[1]
Sugar, preserves, sweet spreads	5 (6)	1.8	9 (8)	2.5
Confectionery	12 (12)	4.3	18 (13)	5.3
Sugar-sweetened soft drinks	88 (105)	1.7	N/A[4]	N/A[4]
Breakfast cereals high sugar[2]	11 (8)	4.1	14 (10)	4.8
Biscuits, buns, cakes, pastries	24 (20)	9.3	31 (21)	10.7
Puddings	30 (24)	2.9	41 (28)	3.8
Crisps and savoury snacks	8 (7)	4.0	11 (7)	4.3
Salted nuts	N/A[4]	N/A[4]	N/A[4]	N/A[4]
Ice cream	18 (13)	3.0	24 (20)	3.7
Chips and fried potato products	20 (17)	3.8	25 (18)	4.1
Processed meat[3]	20 (16)	7.1	26 (21)	7.9
Flavoured milks	N/A[4]	N/A[4]	N/A[4]	N/A[4]
All HFSS foods	114 (85)	26.9	179 (122)	34.1
Number of participants	306	306	102	102

Abbreviations: HFSS, foods high in (total) fat salt free sugars; N/A, not applicable; SD, standard deviation; TDEI, total dietary energy intake.

Data from NDNS years 2016/17 to 2018/19.

1 Excluding non-consumers.

2 Products with sugar content >22.5g per 100g.

3 Bacon, ham, sausages, burgers and kebabs, meat pies.

4 Too few consumers (fewer than 30) to present data.

5.123 Evidence from SRs on the health impact of SSBs is described in Chapter 3 (within the ‘Carbohydrates’ section). No other evidence from SRs was identified on the health impact of other types of HFSS considered individually in children aged 1 to 5 years. However, evidence from SRs was identified on the health impact of dietary patterns characterised by the consumption of HFSS (using the term ‘processed foods’) and is described in the dietary patterns section (See section on Diet quality, page 217).

Dietary patterns

Background

5.124 Dietary pattern analysis is used to examine dietary behaviours of populations representing the combinations of foods and nutrients that are eaten in real life and reflecting the whole diet of individuals (Schulz et al, 2021). Many dietary patterns provide an indication of adherence to dietary guidelines or the overall 'healthiness' of a diet, commonly described as diet 'quality' (Gherasim et al, 2020). Dietary patterns analysis can also identify other types of dietary patterns depending on the aim and methods (Ocke, 2013).

5.125 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).

5.126 Hypothesis-driven approaches (also known as a prior 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 some subjectivity and therefore their use is not entirely objective (Gherasim et al, 2020).

5.127 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 also have limitations. Many DQIs are based on dietary guidelines that are population specific, which may limit their applicability and generalisability. Also, it can be difficult to compare various scoring systems (Gherasim et al, 2020; Gil et al, 2015). Furthermore, only a few DQIs have been assessed for validity and reliability in children or have assessed prospective health outcomes. A recent SR (Dalwood et al, 2020) that examined the validity and reliability of DQIs in children up to age 18 years reported that higher diet quality measured by 'rigorously developed' DQIs was associated with improved outcomes such as nutritional status. However, the SR concluded that more longitudinal studies were required to ascertain the ability of DQIs to assess optimal growth and other health-related outcomes in children and adolescents (Dalwood et al, 2020).

5.128 Exploratory approaches (also known as a posterior approaches) do not begin from predefined dietary patterns but instead 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 (PCA). The identified dietary patterns are labelled 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 to dietary guidelines (reflecting diet quality) in the specific population based on the characteristics of the identified patterns. 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 have limited generalisability to other populations (Schulz et al, 2021).

5.129 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 (RRR) (Gherasim et al, 2020; Ocke, 2013; Schulz et al, 2021).

5.130 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).

5.131 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).

Evidence identified on diet quality

5.132 In this draft report, dietary patterns that were identified in the SR evidence were divided into 2 groups: dietary patterns that could be considered to reflect diet quality (see paragraphs 5.124 to 5.128) (evidence described from page 217) and other dietary patterns (evidence described from page 223).

Evidence identified on diet quality

5.133 Two SRs without MAs (Costa et al, 2018; Tandon et al, 2016) were identified that included studies that examined the relationship between diet quality and obesity-related and other health outcomes.

5.134 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 7 (Tables A7.4 and A7.5). Additional data extracted on the primary studies can be found in Annex 8 (Table A8.27).

5.135 The criteria used to grade the evidence are provided in Chapter 2 (Table 2.4, paragraphs 2.43 to 2.47). Summary tables of the evidence grading process for this section are provided in Annex 9 (Tables A9.14, A9.15 and A9.29).

5.136 Key outcomes examined were body composition (body fat) and cognitive development.

5.137 Among the primary studies included in the SRs, 1 assessed diet quality by DQI (see paragraphs 5.126 and 5.127) while the remaining primary studies used exploratory approaches (see paragraph 5.128) to derive dietary patterns that could be considered to assess diet quality. The primary studies used a variety of names to describe the derived dietary patterns, including ‘junk food dietary pattern’, ‘discretionary dietary pattern’, ‘health-conscious dietary pattern’, and ‘nutrient-dense dietary pattern’. For this draft report, the derived dietary patterns were categorised into ‘healthy’ and ‘unhealthy’ dietary patterns (See Annex 8 Table A8.29) based on the descriptions used in the primary studies and their similarity to current UK dietary recommendations.

5.138 All primary studies included in the SRs were from HICs.

Diet quality and body composition

5.139 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 and body composition and included 3 PCS that examined this relationship in children aged 1 to 5 years.

5.140 One PCS (in 292 participants) reported that children aged 3.8 to 4.8 years who scored in the highest quartile for the dietary pattern “that contained mostly ultra-processed foods” (as described by the SR and identified using reduced rank regression) had higher fat mass (kg) than 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, height, exact age, TDEI, calcium intake, accelerometer counts per minute, TV viewing time, outdoor playtime.

5.141 One PCS (in 585 participants) reported that among boys (196 included in the analysis), a dietary pattern described by the SR as '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 feeding environments (such as day-care centers and schools) as the aim of the SR was to focus on eating within the family settings.

5.142 One PCS (in 4750 participants) reported that higher scores for a 'junk food dietary pattern' (as described by the SR and identified through PCA) at age 38 months was associated with an increase in 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 3.44 and 3.45), 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

5.143 The evidence identified from SRs on the relationship between 'unhealthy' dietary patterns (see paragraph 5.137) and body composition is summarised in Table 5.16.

Table 5.16. Summary of the evidence on diet quality and body composition

Exposure	Outcome	Direction of association[1]	Certainty of evidence
'Unhealthy' dietary pattern[2]	Body fat	↑	Limited

[1] Direction of association for reported outcomes: ↑increase; ↓inverse.
[2] See paragraph 5.137.

5.144 The available evidence on the relationship between dietary patterns characterised as 'unhealthy' and body composition is from 1 SR (Costa et al, 2018) given a moderate confidence rating using the AMSTAR 2 tool.

5.145 Evidence from 3 PCS included in the SR without MA by Costa et al (2018) suggests that greater adherence to 'unhealthy' dietary patterns in children aged 1 to 5 years are associated with greater adiposity (body fat) in later childhood. The evidence was graded as **limited** given that there were 3 PCS providing evidence of a consistent direction of association.

Diet quality and cognitive outcomes

5.146 One SR without MA (Tandon et al, 2016) was identified that examined the relationship between diet quality and cognitive outcomes. 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 Annex 13, Glossary) and Intelligence Quotient (IQ).

5.147 One PCS (in 1346 participants) examined the relationship between diet quality (measured by the Eating Assessment in Toddlers [EAT] diet score; see Annex 13, 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).

5.148 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 (statistics NR) and that a ‘healthy dietary pattern’ was “weakly” associated with higher IQ at age 8 years but not at age 15 years (statistics NR). The analyses were adjusted for maternal characteristics (age, education, SES, tobacco use during pregnancy), ethnicity, duration of breastfeeding.

5.149 Another study (in 1366 participants) using the same dataset as the study described in paragraph 5.148 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 (statistics NR). The analyses were adjusted for maternal characteristics (age, education, SES, tobacco use during pregnancy), ethnicity and duration of breastfeeding.

5.150 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 PCA)

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 this dietary pattern at age 3 years was associated with a decrease in IQ at age 8.5 years (statistics NR). The analyses were 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.

5.151 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 Annex 13, 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 (statistics NR) but that adherence to the 'health-conscious dietary pattern' at age 38 months was not associated with KS2 results (statistics NR). Results for ages 54 and 81 months were not reported. The analysis was 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

5.152 The evidence identified from SRs on diet quality and cognitive outcomes is summarised in Table 5.17.

Table 5.17. Summary of the evidence on diet quality and cognitive outcomes

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Diet quality measured by score or index	Receptive vocabulary	N/A	Insufficient
Diet quality measured by score or index	Non-verbal vocabulary	N/A	Insufficient
'Healthy' dietary pattern[2]	IQ	N/A	Insufficient

Exposure	Outcome	Direction of association[1]	Certainty of evidence
'Healthy' dietary pattern[2]	Verbal IQ	N/A	Insufficient
'Healthy' dietary pattern[2]	KS2 performance	N/A	Insufficient
'Unhealthy' dietary pattern[2]	IQ	N/A	Insufficient
'Unhealthy' dietary pattern[2]	Verbal IQ	N/A	Insufficient
'Unhealthy' dietary pattern[2]	KS2 results	N/A	Insufficient

Abbreviations: IQ, intelligence quotient; KS2, key stage 2; N/A, not applicable.
[1] N/A: not enough evidence to draw conclusions and recommendations.
[2] See paragraph 5.137.

5.153 The available evidence on the relationship between diet quality and cognitive outcomes is from 1 SR without MA given a critically low confidence rating using the AMSTAR 2 tool.

5.154 There was **insufficient** evidence 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.

5.155 There was also **insufficient** evidence 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.

5.156 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 was graded **insufficient** and no conclusions can be drawn on.

Other dietary patterns

5.157 This section describes the evidence identified from SRs on dietary patterns that did not describe differences in 'diet quality' (as defined on page 215). 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.

5.158 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 draft report.

Vegetarian and vegan diets

5.159 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 has 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 vegan diet, whereas in 2019 it was 1.21% (The Vegan Society, 2022).

5.160 Vegetarian diets exclude foods derived from animal flesh such as meat, poultry, seafood and their products. 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 vegetables and fruit, grains, pulses, nuts and seeds (Baroni et al, 2019).

5.161 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).

5.162 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 status which in extreme cases, can cause long term malnutrition and adversely affect growth and development (Dagnelie & van Staveren, 1994; DH, 1994b).

5.163 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 non-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 than 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 evidence of harm arises from case studies and case series (Kiely, 2021; Lemoine et al, 2020). 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).

Evidence identified on other dietary patterns

5.164 One SR without MA (Tandon et al, 2016) was identified that included studies that examined the relationship between adherence to dietary patterns variously described by the SR as ‘snacking’, ‘ready-to-eat’, ‘freshly-cooked’ and ‘traditional’ dietary patterns, and their relationship with cognitive development.

5.165 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 7 (Table A7.5). Additional data extracted on the primary studies can be found in Annex 8 (Table A8.28).

5.166 The criteria used to grade the evidence are provided in Chapter 2 (Table 2.4, paragraphs 2.43 to 2.47). Summary tables of the evidence grading process for this section are provided in Annex 9 (Table A9.29).

5.167 All primary studies included in the SRs were from HICs.

‘Snacking’ and IQ

5.168 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 (statistics 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

5.169 The evidence identified from SRs on ‘snacking’ and IQ is summarised in Table 5.18.

Table 5.18. Summary of the evidence on 'snacking' and IQ

Exposure	Outcome	Direction of effect/association[1]	Certainty of evidence
'Snacking'[2]	IQ	N/A	Insufficient

Abbreviations: IQ, intelligence quotient; N/A, not applicable.
[1] N/A: not enough evidence to draw conclusions and recommendations.
[2] Defined and characterised by the SR (Tandon et al, 2016).

5.170 The available evidence on the relationship between 'snacking' and IQ is from 1 SR given a critically low confidence rating using the AMSTAR 2 tool. There was **insufficient** evidence 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

5.171 Tandon et al (2016) included 3 PCS that examined the relationship between adherence to 'ready-to-eat or freshly cooked' dietary patterns (a phrase used in the SR) by children aged 1 to 5 years and IQ in later childhood.

5.172 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 commercially manufactured foods for children at age 15 months; and 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 (statistics NR). The analysis was adjusted for maternal characteristics (age, education, SES, tobacco use during pregnancy), ethnicity, and duration of breastfeeding.

5.173 The second study (in 1366 participants), which used the same dataset as the study described in the previous paragraph (5.127), examined the relationship between adherence to 'ready-to-eat' or 'ready-to-eat baby foods' dietary patterns (as described by the SR) 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 (statistics 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 (statistics NR). The analyses were adjusted for maternal characteristics (age, education, SES, marital status, tobacco use) ethnicity.

5.174 One PCS (in 5217 participants) examined the relationship between adherence to a 'freshly cooked' dietary pattern (as described by the SR) 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 with 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 (statistics NR) and higher cognitive performance at age 5 years (statistics 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

5.175 The evidence identified from SRs on the relationship between adherence to 'ready-to-eat or freshly cooked' dietary patterns and IQ is summarised in Table 5.19.

Table 5.19. Summary of the evidence on 'ready-to-eat or freshly cooked' dietary patterns and cognitive outcomes

Exposure	Outcome	Direction of effect/association[1]	Certainty of evidence
'Ready-to-eat' dietary pattern[2]	IQ	N/A	Insufficient
'Ready-to-eat' dietary pattern[2]	Verbal IQ	N/A	Insufficient
'Ready prepared baby foods' dietary pattern[2]	IQ	N/A	Insufficient
'Ready prepared baby foods' dietary pattern[2]	Verbal IQ	N/A	Insufficient
'Freshly cooked' dietary pattern[2]	Vocabulary	N/A	Insufficient
'Freshly cooked' dietary pattern[2]	Cognitive performance	N/A	Insufficient

Abbreviations: IQ, intelligence quotient; N/A, not applicable.

[1] N/A: not enough evidence to draw conclusions and recommendations.

[2] Defined and characterised by the SR (Tandon et al, 2016).

5.176 The available evidence on the relationship between adherence to 'ready-to-eat or freshly cooked' dietary patterns and IQ is from 1 SR given a critically low confidence rating using the AMSTAR 2 tool.

5.177 There was **insufficient** evidence 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

5.178 Tandon et al (2016) included 1 PCS that examined the relationship between adherence to a 'traditional' dietary pattern (a phrase used in the SR) by children aged 1 to 5 years and IQ in later childhood. 'Traditional' dietary patterns were characterised by meat, cooked vegetables, and puddings.

5.179 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 (statistics 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

5.180 The evidence identified from SRs on the relationship between adherence to a 'traditional' dietary pattern and IQ is summarised in Table 5.20.

Table 5.20. Summary of the evidence on 'traditional' dietary patterns and IQ

Exposure	Outcome	Direction of association[1]	Certainty of evidence
'Traditional' dietary pattern[2]	IQ	N/A	Insufficient

Abbreviations: IQ, intelligence quotient; N/A, not applicable.

[1] N/A: not enough evidence to draw conclusions and recommendations.

[2] Defined and characterised by the SR (Tandon et al, 2016).

5.181 The available evidence on the relationship between a 'traditional' dietary pattern and IQ is from 1 SR given a critically low confidence rating using the AMSTAR 2 tool.

5.182 There was **insufficient** evidence 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

5.183 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

5.184 The term ‘probiotics’ is scientifically defined as a product containing living microorganisms, usually bacteria, which can provide health benefits to the host when consumed in adequate amounts (Bibek & Bhunta, 2008).

5.185 The most common microorganisms considered to be beneficial are the bacterial species Lactobacillus and Bifidobacterium (Guarner et al, 2017). Often specific strains of Lactobacillus acidophilus and Lactobacillus reuteri are used in probiotic products (Bibek & Bhunta, 2008). The largest group of products containing probiotics are fermented dairy products such as yogurt, kefir and cheese (Douglas & Sanders, 2008). New products containing probiotics are being developed, including granola bars, fruit juices and ice-cream (Vandenplas et al, 2014).

5.186 Some infant or young child formula (see Annex 13, Glossary) are supplemented with probiotics although little is known of their effects in this age group. ESPGHAN reviewed the existing evidence on probiotics in infant formula and concluded that there was a lack of data on long term health effects. Although the evidence suggested that probiotic-supplemented formula for healthy infants did not raise safety concerns, ESPGHAN does not recommend routine use of probiotic-supplemented formula (Braegger et al, 2011).

Guidance on probiotic use

5.187 FAO/WHO guidelines on probiotics in food state that in order for a product to gain approved probiotic status, the genus, species and strain level of the bacteria needs to be assessed, must be resistant to gastric acid and digestive enzymes, and has to be proven to be safe to the consumer. In addition, the health effects in the host must be assessed (WHO and FAO, 2006).

5.188 The UK government states that probiotics “appear to be safe” and children with healthy immune systems can use them (NHS, 2018). At an international level, the use of probiotics is also generally regarded as safe, however a risk of pathogenic effects of consumption of large doses has been recognised (Vandenplas et al, 2015). In the European Union, the word ‘probiotics’ and any health claims about their effectiveness, are not permitted in food labelling (Reid, 2016). Therefore,

products containing the term ‘probiotics’ in their description can only be obtained as food supplements. The European Society for Primary Care Gastroenterology (ESPCG) has provided a guide to these products (Hungin et al, 2013).

Evidence identified on probiotics

5.189 One SR with MA (Onubi et al, 2015) were identified that included studies that examined the relationship between probiotics and growth (linear growth and weight gain).

5.190 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 7 (Table A7.6). Additional data extracted on the primary studies can be found in Annex 8 (Table A8.30).

5.191 The criteria used to grade the evidence are provided in Chapter 2 (Table 2.4, paragraphs 2.43 to 2.47). Summary tables of the evidence grading process for this section are provided in Annex 9 (Table A9.29).

5.192 From the 2 primary studies included in the SR, 1 RCT was from HIC and 1 from UMIC.

Probiotics and growth outcomes

5.193 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 HIC and the other was conducted in an upper middle income country (UMIC).

5.194 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 (1x107 Bifidobacterium lactis Bb12 and Streptococcus thermophilus CFU per gram) in a standard milk-based formula while the second group received a low dose probiotic (1x106 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 (statistics NR).

5.195 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 (statistics 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

5.196 The evidence identified from SRs on probiotics and growth outcomes is summarised in Table 5.21.

Table 5.21. Summary of the evidence on probiotics and growth outcomes

Exposure	Outcome	Direction of effect[1]	Certainty of evidence
Probiotics	Change in body weight or WAZ	N/A	Insufficient
Probiotics	Linear growth	N/A	Insufficient
Probiotics	Change in WLZ	N/A	Insufficient

Abbreviations: N/A; not applicable; WAZ, weight-for-age z-score; WLZ, weight-for-length z-score.
[1] N/A: not enough evidence to draw conclusions and recommendations.

5.197 The available evidence on the relationship between probiotics and growth outcomes (change in body weight or WAZ or WLZ) and linear growth is from 1 SR given a low confidence rating using the AMSTAR 2 tool. There was **insufficient** evidence 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 and non-caloric sweeteners

Introduction

5.198 Low and non-caloric sweeteners (LNCS), are a range of artificial or nature-derived chemical substances that can be used to sweeten foods and drinks in place of 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 caloric 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). LNCS approved for use in the UK include for example acesulfame K, aspartame, saccharin, sorbitol, sucralose, steviol glycosides, thaumatin and xylitol (FSA, 2022; NHS, 2019b).

5.199 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).

5.200 There is a lack agreed terminology on LNCS. In the evidence section below, the term ‘non-nutritive sweeteners’ was used to describe the evidence identified based on the terminology used by the SR.

Evidence identified on ‘non-nutritive sweeteners’

5.201 For this draft report, 1 SR with MA (Karalexi et al, 2018) was identified that examined the relationship between consumption of ‘non-nutritive sweeteners’ (term used by the SR) and metabolic health outcomes.

5.202 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 7 (Table A7.6). Additional data extracted on the primary studies can be found in Annex 8 (Table A8.30).

5.203 The criteria used to grade the evidence are provided in Chapter 2 (Table 2.4, paragraphs 2.43 to 2.47). Summary tables of the evidence grading process for this section are provided in Annex 9 (Table A9.29).

5.204 All primary studies included in the SRs were from HIC.

‘Non-nutritive sweeteners’ and BMI

5.205 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 (in a total of 1522 participants) was ‘diet soda’ (term used by the SR). 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, the SR authors calculated a new estimate of association (odds ratio) for pooling into a MA and it is unclear whether this estimate is crude or adjusted. The other PCS adjusted for TDEI at age 3 years (see Chapter 3, paragraphs 3.44 and 3.45).

Summary: 'non-nutritive sweeteners' and BMI

5.206 The evidence identified from SRs on consumption of 'non-nutritive sweeteners' and growth and BMI is summarised in Table 5.22.

Table 5.22. Summary of the evidence on 'non-nutritive sweeteners' and obesity outcomes

Exposure	Outcome	Direction of association[1]	Certainty of evidence
'Non-nutritive sweeteners'	BMI (or BMI z-score)	N/A	Insufficient

Abbreviations: BMI, body mass index; N/A, not applicable.
[1] N/A: not enough evidence to draw conclusions and recommendations.

5.207 The available evidence on the relationship between 'non-nutritive sweeteners' and BMI (or BMI z-score) is from 1 SR given a low confidence rating using the AMSTAR 2 tool. There was **insufficient** evidence to enable conclusions to be drawn on any relationship between '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.

'Non-nutritive sweeteners' and type 1 diabetes

5.208 One SR (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), and reported no association with islet immunity and progression to type 1 diabetes after 10.2 years of follow up. The analysis adjusted for genotype associated with autoimmune diseases (see Annex 8, Table A8.30 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: 'non-nutritive sweeteners' and type 1 diabetes

5.209 The evidence identified from SRs on 'non-nutritive sweeteners' and outcomes related to type 1 diabetes is summarised in Table 5.23.

Table 5.23. Summary of the evidence on 'non-nutritive sweeteners' and type 1 diabetes

Exposure	Outcome	Direction of association[1]	Certainty of evidence
'Non-nutritive sweeteners'	Islet autoimmunity	N/A	Insufficient
	Progression to type 1 diabetes	N/A	Insufficient

Abbreviations: N/A, not applicable.
[1] N/A: not enough evidence to draw conclusions and recommendations.

5.210 The available evidence on the relationship between 'non-nutritive sweeteners' and outcomes related to type 1 diabetes is from 1 SR given a low confidence rating using the AMSTAR 2 tool. There was **insufficient** evidence to enable conclusions to be drawn on any relationship between '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 Eating and feeding behaviours

Background

6.1 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).

6.2 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).

6.3 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 'picky' eating or food fussiness, 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).

6.4 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).

6.5 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).

6.6 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).

6.7 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.

6.8 A distinction has been made between general caregiver or parenting styles and practices (Peters et al, 2012; Vollmer & Mobley, 2013; Wood et al, 2020).

6.9 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).

6.10 When applied to specific eating and feeding interactions, caregiver or parenting styles have been termed 'feeding styles' (Vollmer & Mobley, 2013).

6.11 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.

6.12 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). 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).

6.13 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).

6.14 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.

Evidence identified on children's eating behaviours and weight status

6.15 Three SRs without MAs were identified that examined the relationship between children's eating behaviours and body composition (BMI) or weight status in later childhood and adolescence (Blondin et al, 2016; Brown et al, 2016; Caleza et al, 2016). The eating behaviours covered in these SRs that was specific to children aged 1 to 5 years were picky eating or food fussiness, ability to delay gratification, and breakfast consumption. For this draft report, the term 'picky eating' was used to describe the evidence as this was the term employed by the relevant SR.

6.16 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 7 (Table A7.7). Additional data extracted on the primary studies can be found in Annex 8 (Table A8.31). The criteria used to grade the evidence are provided in Chapter 2 (Table 2.4, paragraphs 2.43 to 2.47). Evidence grades are provided in Annex 9 (Table A9.29) and summarised at the end of this section.

Limitations of the evidence on eating behaviours

6.17 Across the SRs, there was a paucity of large, adequately powered RCTs of sufficient length to capture habitual behaviour.

6.18 Many PCS included in the SRs did not adjust for potential confounding factors, which included 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), which potentially removes the mechanism by which children's eating behaviours may impact on their body weight.

6.19 Primary studies also lacked consistent use of terminology or standardised definitions for key exposures (for example, picky eating or food fussiness, food neophobia) as well as standardised instrument(s) to measure or assess eating and feeding behaviours. This limited the ability to combine data for meta-analysis or draw overarching conclusions.

6.20 The areas covered in 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 or eating in the absence of hunger in children aged 1 to 5 years on nutritional or weight status. Similarly, no SR evidence was identified on informal eating behaviours (including snacking and eating while watching television) on dietary intake or weight status of children in this age group.

Children's eating behaviours and body composition or weight status

Picky eating

6.21 One SR without MA (Brown et al, 2016) (AMSTAR 2 confidence rating: moderate) examined the relationship between picky eating and weight status in children and adolescents and included 4 PCS in children aged 1 to 5 years.

6.22 One PCS (in 156 participants) reported no association between picky eating (measured by the Child Eating Behaviour Questionnaire) at ages 2 to 4 years and BMI z-score at ages 3 to 5 years, after adjusting for baseline child BMI z-score, age, sex and maternal characteristics (age, BMI and education).

6.23 Two PCS examined the relationship between picky eating and change in weight status over time. One PCS (in 486 participants) reported no association between picky eating (identified through cluster analysis) at age 1 year and change in BMI z-score from ages 1 to 3 years. The other PCS (in 135 participants) reported no association between picky eating (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 picky eating at age 4 years experienced a greater increase in BMI over 1 year (from 15.3 to 15.7kg/m^2) compared with girls without picky eating (from 16.4 to 16.3kg/m^2). There was no evidence of difference in change in BMI between boys with and without picky eating. The analyses from both PCS were not adjusted for potential confounding factors other than sex, however, in both PCS, children with picky eating at baseline were lighter than children without picky eating.

6.24 The fourth PCS (in 1498 participants) reported that children with picky eating at ages 2.5, 3.5 and 4.5 years had an increased odds of being underweight at age 4.5 years compared with children who were never picky (OR 2.4; 95% CI 1.4 to 4.2; p-value NR). However, there was no association with weight status if children were picky at one or 2 of the ages when measurements were taken, compared with children who were never picky (statistics 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

6.25 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.

6.26 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.

6.27 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 (statistics NR). 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 lower BMI) cannot be ruled out.

Breakfast consumption

6.28 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

6.29 The evidence on the relationship between children's eating behaviours and body composition or weight status is summarised in Table 6.1.

Table 6.1. Summary of the evidence on children's eating behaviours and body composition or weight status.

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Picky eating	BMI z-score	N/A	Insufficient
Picky eating	Change in BMI or standardised weight	N/A	Insufficient
Picky eating	Odds of underweight	N/A	Insufficient
Inability to delay gratification	Risk of overweight	N/A	Insufficient
Inability to delay gratification	Change in BMI z-score	N/A	Insufficient
Skipping breakfast versus eating breakfast	Odds of overweight	N/A	Insufficient

Abbreviations: BMI, body mass index; N/A, not applicable.

[1] N/A: not enough evidence to draw conclusions and recommendations.

6.30 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.

6.31 There was **insufficient** evidence to enable conclusions to be drawn on any association between the eating behaviours examined (picky eating, ability to delay gratification, skipping versus eating breakfast) in children aged 1 to 5 years and body composition or weight status in later childhood as fewer than 3 primary studies included in the SRs examined these relationships.

Evidence identified on caregiver feeding practices and styles on children's food acceptance, dietary intake and body composition or weight status

6.32 Two SRs with MAs (Hodder et al, 2018; 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.

6.33 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 7 (Table A7.7). Additional data extracted on the primary studies can be found in Annex 8 (Tables A8.32 to A8.35). The criteria used to grade the evidence are provided in Chapter 2 (Table 2.4, paragraphs 2.43 to 2.47). Summary tables of the evidence grading process for this section are provided in Annex 9 (Tables A9.16 to A9.20 and Table A9.29).

6.34 Most of the primary studies included in the identified 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); and use of rewards (food and non-food) to reinforce or encourage eating of the target food(s).

6.35 Studies took place in a mix of settings including the child's home, childcare centres or preschools, and laboratory settings. Study designs varied considerably (RCTs, non-randomised controlled trials, quasi-experimental designs, and pre-post designs).

6.36 Some 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.

6.37 Most of the studies included in the SRs or MAs in children aged 1 to 5 years were conducted in high-income countries (HIC) including the UK.

Limitations of the evidence on feeding practices and styles

6.38 All the primary studies included in the SRs or MAs examined the effect of feeding practices on children's eating behaviours and body weight in the shorter 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 effects of such practices.

6.39 Most primary studies had small sample sizes. Whether they were adequately powered was either not considered or reported in the SR.

6.40 Risk of publication bias was evident in 2 SRs with MAs (Hodder et al, 2018; 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 the SRs/Mas (Higgins et al, 2022).

6.41 SRs did not always report or consider whether non-randomised intervention studies and PCS adjusted for potential confounding factors, such as child weight status and eating behaviours at baseline. PCS that report associations between parental restrictive practices around food and higher risk of child overweight 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.

6.42 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 align with observational measures of parental feeding (Bergmeier et al, 2015; Hurley et al, 2011).

6.43 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).

6.44 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 Chapter 5), only 1 SR (Hodder et al, 2018) specifically examined interventions to increase vegetable and fruit acceptance or consumption in these children.

6.45 No evidence from SRs 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 (collectively) on increasing children's acceptance or consumption of fruit or vegetables

6.46 Two SRs with MAs (Hodder et al, 2018; Nekitsing et al, 2018) examined the effectiveness of feeding practices (collectively) 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.

6.47 Hodder et al (2018) (AMSTAR 2 confidence rating: high) included 30 RCTs in children aged 1 to 5 years. Findings from 13 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 0.33; 95% CI 0.13 to 0.54; p=0.0014; I^2=70%; random-effects model; 13 RCTs, 1741 participants). The SR stated that this effect size was equivalent to an increase of 3.50g as-desired consumption of vegetables. Intervention duration was up to 6 months.

6.48 Results of a sensitivity analysis that removed 8 studies at high risk of bias resulted in a smaller effect size (SMD 0.23; 95% CI 0.03 to 0.44; p=0.026; I^2=14%; random-effects model; 5 trials, 487 participants) as did a sensitivity analysis that included 8 studies with low attrition or high attrition with intention-to-treat analysis (SMD 0.29; 95% CI 0.10 to 0.48; p=0.0022; I^2=27%; random-effects model; 8 trials, 757 participants).

6.49 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). 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).

6.50 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).

6.51 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

6.52 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.

6.53 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

6.54 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).

6.55 For this draft report, 1 SR with MA (Hodder et al, 2018; 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.

6.56 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^2=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^2 NR; 5 intervention arms, 134 participants).

6.57 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.

6.58 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.

6.59 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 6.3 and 6.4) or whether taste exposure is equally effective at increasing acceptance or consumption of vegetables across the 1 to 5 year age group.

6.60 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 (statistics 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)

6.61 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) (statistics not reported).

Repeated taste exposure and pairing on children's vegetable consumption

6.62 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 shorter term (less than 12 months) in children aged 5 years and under.

6.63 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^2 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 6.56). The comparison between the 2 intervention strategies should be interpreted with caution because Nekitsing et al (2018) did not report performing a formal statistical comparison between the two.

6.64 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 (statistics not reported).

Repeated visual exposure on children's acceptance or taste preference for fruit or vegetables

6.65 Mura Paroche et al (2017) (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.

6.66 One of the studies (in 20 participants, described in paragraph 6.65), 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)

6.67 The evidence 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 6.2.

6.68 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 6.2. Summary of the evidence of the effect of feeding practices on increasing children's vegetable or fruit consumption (short term, up to 8 months)

Intervention[1]	Outcome	Direction of effect[2]	Certainty of evidence
Feeding practices (collectively)[3]	Vegetable consumption	↑	Moderate
Feeding practices[4] and social or economic deprivation	Vegetable or fruit consumption	N/A	Insufficient
Repeated taste exposure	Vegetable consumption	↑	Moderate
Repeated taste exposure	Acceptance of textures (vegetable or fruit)	N/A	Insufficient
Repeated taste exposure plus pairing[5]	Vegetable consumption	↑	Moderate
Repeated visual exposure	Preference/acceptance (fruit)	N/A	Insufficient
	Preference/acceptance (vegetable)	N/A	Insufficient

Abbreviations: N/A, not applicable.

[1] Compared with a control group (no intervention, usual care, or treatment received after the intervention phase).

[2] Direction of effect for reported outcomes: ↑increase; ↓decrease; N/A: not enough evidence to draw conclusions and recommendations.

[3] 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).

[4] Includes repeated taste exposure, non-food rewards, vegetable and fruit provision, child and parent nutrition education.

[5] Repeated taste exposure to vegetables that were paired with liked foods, or additional flavours or dietary dietary energy.

Summary: feeding practices (collectively)

6.69 Evidence from the MA conducted by Hodder et al (2018) suggested that feeding practices (repeated exposure, pairing vegetables with positive stimuli, and general infant and young child feeding practices) can increase children's vegetable consumption in the short term (up to 6 months) by approximately 3.50g of as-desired vegetable consumption. These findings were supported by 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 (2018), 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 together with a small effect size and non-specificity of interventions prevented the evidence from being graded *adequate*.

6.70 There was **insufficient** evidence 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.

6.71 No evidence from SRs was identified on the longer term impact of feeding practices on children's vegetable or fruit consumption.

Summary: repeated exposure

6.72 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 6.3 and 6.4) 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*.

6.73 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.

6.74 There was **insufficient** evidence 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

6.75 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

6.76 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 ab 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

6.77 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 8, Table A8.34 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; $p=0.35$).

6.78 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.

6.79 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 6.3 and 6.4) 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.

6.80 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

6.81 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.

6.82 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 (statistics not reported). However, the effect disappeared 1 month after the study completed.

6.83 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 (statistics not reported).

Use of rewards (food or non-food)

6.84 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

6.85 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

6.86 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

6.87 The evidence 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 6.3.

Table 6.3. 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[1]	Certainty of evidence
Restriction	Dietary energy intake	N/A	Insufficient
Adult modelling	Food acceptance or consumption	Inconsistent	Inconsistent
Peer modelling	Food acceptance or preference or consumption	N/A	Insufficient
Use of rewards	Food acceptance or consumption	N/A	Insufficient
Verbal encouragement	Food acceptance or consumption	N/A	Insufficient
Choice offering	Food acceptance or consumption	N/A	Insufficient

Abbreviations: N/A, not applicable.

[1] N/A: not enough evidence to draw conclusions and recommendations.

6.88 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.

6.89 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 6.3 and 6.4).

6.90 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.

6.91 There was **insufficient** evidence 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

6.92 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).

6.93 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.

6.94 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.

6.95 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.

6.96 In addition, Appleton et al (2018) identified 2 PCS that examined whether exposure to sugars-sweetened beverages (SSBs) and fruit juice was associated with later consumption of these beverages. Quantitative data were not reported for either study.

6.97 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.

6.98 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

6.99 The evidence 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 6.4.

Table 6.4. 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[1]	Certainty of evidence
Exposure to sweet food	Preference for or consumption of sweet foods	N/A	Insufficient
Exposure to SSBs or fruit juice	Consumption of SSBs or fruit juice	N/A	Insufficient

Abbreviations: SSBs, sugars-sweetened beverages; N/A, not applicable.
[1] N/A: not enough evidence to draw conclusions and recommendations.

6.100 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.

6.101 There was **insufficient** evidence 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

6.102 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 6.41).

6.103 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 (statistics NR). The analysis was adjusted for sex, ethnicity, baseline weight-for-height z-score and food consumption (servings per day).

6.104 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 (statistics NR).

Pressuring a child to eat

6.105 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.

6.106 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 (statistics 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).

6.107 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 (statistics NR).

Caregiver feeding styles on children's body composition

6.108 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.

6.109 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 (statistics 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.

6.110 No evidence from SRs was identified on the effect of responsive feeding styles see paragraphs 6.9 and 6.10).

Summary: Caregiver feeding practices and styles on children's body composition

6.111 The evidence on the effect of caregiver feeding practices and styles on children's body composition (short term, less than 12 months) is summarised in Table 6.5 and Table 6.6.

Table 6.5. Summary of the evidence on caregiver feeding practices on children's body composition

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Restrictive feeding practices	Change in BMI z-score	N/A	Insufficient
Pressuring a child to eat	WLZ and BMI z-score	N/A	Insufficient

Abbreviations: BMI, body mass index; N/A, not applicable; WLZ, weight-for-length z-score.
[1] N/A: not enough evidence to draw conclusions and recommendations.

Table 6.6. Summary of the evidence on caregiver feeding styles on children's body composition

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Non-responsive feeding (intrusiveness)	WLZ and BMI z-score	N/A	Insufficient
Responsive feeding	Body composition measures	N/A	No evidence identified

Abbreviations: BMI, body mass index; N/A, not applicable; WLZ, weight-for-length z-score.
[1] N/A: not enough evidence to draw conclusions and recommendations.

6.112 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.

6.113 There was **insufficient** evidence 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.

7 Excess weight and obesity

Background

7.1 Overweight and obesity are defined by the World Health Organization (WHO) as "abnormal or excessive fat accumulation that presents a risk to health' (WHO, 2020).

7.2 The most widely used indicator of overweight and obesity is body mass index (BMI), a measure of body weight adjusted for height. 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. BMI also provides no information about body shape, pattern of fat distribution or fat-to-lean mass ratio (SACN, 2011b). 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).

7.3 Nevertheless, BMI is still accepted as the most appropriate measure of weight status in children above age 2 years (and in adolescents).

7.4 Overweight and obesity in children are classified by comparing their BMI with a reference population that describes the distribution of BMI by both age and sex (SACN and RCPCH, 2012).

7.5 In the UK, young child growth is monitored using the UK-WHO growth charts (RCPCH, 2013) which use the WHO Child Growth Standards for children aged 2 weeks to 4 years (WHO Multicentre Growth Reference Study Group 2006) and the UK 1990 reference values for birth and for children (and adolescents) for those aged over 4 years (Freeman et al, 1995; Wright et al, 2010).

7.6 Several BMI thresholds or cut-offs are used to define overweight and obesity for children aged over 2 years. The UK-WHO growth charts use BMI above the 91st percentile (+1.33 SD) to indicate overweight and a BMI above the 98th percentile (+2 SD) to indicate very overweight (clinically obese). For public health surveillance and analysis in the UK (for example, the National Child Measurement Programme [NCMP] in England), the less stringent definitions of the 85th percentile (+1 SD) to indicate overweight and the 95th percentile (+1.65 SD) to indicate obesity are used in order to maximise the statistical power of analyses of geographical and secular differences. The NCMP also classifies children at or above the 99.6th percentile as severely obese. It is important to note that the 85th and 95th centiles used in the NCMP 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, 2016c).

7.7 The WHO recommends using the less stringent (public health) definition for overweight (+1 SD) in school-aged children while recommending the more stringent definition for obesity (+2 SD) as used in the UK-WHO growth charts. However, for children younger than age 2 years, the WHO recommends even more stringent definitions for overweight (+2 SD) and obesity (+3 SD) on the premise that obesity is intrinsically less prevalent in this age group. Research analysing body composition data suggests that high BMI index percentiles may over-diagnose obesity in children under age 6 years (Wright et al, 2021).

Early life determinants of obesity

7.8 Early life determinants of overweight or obesity in later childhood and adulthood can be divided into those that are modifiable and those that are not.

7.9 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) and the characteristics of the child's household and wider community (such as household socioeconomic status, food insecurity, access to healthy 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 a child's later risk of overweight or obesity or merely predictive.

7.10 A good example of a non-modifiable determinant of overweight or obesity is genetics. Genome-wide association studies indicate that 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 the body composition of children aged under 5 years (Alves et al, 2019; Elks et al, 2014). Even though an individual's genetic susceptibility to becoming overweight or obese is not modifiable, environmental factors can alter the effect of these genetic factors. For example, findings from the Gemini birth cohort twin study in England and Wales indicate that the heritability of BMI in children aged under 5 years may be higher among those living in more obesogenic environments compared with those living in 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.

7.11 For children with overweight or obesity, evidence suggests that a range of diet, exercise and behavioural therapy interventions may help reduce BMI or body

weight in children and adolescents (Salam et al, 2020) and that interventions which are home based and that include parents or families may be the most effective in preventing childhood obesity (Flynn et al, 2022).

Weight status of young children in the UK

7.12 Globally, there has been an increasing prevalence of overweight and obesity in children. In the UK, a number of national public health surveys regularly measure and monitor the prevalence of overweight or obesity in children.

7.13 The Diet and Nutrition Survey of Infants and Young Children (DNSIYC), which surveyed children from all 4 countries in the UK in 2011, showed that 29% of boys and 27% of girls aged 12 to 18 months exceeded the 91st percentile (defined as overweight) while 10% of boys and 9% of girls aged 12 to 18 months exceeded the 98th percentile (defined as very overweight or clinically obese) of the UK-WHO growth charts for body weight (Lennox et al, 2013). No such data exist in children aged 18 to 60 months from the National Diet and Nutrition Survey (NDNS).

7.14 In Scotland, the Scottish Health Survey (SHeS) measures the BMI of children aged 2 to 6 years (and ages 7 to 11 years and ages 12 to 16 years) to assess the prevalence of overweight and obesity (by comparing against the UK 1990 reference curves (Freeman et al, 1995). Since 1998, the combined prevalence of overweight and obesity (using public health definitions, see paragraph 7.6) in children aged 2 to 6 years has ranged from 26% to 33%. The 2019 SHeS (the latest available survey) showed that the combined prevalence of overweight and obesity in this age group was 30%, while 7% of children in this age group were at risk of obesity (defined as at or above the 95th percentile, not including severe obesity) (Scottish Government, 2020).

7.15 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 overweight and obesity levels in children attending primary school. Like the SHeS, BMI values (derived from height and weight data) are compared against the UK 1990 reference population data to calculate age- and sex-adjusted percentiles. The latest available NCMP data on weight status are presented in Figure 7.1 to Figure 7.7 and in Annex 12 (Tables A12.1 to A12.4).

7.16 For the NCMP collection years 2006/07 to 2019/20 the combined prevalence of overweight and obesity (using public health definitions, see paragraph 7.6) for children aged 4 to 5 years was fairly stable, at 22% to 23% (see Figure 7.1) (NHS Digital, 2021). For the same years, the prevalence of obesity (including severe obesity) ranged from 9.1% (2014/15) to 9.9% (2006/07 and 2019/20) and the prevalence of severe obesity ranged from 2.1% (2012/13 and 2014/15) to 2.5% (2019/20).

7.17 However, the combined prevalence of overweight and obesity increased substantially over the first year of the COVID-19 pandemic (NCMP collection years 2019/20 to 2020/21), from 23.0% to 27.7%. The prevalence of obesity (including severe obesity) increased from 9.9% to 14.4% and severe obesity increased from 2.5% to 4.7% (Figure 7.1 and Annex 12, Table A12.1). While the prevalence of obesity and severe obesity was similar in boys and girls in year 2020/21 (Figure 7.2), the prevalence of obesity and severe obesity increased more in boys than in girls during the first year of the COVID-19 pandemic (years 2019/20 to 2020/21) (Figure 7.3).

7.18 The overall increase in prevalence of overweight and obesity suggests that total dietary energy intake (TDEI) in school-aged children substantially exceeded energy requirements. This may be due in part to the reported decrease in physical activity levels leading up to and during the first national lockdown in the UK due to the COVID-19 pandemic (Sport England, 2021a; Sport England, 2021b; Sport England, 2021c). Data are currently insufficient to determine whether TDEI also increased in young children during the national lockdown.

Figure 7.1 Prevalence of overweight, obese and severely obese children aged 4 to 5 years in England for NCMP collection years 2006/07 to 2020/21 (NHS Digital, 2021)

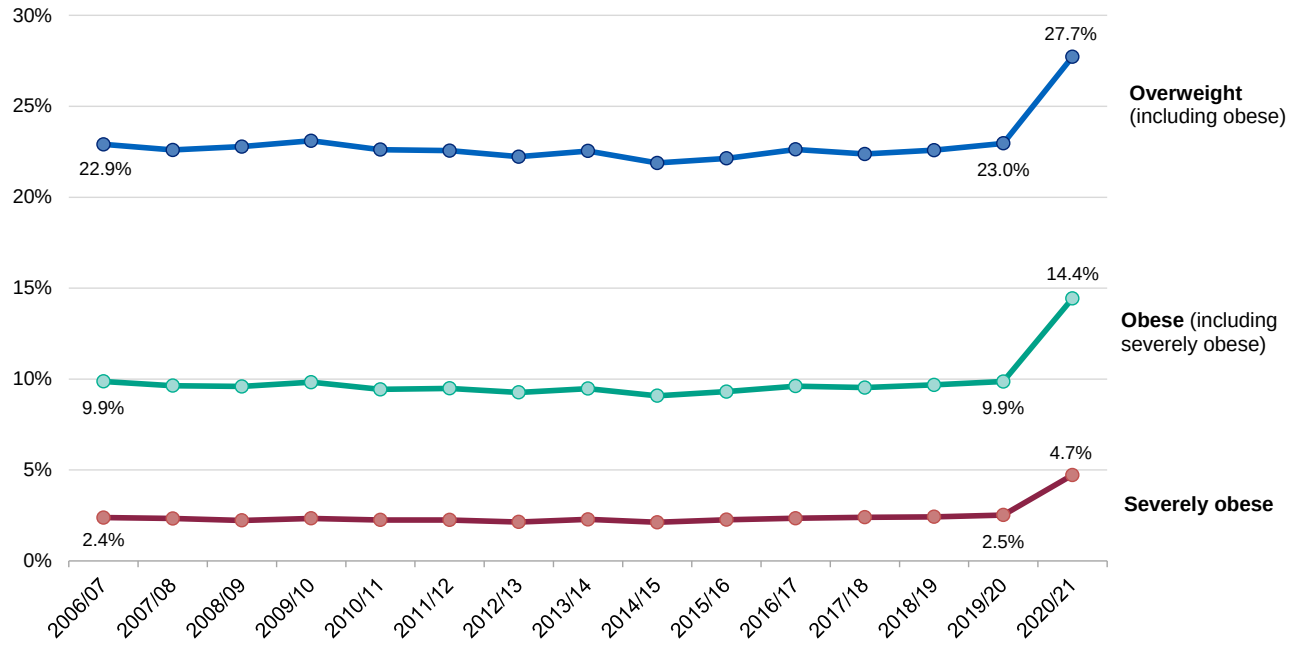


Figure 7.2 Weight status prevalence of boys and girls aged 4 to 5 years in England for NCMP collection year 2020/21 (NHS Digital, 2021)

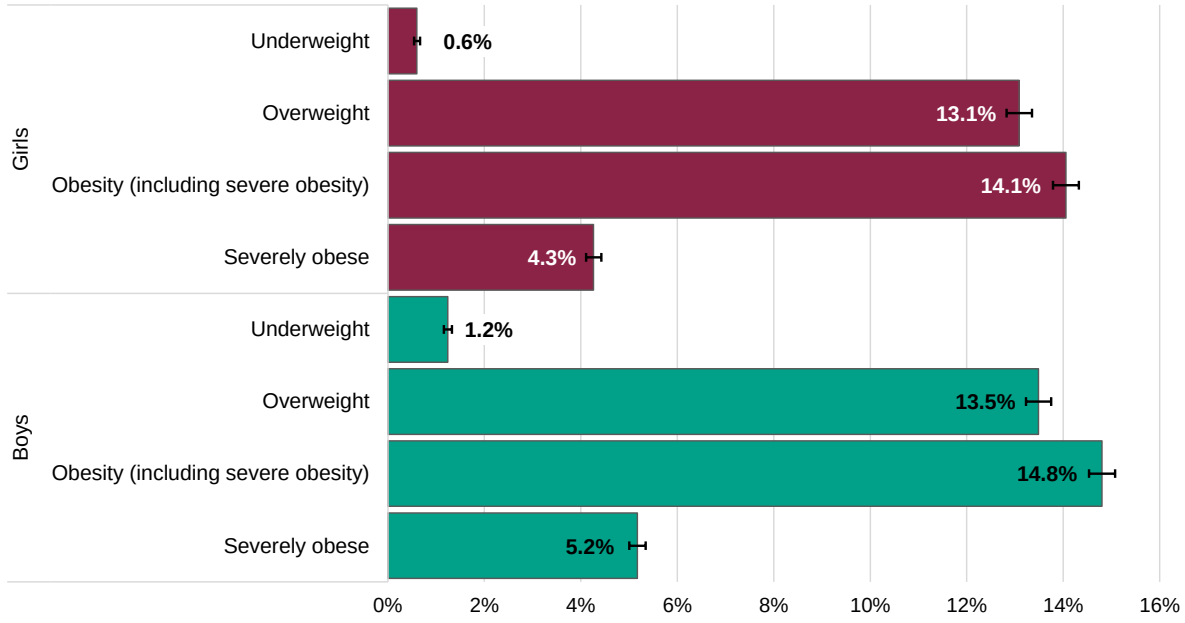


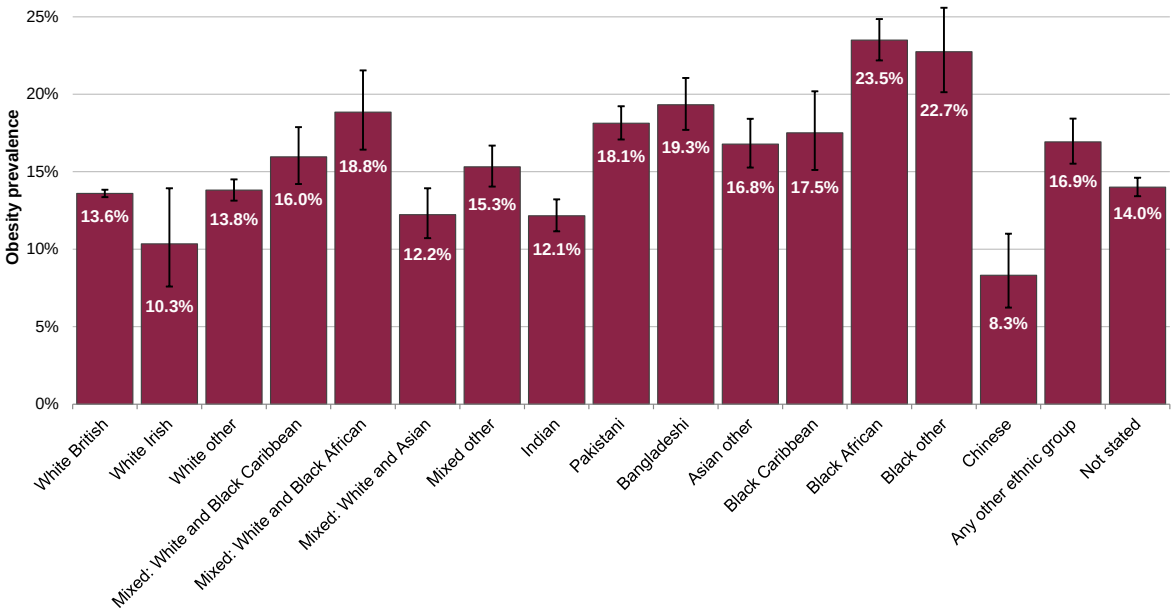
Figure 7.3 Change in prevalence of obesity and severe obesity among children aged 4 to 5 years in England for NCMP collection years 2019/20 and 2020/21 (OHID, 2022)

Weight status by ethnic minority group

7.19 Obesity prevalence (including severe obesity) for children aged 4 to 5 years from different ethnic minority groups is shown in Figure 7.4 (OHID, 2021). The prevalence of obesity was highest for children categorised as Black African (23.5%), Black other (22.7%), Bangladeshi (19.3%), Mixed – White and Black (18.8%), Pakistani (18.1%) and Black Caribbean (17.5%). Obesity prevalence was lowest for children categorised as Chinese (8.3%), White Irish (10.3%), Indian (12.1%), Mixed – White and Asian (12.2%) and White British (13.6%).

7.20 The prevalence of severe obesity was highest for children categorised as Black other (9.2%), Black African (8.3), Pakistani and Mixed – White and Black African (both 7.7%). Prevalence was lowest for children categorised as Irish (2.5%), Chinese (3.2%), Mixed – White and Asian (3.9%) and White British (4.1%). The prevalence of underweight was highest for children categorised as Indian (4.5%), Pakistani (3.1%), Bangladeshi (2.5%), Asian other (2.3%) and Mixed – White and Asian children (1.8%). More detailed data on prevalence by weight status (from underweight to severe obesity) by ethnic minority group is available in Annex 12, Table 12.2.

Figure 7.4 Prevalence of obesity (including severely obese) in children aged 4 and 5 years by ethnic minority group (OHID, 2021)



Weight status and deprivation

7.21 Weight status by index of multiple deprivation (IMD) (see Annex 13, Glossary) for children aged 4 to 5 years is shown in Figure 7.5 to Figure 7.7, and in Annex 12, Tables 12.3 and 12.4.

7.22 Obesity prevalence for the 10% of children living in the least or most deprived areas for NCMP collection years 2006/07 to 2020/21 is shown in Figure 7.5. For all years, obesity prevalence is substantially higher for children living in the most deprived areas. The gap in obesity prevalence between children living in the most and least deprived areas has increased from 5.3% in 2006/7 to 7.3% in 2019/20, and this gap increased further during the first year of the COVID-19 pandemic to 12.4% (2020/21) (OHID, 2021).

7.23 The data for 2020/21 showed a strong relationship between the prevalence of obesity and deprivation. Obesity prevalence (including severe obesity) increased with each IMD decile, from the least deprived (7.8%, in decile 10) to the most deprived decile (20.3%, in decile 1) (see Figure 7.6). For severe obesity (see Figure 7.7), prevalence was 4 times higher in children living in the most deprived areas (7.6%) than those in the least deprived areas (1.9%) (OHID, 2021).

Figure 7.5 Gap in the prevalence of obesity for children aged 4 to 5 years in England between children living in the least and most deprived Index of Multiple Deprivation (IMD) decile (based on the postcode of the child) (NCMP collection year 2020/21) (OHID, 2021)

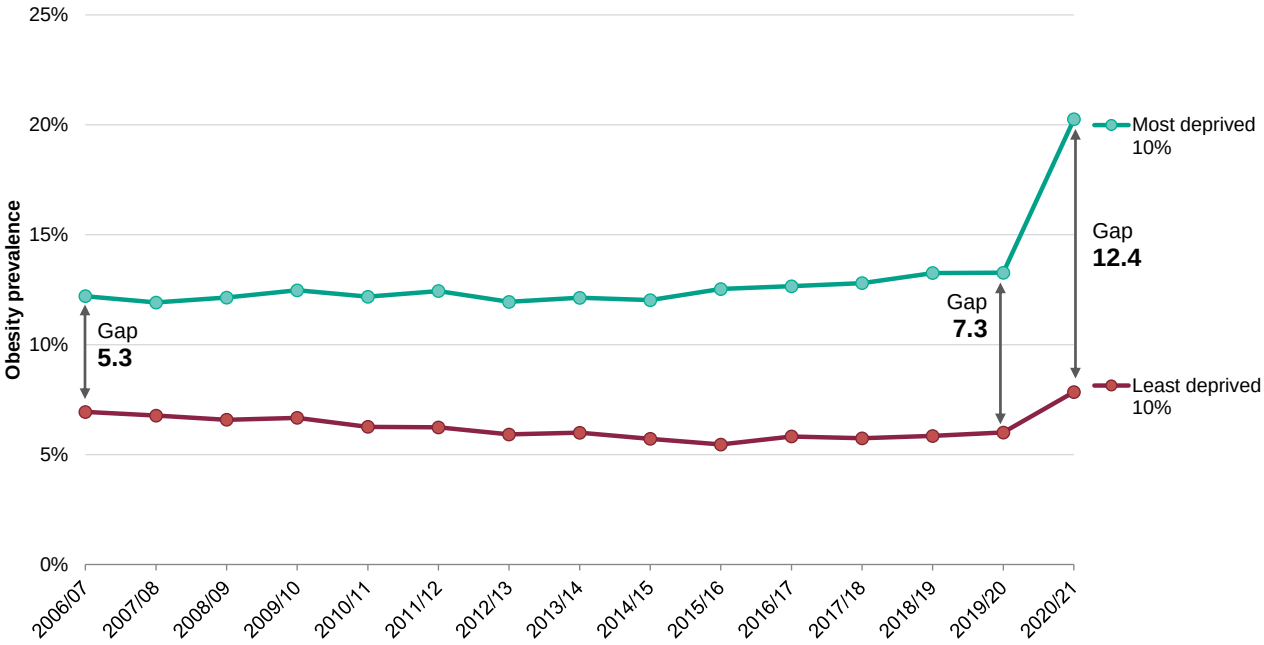


Figure 7.6 Prevalence of obesity (including severe obesity) for children aged 4 to 5 years in England by Index of Multiple Deprivation (IMD) decile (based on the postcode of the child) (NCMP collection year 2020/21) (OHID, 2021)

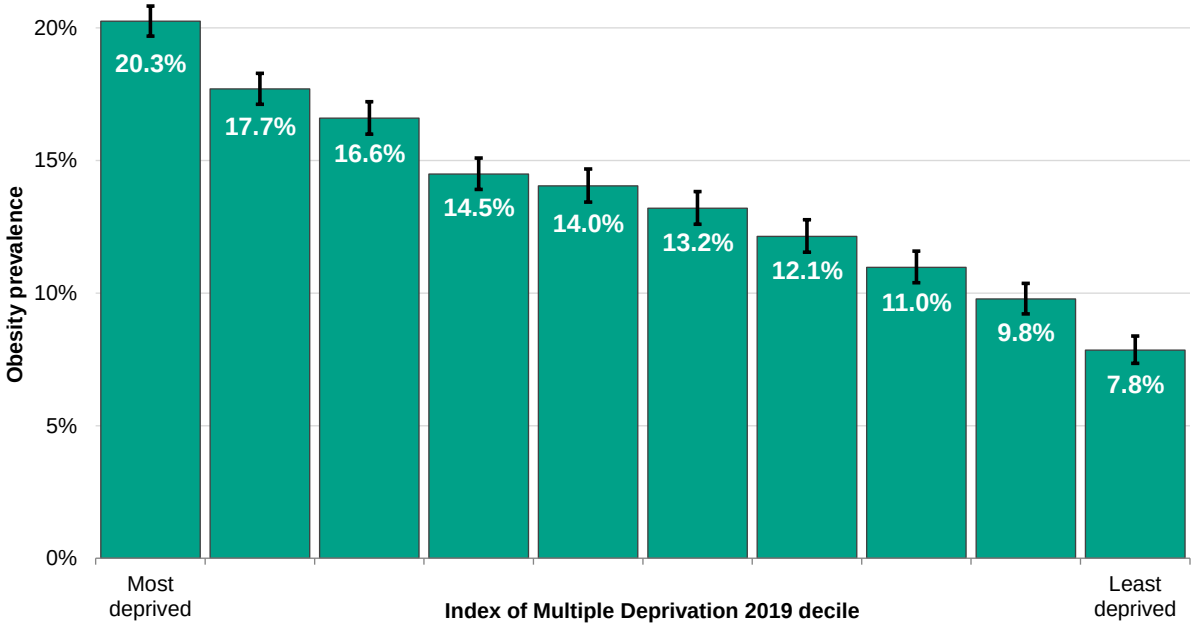
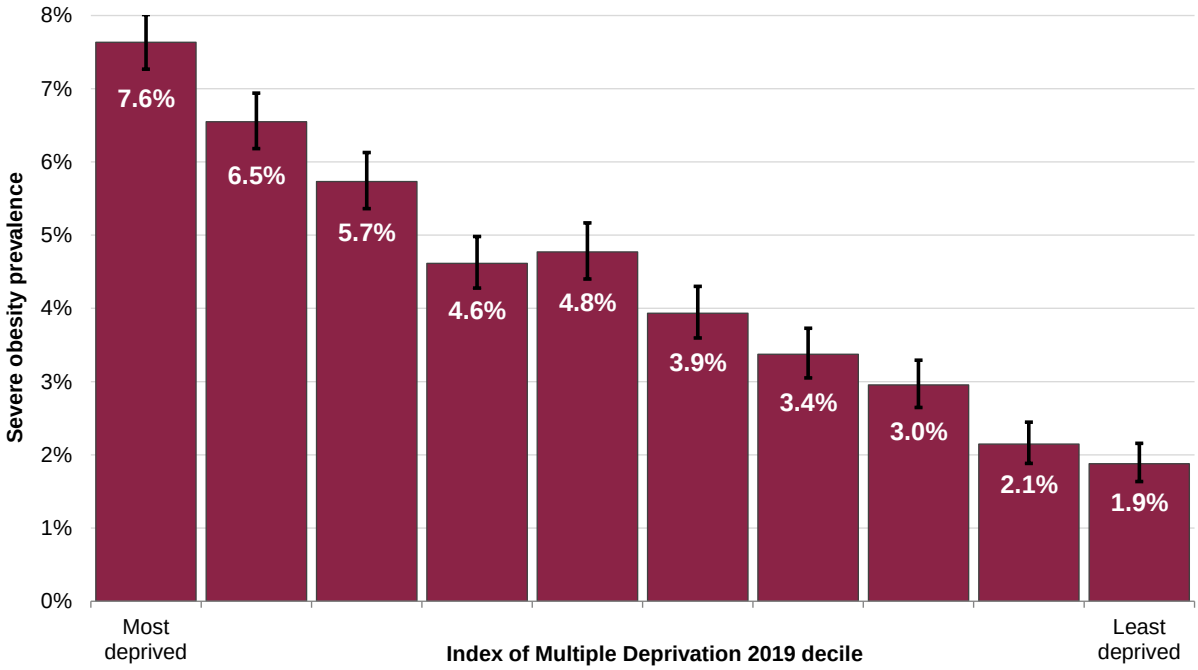


Figure 7.7 Prevalence of severe obesity for children aged 4 to 5 years in England by Index of Multiple Deprivation (IMD) decile (based on the postcode of the child) (NCMP collection year 2020/21) (OHID, 2021)



Limitations of the evidence on excess weight and obesity

7.24 For this draft report, much of the evidence on excess weight and obesity identified from systematic reviews (SRs) 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.

7.25 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. There is also no single accepted threshold for defining infant and child overweight and obesity (see paragraphs 7.6 to 7.7) and widely differing cutoffs were used in different studies.

7.26 The reporting of body weight or BMI varied between primary studies, with some reporting outcomes on a continuous scale, while others reporting on the proportion of children with overweight (variously defined) either combined with or separated from the proportion of children with obesity (variously defined).

7.27 Although there are substantial disparities in child overweight and obesity based on differences in socioeconomic status (SES) and ethnic minority backgrounds, much of the SR evidence identified was derived from populations of mostly affluent, white children.

7.28 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.

7.29 Prospective cohort studies (PCS) that relate child BMI to adult BMI or weight status may be able to describe the natural development or history of becoming overweight and obesity but cannot usually provide mechanistic insights to allow causal inferences to be made. This is due to the great number of potential confounding factors that are often not measured or adjusted for (see paragraphs 7.27 and 7.28).

7.30 While there was potential for publication bias in this area of research, it was not assessed by the SRs identified for this draft report.

Evidence identified on child growth trajectory and adult BMI or weight status

7.31 '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.

7.32 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.

7.33 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 A5.5). Quality assessment of the SRs using the AMSTAR 2 tool can be found in Annex 7 (Table A7.8). Additional data extracted on the primary studies can be found in Annex 8 (Table A8.36). The criteria used to grade the evidence are provided in Chapter 2 (Table 2.4, paragraphs 2.43 to 2.47). Summary tables of the evidence grading process for this section are provided in Annex 9 (Tables A9.21 to A9.23, and Table A9.29).

7.34 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

7.35 'Rapid early growth' describes the increase in body size, usually measured by BMI, beyond what would be expected at a particular stage of growth. In this draft report, rapid growth occurring beyond the age of 1 year was considered.

7.36 One SR without MA (Brisbois et al, 2012) examined the association between rapid early growth and adult BMI.

7.37 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).

7.38 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).

7.39 It should be emphasised that while these findings may highlight the natural history of becoming overweight and obesity in adulthood, it does not provide mechanistic insights to allow causal inferences to be made.

Summary: rapid early growth and adult BMI

7.40 The evidence on rapid early growth and adult BMI is summarised in Table 7.1.

Table 7.1. Summary of the evidence on rapid early growth and adult BMI

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Rapid early growth	Adult BMI	N/A	Insufficient

Abbreviations: BMI, body mass index; N/A, not applicable.
[1] N/A: not enough evidence to draw conclusions and recommendations.

7.41 The available evidence 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.

7.42 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 was graded **insufficient** given the small number of primary studies identified.

Age at adiposity rebound (AR) and adult BMI or risk of obesity

7.43 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 thus 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.

7.44 For this draft 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).

7.45 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

7.46 The evidence on age at AR and obesity is summarised in Table 7.2.

Table 7.2. Summary of the evidence on age at AR and BMI or risk of obesity

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Age at AR	Adult BMI or risk of obesity	↓	Limited

Abbreviations: AR, adiposity rebound; BMI, body mass index.
[1] Direction of association for reported outcomes: ↑increase; ↓inverse.

7.47 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.

7.48 Evidence from 4 PCS included in the SR by Brisbois et al (2012) suggests that AR occurring before the age of 5 years is associated with higher adult BMI or risk of adult obesity. The evidence was graded **limited** given the small number of studies identified and the lack of adjustment for key confounding factors (baseline BMI).

Child BMI or weight status and adult BMI or weight status

7.49 One SR without MA examined the relationship between child BMI or weight status and adult BMI or weight status (Brisbois et al, 2012) and included 11 PCS (in a

total of 4296 participants) that included children aged 1 to 5 years at baseline. Ten PCS (in a total of 3590 participants) reported that a higher BMI (or a BMI above the 75th or 85th percentile) at ages 1 to 5 years was associated with higher adult BMI, while 1 PCS reported no association (statistics NR). Of the 10 PCS that reported an association, 4 reported that a higher BMI (or a BMI above the 75th or 85th 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).

7.50 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, quantitative findings from 5 were reported in the SR. Of these, 1 PCS adjusted for parental weight status and the other 3 were unadjusted.

7.51 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

7.52 The evidence on child BMI or weight status and adult BMI or weight status is summarised in Table 7.3.

Table 7.3. Summary of the evidence on the relationship between 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[1]	Certainty of evidence
BMI or weight status of children aged 1 to 5 years	Adult BMI or risk of adult overweight or obesity	↑	Adequate

Abbreviations: BMI, body mass index.
[1] Direction of association for reported outcomes: ↑increase; ↓inverse.

7.53 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.

7.54 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.

Evidence identified on child BMI and other health outcomes in adulthood

7.55 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.

7.56 Details of the SR can be found in Annex 5 (Table A5.5). Quality assessment of the SRs using the AMSTAR 2 tool can be found in Annex 7 (Table A7.8). Additional data extracted on the primary studies can be found in Annex 8 (Table A8.37). The criteria used to grade the evidence are provided in Chapter 2 (Table 2.4, paragraphs 2.43 to 2.47). Summary tables of the evidence grading process for this section are provided in Annex 9 (Tables A9.24 and A9.25, and Table A9.29).

7.57 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 as the focus of the SR was to examine the association between childhood obesity and morbidities without knowledge of later adult obesity.

7.58 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.

7.59 Limitations of this SR 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.

Child BMI and adult type 2 diabetes (T2D)

7.60 Llewellyn et al (2016) (AMSTAR 2 confidence rating: critically low) included 1 PCS that examined the relationship between BMI in children aged 1 to 5 years and incidence of T2D in adulthood. The PCS (number of participants NR) reported that BMI at age 6 years and under was associated with T2D in adulthood (OR per SD of BMI 1.23; 95% CI 1.10 to 1.37).

Summary: child BMI and adult T2D

7.61 The evidence on childhood BMI and T2D is summarised in Table 7.4.

Table 7.4. Summary of the evidence on the relationship between child BMI and adult T2D

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Child BMI[2]	Adult T2D	N/A	Insufficient

Abbreviations: BMI, body mass index; N/A, not applicable; T2D, type 2 diabetes.
[1] N/A: not enough evidence to draw conclusions and recommendations.
[2] Children were aged 6 years and under.

7.62 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 included only 1 PCS that examined this relationship in children aged 1 to 5 years, the evidence was graded **insufficient**.

Child BMI and adult coronary heart disease (CHD)

7.63 Llewellyn et al (2016) included a subgroup MA that reported no association between BMI in children aged 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 in older age groups that were examined (age 7 to 11 years and 12 to 18 years) and incidence of CHD.

Summary: child BMI and adult CHD

7.64 The evidence on child BMI and adult CHD is summarised in Table 7.5.

Table 7.5. Summary of the evidence on the relationship between child BMI and adult CHD

Exposure	Outcome	Direction of association	Certainty of evidence
Child BMI[1]	Adult CHD	Null	Moderate

Abbreviations: body mass index (BMI); coronary heart disease (CHD).
[1] Children were aged 6 years and under.

7.65 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.

7.66 Evidence from the subgroup MA conducted by Llewellyn et al (2016) reported no association between BMI at age 6 years and under and 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

7.67 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

7.68 The evidence on child BMI and adult stroke is summarised in Table 7.6.

Table 7.6. Summary of the evidence on the relationship between child BMI and adult stroke

Exposure	Outcome	Direction of association	Certainty of evidence
Child BMI[1]	Adult stroke	Null	Moderate

Abbreviations: BMI, body mass index.
[1] Children were aged 6 years and under.

7.69 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.

7.70 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

7.71 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 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

7.72 The evidence on child BMI and adult breast cancer is summarised in Table 7.7.

Table 7.7. Summary of the evidence on the relationship between child BMI and adult breast cancer

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Child BMI[2]	Adult breast cancer	N/A	Insufficient

Abbreviations: BMI, body mass index; N/A, not applicable.

[1] N/A: not enough evidence to draw conclusions and recommendations.

[2] Children were aged 6 years and under.

7.73 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. The MA by Llewellyn et al (2016) included only 1 PCS that examined this relationship. The evidence was graded **insufficient**.

Summary of the evidence relating to excess weight and obesity

7.74 This section draws together the evidence relating to excess weight and obesity from throughout this draft report, including the current chapter.

7.75 Overall, there was a paucity of evidence identified from SRs on the majority of dietary exposures and their relationship with excess weight and obesity, in children aged 1 to 5 years. Much of the evidence identified was graded **insufficient** (see Annex 9, Table A9.29).

7.76 Table 7.8 lists the exposure-outcome relationships for which some evidence was identified (graded **adequate**, **moderate**, or **limited**).

7.77 The strongest evidence identified relates to the health impact of consuming sugars-sweetened beverages (SSBs). There is **adequate** evidence that higher consumption of SSBs in children aged 1 to 5 years is associated with an increased odds of overweight or obesity in later 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 later childhood. 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).

7.78 There is **moderate** evidence that higher total protein intake in children aged 1 to 5 years is associated with increased BMI in later 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).

7.79 There is **adequate** evidence 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. Together with findings from the latest NCMP of the recently increasing prevalence of overweight and obesity in children entering primary school in England, this is a cause for concern.

7.80 This draft report also identified:

- **moderate** evidence that there is no association between total cows' milk intake and BMI in children aged 1 to 5 years
- **moderate** evidence that increasing portion sizes (in grams or energy intake) of snack or lunch foods in preschool settings increases children's food and energy intake in the short term (interventions lasting for up to 3 months). However, no evidence was identified on whether portion sizes directly impact children's body weight

- **moderate** evidence that there is no association between BMI at age 6 years and under and adult coronary heart disease
- **moderate** evidence that there is no association between BMI at age 6 years and under and adult stroke.

Table 7.8. Summary of the evidence identified with obesity related outcomes

Exposure (in children aged 1 to 5 years)	Outcome	Direction of effect or association[1]	Certainty of evidence	Evidence location (page)
Dietary energy				
Portion size	Food and energy intake (short term, up to 3 months)	↑	Moderate	48 to 51
Carbohydrates				
SSB consumption	Odds of overweight	↑	Adequate	68 to 71
	Change in weight status[2]	↑	Moderate	
Dietary fat				
Total fat intake	BMI or body weight (shorter term)	Null	Limited	85 to 87
Protein				
Total protein intake	BMI	↑	Moderate	191 to 193
Foods, dietary components, and dietary patterns				
Fruit juice consumption	Change in BMI	↑ (non-TDEI adjusted) Null (TDEI-adjusted)	Limited	168 to 171
Total cows' milk consumption	BMI	Null	Moderate	197 to 199
'Unhealthy' dietary patterns[3]	Body fat	↑	Limited	217 to 218
Excess weight and obesity				
Rapid early weight gain or growth	Adult BMI	↑	Limited	266 to 267
Age at AR	Adult BMI	↓	Limited	267 to 268
Child BMI or weight status	Adult BMI or risk of overweight or obesity	↑	Adequate	268 to 269
Child BMI[4]	Adult CHD	Null	Moderate	271 to 272
Child BMI[4]	Adult stroke	Null	Moderate	272

Abbreviations: AR, adiposity rebound; BMI, body mass index; CHD, coronary heart disease; SSB, sugars-sweetened beverage

[1] Direction of effect or association for reported outcomes: ↑increase; ↓inverse.

[2] BMI, BMI z-score, weight-for-height z-score.

[3] Defined in Chapter 5, paragraph 5.137.

[4] Children aged under 6 years.

8 Oral Health

Background

Oral health of children in the UK

8.1 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).

8.2 Dental caries is a biofilm-mediated multifactorial and dynamic disease driven by dietary sugars that results in the phased demineralisation and remineralisation of dental hard tissues (Pitts et al, 2017). 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). This acid causes a drop in pH levels which makes the tooth susceptible to demineralisation. 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 process (ten Cate, 2013).

8.3 Tooth decay in early childhood is known as early childhood caries (ECC) and is defined as “the presence of one or more decayed (noncavitated 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).

8.4 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 are 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

8.5 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 2015/6 (Cardiff University, 2017), 23.4% in England in 2019 (PHE, 2020) and 26.5% in Scotland in 2020 (Public Health Scotland, 2020). 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, 2020; PHE, 2021b). Just under 50,000 children aged 0 to 19 years were admitted to hospital to have teeth removed under general anaesthesia in 2019/2020 (PHE, 2021b). This pattern is similar or worse in Scotland, Wales and Northern Ireland (SACN, 2018).

8.6 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 England the prevalence among the most deprived children at age 5 years is 34% and 14% for the least deprived (PHE, 2021c), with 38% of variation in the prevalence of tooth decay explained by deprivation.

Impact of oral health problems on children and families

8.7 Dental caries have 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; PHE, 2022).

UK guidance for oral health improvement

8.8 Existing government guidance on dental caries prevention in young children (DHSC, 2021) include the following recommendations:

- 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 amount and frequency of consumption of sugars-containing foods and drinks
- avoid sugars-containing foods and drinks at bedtime when saliva flow is reduced and buffering capacity is lost
- parents/carers should brush their children's teeth up to the age of 3 years, and brush or supervise toothbrushing 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.

Breastfeeding and bottle feeding and oral health

8.9 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 (see Annex 13, Glossary) (SACN, 2018).

8.10 Human milk, cows' milk and infant formula all contain sugars, primarily lactose. Cows' milk contains approximately 4% lactose while containing high levels of calcium, phosphate and proteins that have a protective effect against dental caries (Grenby et al, 2001). 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). Therefore, it is theoretically possible that exposure to breast milk and infant formula both carry potential risks of dental caries. However, data show that breastfeeding up to 1 year of age is protective against dental caries compared with formula feeding (SACN, 2018). Data for breastfeeding beyond 12 months of age are less clear, as few studies have compared the risk of ECC in children who are breastfed beyond 12 months with children fed formula milk beyond 12 months or weaned onto cows' milk from 12 months (Moynihan et al, 2019).

8.11 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 dental caries in childhood: 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.

8.12 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.

Evidence identified on oral health

8.13 Eight systematic reviews (SRs) were identified that examined the relationship between feeding practices, nutrition 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). Two of the SRs included meta-analyses (MAs) (Moynihan & Kelly, 2014; Thomaz et al, 2018).

8.14 Details of the 8 SRs can be found in Annex 5 (Table A5.6). Quality assessment of the SRs using the AMSTAR 2 tool can be found in Annex 7 (Table A7.9). Additional data extracted on the primary studies can be found in Annex 8 (Table A8.38 to A8.44). The criteria used to grade the evidence are provided in chapter 2 (Table 2.4, paragraphs 2.43 to 2.47). Summary tables of the evidence grading process for this section are provided in Annex 9 (Tables A9.26 to A9.29).

8.15 Key exposures were sugars intake, breastfeeding beyond 12 months, use of bottles for feeding beyond 12 months, night time bottle feeding, milk and dairy consumption, and body weight. Key outcomes were ECC and malocclusion.

Limitations of the evidence on oral health

8.16 Most of the evidence from SRs that examined the relationship between breastfeeding or use of bottles for feeding and ECC risk is derived from studies conducted in LIC or LMIC. This may limit the generalisability of the findings to children living in the UK.

8.17 Primary studies included in the SRs identified varied in the populations included, exposures (for example, duration of breastfeeding versus ever or never breastfed) and outcome measures (for example, caries incidence/prevalence, caries increment, dmfs, dmft – see Annex 13, Glossary), making comparisons between studies difficult.

8.18 When considering the impact of infant and young child feeding practices on risk of dental caries, it is important to take potential modifying and confounding factors into account. Potential confounding factors include the consumption of free sugars from foods and drinks, nocturnal feeding, and household socioeconomic status (SES). Important modifying factors include poor oral hygiene practices (for example, the infrequent or delayed introduction of toothbrushing and not using fluoride-containing toothpaste) and exposure to fluoride through water. These factors might 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.

8.19 Confounding from sugars in the complementary diet (in breast and bottle feeding studies) is often not robustly measured or adequately controlled for in statistical analysis and needs to be considered when assessing the quality of evidence and drawing conclusions on the potential impact of young child feeding practices on caries development.

8.20 Caution should also be applied to findings from studies funded by industries and companies that sell or promote the use of infant formula.

Sugars intake and dental caries

8.21 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.

8.22 Two PCS reported sugars consumption frequency in relation to dental caries risk in the primary dentition but provided little evidence of an association. However, it was unclear what was precisely meant by the exposure term ‘sugar’, as further details were not reported.

8.23 The PCS mostly reported that higher frequencies of intake of sugars-containing drinks (including non-carbonated fruit drinks and fruit juice) increased the risk of caries in primary dentition (5 out of 6 studies).

8.24 Reported associations between frequency of sweets intake (including confectionery and candy) and risk of dental caries in primary dentition were less consistent. Half the studies (2 out of 4) reported an association between higher

frequency of intake and increased risk while the other half found no relationship. The exposure term ‘sweets’ (including confectionery and candy) was unclear, as details were not reported.

8.25 For the current draft report, 2 SRs without MAs (Baghlaf et al, 2018; Hooley et al, 2012b) and 1 SR with MA (Moynihan & Kelly, 2014) were identified that examined the relationship between free sugars’ intake or consumption of sugars-containing foods and drinks and development of dental caries in children. Outcome measures were mostly a measure of caries increment over time using the WHO diagnostic criteria, but also included caries incidence and prevalence.

8.26 Although Moynihan & Kelly (2014) performed MAs, findings from PCS in children aged 1 to 5 years were not pooled into a single MA and are therefore reported separately below.

8.27 Moynihan & Kelly (2014) (AMSTAR 2 confidence rating: high) included 4 PCS that examined the relationship between the intake of free sugars and dental caries in children aged 1 to 5 years. The studies were in children aged 1 to 3 years at baseline and followed up for 1 to 4 years.

8.28 Three PCS (in a total of 1465 participants in mainly UMIC) reported that higher intake of free sugars at ages 3 and 4 years was associated with the development of dental caries 1 and 3 years later; while 1 PCS (in 259 participants) reported no association (see Annex 8, Table A8.38 for detailed results). Of the PCS that reported an association, 2 also reported that restricting intake of free sugars to less than 10% of total dietary energy intake (TDEI) protected against dental caries. For example, 1 study (in Brazil in 510 participants) reported that children who consumed more than 10% TDEI (32.6 grams) from free sugars per day were 2.99 times more likely to have a high caries increment than those consuming less than 10% TDEI from sugar (adjusted OR 2.99; 95% CI 1.82 to 4.91; p<0.001). Two of the 3 studies that reported an association accounted for differences in fluoride exposure and adjusted for SES and toothbrushing. The study that reported no association did not adjust for confounding factors.

8.29 Baghlaf et al (2018) (AMSTAR 2 confidence rating: high) examined the impact of sugars’ intake around bedtime on dental caries and included 1 PCS in children aged 1 to 5 years. The PCS (in 1782 participants in Singapore) 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 controlled for frequency of between-meal intake of sweets, tooth-brushing, and fluoride exposure (although not specifically bedtime tooth brushing).

8.30 Hooley et al (2012b) (AMSTAR 2 confidence rating: critically low) included 3 additional PCS (all in HIC) that examined the relationship between intakes of sweet foods and drinks and ECC (caries increment and prevalence). The SR reported that ‘frequent’ consumption (frequency unspecified) of sweet foods and

drinks (including adding sugars to fluids and solids) at ages 18 months and 3 years was associated with increased caries increment 1 to 3.5 years later in all 3 PCS (in a total of 883 participants) (statistics NR). All 3 studies adjusted for a combination of confounding factors, including SES (in 2 studies) and tooth-brushing (in 3 studies).

Summary: sugars intake and dental caries

8.31 The evidence on intake of free sugars and sugars-containing foods and drinks and dental caries is summarised in Table 8.1.

Table 8.1. Summary of the evidence on sugars intake and dental caries

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Free sugars intake	Dental caries	↑	Adequate

[1] Direction of association for reported outcomes: ↑increase; ↓inverse.

8.32 The available evidence from SRs on the relationship between intake of free sugars, and sugars-containing foods and drinks, and dental caries in children is from 3 SRs without MAs, 2 given a high confidence rating using the AMSTAR 2 tool, and 1 given a critically low confidence rating.

8.33 Evidence from 7 PCS (out of 8 PCS) included in the 3 SRs by Moynihan & Kelly (2014), Baghlaf et al (2018) and Hooley et al (2012b) suggests that higher intake of free sugars in children aged 1 to 5 years is associated with increased dental caries (increment, incidence or prevalence) in later childhood. The evidence 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. These findings strengthen those from the SACN report 'Carbohydrates and health'.

8.34 This evidence is consistent with current UK government recommendations that state that the amount and frequency of consumption of sugars-containing food and drinks should be minimised to prevent caries development in children aged 0 to 6 years old (DHSC, 2021).

Breastfeeding and dental caries

8.35 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). As this draft report is concerned with children aged 1 to 5 years, 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 was explored.

8.36 One SR with MA was identified that examined the relationship between breastfeeding beyond 12 months and ECC risk (Cui et al, 2017; Tham et al, 2015). However, the MA conducted by Tham et al (2015) pooled estimates from PCS together with those from cross-sectional and case-control studies. Therefore, findings from individual PCS were considered and are reported separately below. A second SR without MA (Hooley et al, 2012b), which had a much broader research question than modes of feeding, but included 1 additional PCS that examined the relationship between breastfeeding beyond 12 months and ECC risk, is also described below.

8.37 Primary studies in children aged 1 to 5 years included in the 2 SRs were mainly conducted in HIC and UMIC.

8.38 Tham et al (2015) (AMSTAR 2 confidence rating: low) included 4 PCS which examined the relationship between breastfeeding beyond 12 months and ECC risk.

8.39 The 4 PCS compared different breastfeeding durations, with some studies making multiple comparisons. Altogether there were 7 comparisons. Two PCS compared breastfeeding for 12 months and beyond on the risk of ECC (or severe ECC) with breastfeeding for less than 6 months. Three PCS compared breastfeeding for 18 months and beyond with not breastfeeding at 18 months. Two PCS compared breastfeeding for 24 months and beyond with not breastfeeding at 24 months.

8.40 Two out of 2 PCS reported that breastfeeding beyond 12 months was not associated with later ECC (or severe ECC) compared with breastfeeding for less than 6 months. One PCS (in 537 participants, in Brazil) reported an OR of 1.39 (95% CI 0.73 to 2.64; p-value not reported) for severe ECC at age 38 months for children breastfed for 12 to 23 months. The analysis was adjusted for intake of sugars-containing foods and drinks and SES but did not account for oral hygiene practices and fluoride use. The other PCS (in 315 participants, in Japan) reported an OR of 1.09 (95% CI 0.45 to 2.71; p-value not reported) for ECC at age 41 to 50 months for children breastfed for 12 to 17 months. The analysis was adjusted for bottle feeding (sweetened liquids other than milk), bottle feeding while falling asleep, age of introduction of foods, oral hygiene practices, fluoride use, SES but not intake of sugars-containing foods.

8.41 Three out of three PCS reported that breastfeeding for 18 months and longer was associated with increased ECC risk compared with not breastfeeding at 18 months. One PCS (in 315 participants, in Japan) reported an OR of 2.47 (95% CI 0.95 to 6.59; p-value not reported) at age 41 to 50 months for children breastfed for 18 months and longer compared with children breastfed for less than 6 months. The analysis was adjusted for bottle feeding (sweetened liquids other than milk), bottle feeding while falling asleep, age of introduction of foods, oral hygiene

practices, fluoride use, SES but not intake of sugars-containing foods. The other 2 PCS did not adjust for any confounding factors. One PCS (in 392 participants, in Japan) reported an OR of 6.65 (95% CI 2.89 to 15.2; p<0.05) for a caries increment at age 3 years. The other PCS (in 592 participants, in Japan) reported that children breastfed at 18 months had more carious teeth than children not breastfed at 18 months (mean decayed filled teeth 0.36 versus 0.06; p<0.05).

8.42 Two out of 2 PCS reported that breastfeeding for 24 months and longer was associated with increased ECC risk compared with not breastfeeding at 24 months. One study (in 537 participants, Brazil) reported a higher prevalence of severe ECC in children breastfed for 24 months and beyond compared with children breastfed for less than 6 months in 2 statistical models: PR 2.10 (95% CI 1.50 to 3.25; p-value not reported) in a marginal structural model (which accounts for time-varying confounding); and PR 1.85 (95% CI 1.11 to 3.08; p-value not reported) in a standard multiple regression model. The analyses were adjusted for intake of sugars-containing foods and drinks and SES but did not account for oral hygiene practices and fluoride use. The second PCS (in 592 participants, in Japan) reported that children breastfed at 24 months had more carious teeth than children not breastfed at 24 months (mean decayed filled teeth 0.51 versus 0.11; p<0.05). The study did not adjust for any confounding factors.

8.43 Hooley et al (2012b) (AMSTAR 2 confidence rating: critically low) included 1 additional PCS (in Turkey, 56 participants) that examined whether there is a relationship between breastfeeding beyond 12 months and ECC risk 2 years later and reported no association (statistics NR). The study did not adjust for confounding and had a low participant retention rate (<40%) at the final study measurement, which his another potential source of bias.

Summary: breastfeeding and dental caries

8.44 The evidence on breastfeeding beyond 12 months and ECC risk is summarised in Table 8.2.

Table 8.2. Summary of the evidence on breastfeeding beyond 12 months and dental caries

Exposure	Outcome	Direction of association	Certainty of evidence
Breastfeeding beyond 12 months	ECC	Inconsistent	Insufficient

Abbreviations: ECC, early childhood caries.

8.45 The available evidence from SRs on any relationship between breastfeeding beyond 12 months and ECC risk in children is from 2 SRs without MAs, 1 given a low confidence rating using the AMSTAR 2 tool, the other given a critically low confidence rating.

8.46 Evidence from 5 PCS (8 comparisons) from the 2 SRs by Tham et al (2015) and Hooley et al (2012b) was inconsistent. The evidence was graded **insufficient** due to the inconsistency in findings, heterogeneity of breastfeeding durations and comparators, limited adjustment for key confounding and modifying factors (particularly in studies on breastfeeding ≥18 months and ≥24 months) and unclear generalisability of the findings to the UK population.

Use of bottles for milk feeds and dental caries

8.47 One SR without MA (Hooley et al, 2012b) was identified that included PCS that examined the relationship between use of bottles for milk feeds beyond 12 months and ECC risk (although this was not the focus of the SR). However, the research question and search strategy of this SR encompassed any parental/caregiver practices that might relate to dental caries development and was not particular to modes of feeding. Therefore, the literature search conducted by Hooley et al (2012b) cannot be said to be comprehensive for identifying studies on the use of bottles for feeding and the development of dental caries.

8.48 Hooley et al (2012b) (AMSTAR 2 confidence rating: critically low) included 2 PCS (in a total of 684 participants) that reported no association between use of bottles for milk feeds beyond 12 months and ECC risk at age 18 to 36 months (statistics NR). Neither of the studies adjusted for key confounding factors. In one PCS, the content of the bottle was sweetened milk; while in the other study, the content of the bottle was not stated. However, it can be assumed that it was milk given that the study compared ‘being bottle fed’ with ‘being breastfed’. The studies were conducted in children from Japan and Turkey and therefore their findings may have limited generalisability to children living in the UK.

Summary: use of bottles for milk feeds and dental caries

8.49 The evidence on use of bottles for milk feeds beyond 12 months and ECC risk is summarised in Table 8.3.

Table 8.3. Summary of the evidence on use of bottles for milk feeds and dental caries

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Bottle milk feeds	ECC	N/A	Insufficient

Abbreviations: ECC, early childhood caries; N/A, not applicable.
[1]N/A: not enough evidence to draw conclusions and recommendations.

8.50 The available evidence from SRs on the relationship between use of bottles for milk feeding beyond 12 months and ECC risk is from 1 SR without MA given a critically low confidence rating using the AMSTAR 2 tool.

8.51 There was **insufficient** evidence from SRs to enable conclusions to be drawn on the relationship between use of bottles for milk feeding beyond 12 months and ECC risk as fewer than 3 primary studies included in the SR examined this relationship.

8.52 However, as sugars-containing drinks may also be given by bottle, the current advice (DHSC, 2021) 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.

Night time bottle feeding (milk) and dental caries

8.53 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 infant formula. Available studies in children aged 1 to 5 either do not include breastfeeding during the night or are unclear whether they include this mode. This section does not include other foods and drinks consumed around bedtime (see paragraph 8.29 for this exposure).

8.54 One SR without MA (Hooley et al, 2012b) (AMSTAR 2 confidence rating: critically low) included 2 PCS that examined the relationship between bottle feeding (milk) at night time in children beyond 12 months and ECC risk. As the research question and search strategy of this SR encompassed any parental/caregiver practices that might relate to dental caries development and was not particular to modes of 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.

8.55 The 2 PCS (in a total of 1764 participants from HIC in East Asia) 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 an increased ECC risk at age 3 to 7 years (statistics NR). Both studies adjusted for toothbrushing, and 1 study also adjusted for a crude assessment of sugars intake between meals.

Summary: night time bottle feeding (milk) and dental caries

8.56 The evidence on night time bottle feeding (milk) beyond 12 months and ECC risk is summarised in Table 8.4.

Table 8.4. Summary of the evidence on night time bottle feeding (milk) beyond 12 months and dental caries

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Night time bottle feeding (milk) beyond 12 months	ECC	N/A	Insufficient

Abbreviations: N/A; not applicable.

[1] N/A: not enough evidence to draw conclusions and recommendations.

8.57 The available evidence from SRs on the relationship between night time bottle feeding (milk) beyond 12 months and ECC risk is from 1 SR without MA given a critically low confidence rating using the AMSTAR 2 tool.

8.58 There was **insufficient** evidence to enable conclusions to be drawn on any relationship between night time use of bottles for milk feeding beyond 12 months and ECC risk in young children as fewer than 3 primary studies included in the SR examined this relationship.

Milk and dairy consumption and dental caries

8.59 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 different dairy products and dental caries in children aged 1 to 5 years. The PCS (in 642 participants) reported that the median milk consumption at ages 2 and 3 years was lower in children who had 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) non-milk dairy consumption at ages 1 to 3 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) non-milk dairy consumption. The analyses were adjusted for age at dental examination, sex, fluoride exposure, and dietary variables.

Summary: milk and dairy consumption and oral health

8.60 The evidence identified from SRs on milk and dairy consumption and oral health is summarised in Table 8.5.

Table 8.5 Summary of the evidence on dairy consumption and oral health

Exposure	Outcome	Direction of effect/association[1]	Certainty of evidence
Milk	Dental caries	N/A	Insufficient
Non-milk dairy	Dental caries	N/A	Insufficient

Abbreviations: N/A; not applicable.

[1] N/A: not enough evidence to draw conclusions and recommendations.

8.61 The available evidence on the relationship between milk and dairy consumption and oral health is from 1 SR given a critically low confidence rating using the AMSTAR 2 tool.

8.62 There was **insufficient** evidence 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.

Breastfeeding or use of bottles for feeding and malocclusion

8.63 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.

8.64 Malocclusion has been suggested to vary between breast and bottle fed children. The biological plausibility 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.

8.65 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).

8.66 For this draft 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 risk. All the studies identified by Hermont et al (2015) on this relationship were 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.

8.67 Thomaz et al (2018) (AMSTAR 2 confidence rating: moderate) reported that breastfeeding beyond 12 months was associated with a decreased risk 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 risk 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).

8.68 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 malocclusion risk. 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: breastfeeding or use of bottles for feeding and malocclusion

8.69 The evidence on breastfeeding or use of bottles for feeding beyond 12 months and malocclusion risk is summarised in Table 8.6.

Table 8.6. Summary of the evidence on breastfeeding or use of bottles for feeding beyond 12 months and malocclusion risk

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Breastfeeding beyond 12 months	Malocclusion risk	↓	Moderate
Use of bottles for feeding beyond 12 months	Malocclusion risk	N/A	Insufficient

Abbreviations: N/A; not applicable.
[1] Direction of association for reported outcomes: ↑increase; ↓inverse; N/A: not enough evidence to draw conclusions and recommendations

8.70 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.

8.71 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).

8.72 There was **insufficient** evidence to enable conclusions to be drawn on the relationship between use of bottles for feeding beyond 12 months and malocclusion risk as fewer than 3 primary studies included in the SR by Hermont et al (2015) examined this relationship.

Body weight and dental caries

8.73 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, 2019).

8.74 To determine whether there is a relationship between weight status and dental caries, studies need to test against a full range of BMI or weight categories. This is because there is some evidence that the association between BMI or body weight and dental caries in primary dentition is U-shaped and that both overweight and underweight children are at increased risk of dental caries (Hooley et al, 2012a), especially for primary dentition (Tanner et al, 2022).

8.75 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 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 African-American children from low-income households in the USA) 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 dental caries 2 years later. The study adjusted for age, sugars-sweetened beverage consumption, toothbrushing, baseline caries and SES. However, the SR noted that this study assumed a linear relationship between body weight and dental caries, even though almost 25% of the children in the sample had a low weight-for-age percentile.

Summary: body weight and dental caries

8.76 The evidence on the relationship between body weight and dental caries in children aged 1 to 5 is summarised in Table 8.7.

Table 8.7. Summary of the evidence on body weight and dental caries

Exposure	Outcome	Direction of association[1]	Certainty of evidence
Body weight	Dental caries	N/A	Insufficient

Abbreviations: N/A; not applicable.

[1] N/A: not enough evidence to draw conclusions and recommendations.

8.77 The available evidence from SRs on any relationship between body weight and 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 deficiency and dental caries

8.78 Inadequate vitamin D status can have an impact on oral health by interference in the mineralisation of teeth during their development. For example, individuals with rickets, a condition usually caused by lack of vitamin D or calcium, have a higher risk of dental enamel defects, including enamel hypoplasia, which increases susceptibility to dental caries (Zerofsky et al, 2016).

8.79 There is a paucity of evidence examining the effects of vitamin D supplementation and oral health in young children. For this draft report, 1 SR with MA (Hujoel (2013) was identified that examined the effect of vitamin D supplementation on dental caries in children. However, 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 Risks of chemical toxicity

9.1 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.

9.2 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.

9.3 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 9.1 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).

9.4 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 9.2.

Table 9.1. 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)	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. COT agrees with the overall conclusion by EFSA. **Chronic dietary exposure to chlorate is of potential concern for high consumers, particularly to individuals with mild to moderate iodine deficiency.** 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.
Contaminants and process contaminants	Furan and methylfurans (COT, 2019)	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[6] for ready-to eat-meals and total exposure are of **potential toxicological concern**. 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.

[6] Following EFSAs approach the exposure estimates were calculated using age categories 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 draft report.

Substance category	Chemical considered	Summary of COT conclusions
		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.
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 **not of toxicological concern.**
Contaminants and process contaminants	Legacy chemicals[1] (COT, 2019)	Although these chemicals are persistent in the environment, their levels have decreased since their use was banned. **As the levels for legacy chemicals are expected to further decline,** COT confirmed the conclusions of its previous assessments, that **there is no indication of concern** 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 **potential concern** for young children aged 12 to 60 months. 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.

Substance category	Chemical considered	Summary of COT conclusions
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 **potential concern**, 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 (BaP and PAH4) from human breast milk and food are of **low concern** for health for children aged 1 to 5 years. Intakes from soil and dust are not expected to contribute markedly to lifetime exposure.
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 **not to be cause for concern** 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.

Substance category	Chemical considered	Summary of COT conclusions
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 **not of toxicological concern**.
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, **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**.
Natural Toxins - Mycotoxins	Citrinin (COT, 2020)	COT concluded that exposures to citrinin are **not of toxicological concern for nephrotoxicity**. However, it was noted that due to lack and limitations of the available data, **a concern for genotoxicity and carcinogenicity cannot be excluded**.
Natural Toxins - Mycotoxins	Cyclopiazonic acid (COT, 2020)	COT concluded that the exposures in the diet of children aged 1 to 5 years are **not of toxicological concern.**
Natural Toxins - Mycotoxins	Diacetoxyscir-penol (COT, 2020)	COT concluded that the exposures in the diet of children aged 1 to 5 years are **not of toxicological concern.**

Substance category	Chemical considered	Summary of COT conclusions
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 **unlikely to be of toxicological concern**. 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 **not of toxicological concern**.
Natural Toxins - Mycotoxins	Fumonisins (COT, 2020)	COT concluded that the exposures in the diet of children aged 1 to 5 years are **not of toxicological concern**.
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 **not of toxicological concern**. 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 **not of toxicological concern**.
Natural Toxins - Mycotoxins	Nivalenol (COT, 2020)	COT concluded that the exposures in the diet of young children aged 1 to 5 years are **not of toxicological concern**.

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 **not of toxicological concern**, but this is **contingent on resolution of the genotoxic potential of PAT**.
Natural Toxins - Mycotoxins	Sterigmatocystin (COT, 2020)	COT concluded that the exposures in the diet of young children aged 1 to 5 years are **not of toxicological concern**.
Natural Toxins - Mycotoxins	Zearalenone (COT, 2020)	COT concluded that the exposures in the diet of young children aged 1 to 5 years are **not of toxicological concern**.
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[2] 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)	Overall, all estimated acute exposures of young children aged 1 to 5 years to (-)-hyoscyamine and (-)-scopolamine or the sum of **(-)-hyoscyamine and (-)-scopolamine are unlikely to be of toxicological concern**. 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 **high degree of uncertainty to the risks associated with total TAs** in the diet.
Nutrients	Chromium (COT, 2019)	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 **not to be of toxicological concern**. 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.
Nutrients	Selenium (COT, 2019)	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 **unlikely to be of toxicological concern**.

Substance category	Chemical considered	Summary of COT conclusions
Nutrients	Vitamin A (COT, 2019)	Following its update in 2017, COT concluded that the **possibility of adverse effects cannot be excluded in high consumers, primarily those who regularly eat liver**. However, if effects did occur it would be in a small proportion of consumers. 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.
Nutrients	Zinc (COT, 2019)	COT concluded, that overall, estimated dietary exposures do not indicate excessive zinc intakes and are therefore **unlikely to be of toxicological concern**. 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.

[1] (including aldrin, dieldrin, endrin, chlordane, heptachlor, hexachlorobenzene, mirex, toxaphene, DDT, endosulfan, pentachlorobenzene, chlordecone).

[2] 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 9.2. 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 did not suggest **any 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.	Potential risks from acrylamide in the diet of infants and young children (COT, 2016)
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, **do not indicate toxicological concerns** or a need for any modification in advice to Government.	Potential risks from aluminium in the infant diet (COT, 2013) and addendum (COT, 2016)
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 **potential concern** 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 that the current	Statement on arsenic in infants and young children (COT, 2016) Editorial update

Chemical considered	Summary of COT conclusions	Web link
	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	COT is awaiting EFSA's new scientific opinion to conclude if a new COT evaluation is required.	
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 **not a major cause for concern**.	The potential risks from cadmium in the infant diet (COT, 2018)
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 **no toxicological concern** to the health of infants and young children with normal copper homeostasis.	Potential risks from copper in the diets of infants and young children (COT, 2018)
Dioxins and dioxin-like compounds	In 2018, EFSA established a new TWI for dioxin. Due to the uncertainties in EFSAs 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 review the basis and values of the WHO toxic equivalent factors (TEFs). A finalised	Dioxin position paper (COT, 2021) https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/j.efsa.2018.5333

Chemical considered	Summary of COT conclusions	Web link
	assessment by the EC is not expected until 2022, at the earliest. 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.	
Hexabromocyclododecane (HBCDDs)	COT concluded that while the **level of HBCDDs in the diet of young children was not a cause for concern**, 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.	Hexabromocyclododecanes statement (COT, 2015) and addendum (COT, 2016)
Iodine	COT concluded that at current intakes, excess iodine is **unlikely to pose a toxicological risk to health**.	The potential risks from excess iodine (COT, 2017)
Lead	COT concluded that for young children, the **risk from dietary exposure alone is small** 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.	Potential risks from lead in the infant diet (COT, 2013) and addendum (COT, 2016)
Manganese	COT was **unable to draw firm conclusions** 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 dietary exposures. Further data is required to refine this risk assessment, although any	The health effects of manganese in the diets of infants and young children (COT, 2018)

Chemical considered	Summary of COT conclusions	Web link
	risk at current dietary exposures is likely to be low.	
Methylmercury	COT concluded that when taking into consideration the high degree of conservatism in the exposure modelling, there was **low risk to health** 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.	Potential risks from methylmercury in the diet of infants and young children (COT, 2018)
Nickel	COT concluded that **chronic exposure to nickel from food was of no toxicological concern** 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.	Potential risks from nickel in the diet of infants and young children (COT, 2018)
Ochratoxin A (OTA)	COT concluded that in young children consuming commercial foods for these age groups, exposures were well below the TWI and hence there was **no toxicological concern**.	Potential risks from ochratoxin A (OTA) in the diet of infants and young children (COT, 2018)
Perfluorooctanesulfonic (PFOS) acid and Perfluorooctanoic (PFOA) acid	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 currently not sufficiently sensitive. Furthermore, it is unclear what impact processing has on exposure as well as the impact of co-	https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/j.efsa.2018.5194 PFAS opinion for public consultation http://www.efsa.europa.eu/en/consultations/call/public-consultation-draft-scientific-opinion-risks-human-health

Chemical considered	Summary of COT conclusions	Web link
	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 EFSAs publication on PFAS[7]. The COT[8] 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.	
Phthalates	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. 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.	http://www.efsa.europa.eu/sites/default/files/consultation/consultation/Phthalates_in_plastic_FCM_draft_opinion_for_public_consultation.pdf
Plant Based Drinks	COT assessment on the potential risks posed by soya, oat and almond drinks	Overarching statement on consumption of

[7] INSERT COT meeting minutes March

[8] INSERT COT meeting minutes May

Chemical considered	Summary of COT conclusions	Web link
	consumed in the diets of infants and young children concluded that neither the safety of these drinks, nor the suitability of the current guidance, could be confirmed from a toxicological perspective.	plant-based drinks in children aged 6 months to 5 years (COT, 2021)
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 **did not indicate a cause for concern**.	Polybrominated biphenyls (PBBs) in the infant diet (COT, 2015)
Polybrominated diphenyl ethers (PBDEs)	COT concluded that the **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.** 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. **COT however recommended for monitoring to be continued to ensure levels are declining as expected**.	Potential risks from polybrominated diphenyl ethers (PBDEs) in the infant diet (COT, 2015) and addendum (2017)
T-2 toxin, HT-2 toxin and neosolaniol	Whilst an effect on health cannot be entirely excluded at the 97.5th 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 **unlikely to be of any toxicological concern** in young children.	T-2 toxin, HT-2 toxin and neosolaniol in the diets of infants and young children (COT, 2018)

Conclusions

9.5 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).

9.6 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.

9.7 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.

9.8 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.

9.9 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.

9.10 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.

9.11 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.

9.12 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.

9.13 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.

9.14 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.

9.15 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.

9.16 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.

9.17 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 once the joint working group has completed its work and the joint statement has been finalised.

10 Overall summary and conclusions

Background

10.1 This draft report considers the scientific basis of current 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. Existing recommendations for feeding children aged 1 to 5 years are provided in Table 11.1. The draft 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.

10.2 The draft report considers 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 or non-randomised intervention studies. In addition, food and nutrient intake and status data were obtained from the National Diet and Nutrition Survey (NDNS) and the Diet and Nutrition Survey of Infants and Young Children (DNSIYC). Data from the National Child Measurement Programme (NCMP) were also considered in relation to the prevalence of overweight and obesity among children aged 4 and 5 years. Data from national dental health surveys were considered in relation to the prevalence of dental caries in children up to 5 years of age.

10.3 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 9, Table A9.29. The approach taken to grade the evidence is described in Chapter 2.

10.4 Throughout this summary section, 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 survey data and assessment of systematic review evidence

Energy and macronutrients

Energy

10.5 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 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.

10.6 There is **moderate** evidence from SRs that increasing portion sizes (in grams or energy intake) of snacks and meals in preschool settings increases children's food and energy intake in the short term (interventions lasting for up to 3 months).

Carbohydrates

10.7 The current government recommendation for total carbohydrate intake is that it should contribute approximately 50% of total dietary energy. Data from DNSIYC and NDNS indicated that, on average, this was achieved in most age groups. Mean total carbohydrate intake contributed on average 49% of total dietary energy intake (TDEI) in children aged 12 to 47 months and 51% in children aged 48 to 60 months.

10.8 The recommendation for intake of free sugars is that their contribution to TDEI should not exceed 5%. This recommendation currently applies from age 2 years and above. Data from NDNS indicated that mean intake of free sugars was double the maximum recommendation for children aged 18 to 47 months (10% of TDEI) and children aged 48 to 60 months (12% of TDEI). Eighty-five percent and 97% of children in these age groups, respectively, had intakes above the recommendation of 5% of TDEI.

10.9 The DRV for dietary fibre for children aged 2 years and older is 15 grams per day. Data from NDNS indicated that mean dietary fibre intake was lower than recommended at 10 grams per day for children aged 18 to 47 months and 13 grams per day for 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 dietary fibre intakes below the DRV.

10.10 There is **adequate** evidence from SRs that higher intake of free sugars (from food and drinks) in children aged 1 to 5 years is associated with increased dental caries (increment, incidence or prevalence) in later childhood.

10.11 There is **adequate** evidence from SRs that higher consumption of sugars-sweetened beverages (SSBs) in children aged 1 to 5 years is associated with an increased odds of overweight or obesity in later childhood, adjusted for total dietary energy intake (TDEI). There is **moderate** evidence that higher SSBs consumption in children aged 1 to 5 years is associated with a greater increase in BMI (or BMI z-score/weight-for-height z-score) in later childhood, unadjusted for TDEI (see Chapter 3, paragraph 3.45).

Dietary fat

10.12 The DRV for total dietary fat intake is that it should contribute no more than 33% of TDEI. The DRV applies from age 5 years onwards, while 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. Data from DNSIYC and NDNS indicated that the mean intake of total dietary fat as a % TDEI was 35% in children aged 12 to 47 months and 33% in children aged 48 to 60 months. Fifty-three percent of children aged 4 to 5 years (48 to 60 months) had intakes above the DRV.

10.13 The DRV for saturated fat intake is that it should contribute no more than 10% TDEI. The DRV applies from 5 years of age onwards, while a flexible approach is currently recommended to the timing and extent of dietary change for individual children between 2 and 5 years. Data from DNSIYC and NDNS indicated that mean saturated fat intake was 16% of TDEI in children aged 12 to 18 months and 14% in children aged 48 to 60 months where 91% of children had intakes above the DRV.

10.14 There is **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 paragraph 3.166.

10.15 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

10.16 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 46 grams per day, more than twice the RNI for this age group.

10.17 There is **moderate** evidence from SRs that higher total protein intake in children aged 1 to 5 years is associated with increased BMI in later childhood. The role of TDEI is uncertain in this relationship (see Chapter 3 paragraph 3.262).

10.18 There is **limited** evidence from SRs that higher animal protein intake in children aged 1 to 5 years is associated with earlier measures of puberty timing, menarche in girls or voice break in boys.

Micronutrients

10.19 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. Data from NDNS 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 evidence to suggest underreporting in children with intakes below the LRNI for these micronutrients.

10.20 Analyses of data from NDNS (years 2008/09 to 2018/19 of the rolling programme) indicated that inadequate intakes of iron, zinc, vitamin A or vitamin D may be more prevalent among children from lower socioeconomic status households and certain ethnic minority groups. Children with intakes below the LRNI did not obtain any vitamin A from dietary supplements. Current government advice is that all children aged 6 months to 5 years should be given a vitamin supplement containing vitamin A.

10.21 Despite NDNS data indicating that mean intake of vitamin A are 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 4.169 and 4.170).

10.22 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 there are uncertainties in the iron DRVs for children.

10.23 Data from NDNS indicated that 7% of children aged 18 to 47 months had plasma retinol concentrations between 0.35 µmol/L and 0.70 µmol/L, the range associated with mild vitamin A deficiency in adults[9].

10.24 The RNI for vitamin D for children aged 1 to 5 years is 10µg 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. Analysis of data from NDNS (years 2012/13 to 2016/17) 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, data from NDNS (years 2008/09 to 2018/19) indicate that, compared with white children, young children from ethnic minorities are likely to be at higher risk of vitamin D deficiency.

10.25 Data from NDNS 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 data from NDNS (years 2012/13 to 2016/17) for children aged 18 to 36 months indicated that serum 25(OH)D concentrations decreased with increasing deprivation (as measured by equivalised household income).

10.26 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 are adequate with no children with intakes below the LRNI.

10.27 There is **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.

10.28 There is **limited** evidence from SRs that vitamin D fortification of milk or formula improves vitamin D status or decreases the risk of vitamin D deficiency in children aged 1 to 5 years.

Foods, dietary components, and dietary patterns

10.29 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. Data from DNSIYC and NDNS indicated that children aged 12 to 18 months consumed, on average, 170 grams per day of vegetables (excluding potatoes) and fruit (excluding fruit juice). For

[9] There is no equivalent threshold in children

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. In all age groups fruit made a greater contribution to intakes than vegetables.

10.30 Data from NDNS indicated that total milk consumption (all types of cows' milk and other dairy milk) contributed 16% to TDEI for children aged 18 to 47 months, falling to 10% for children aged 48 to 60 months. The contribution of total dairy intake to TDEI was 22% and 15% for the 2 age groups, respectively.

10.31 Substitution modelling 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.

10.32 Data from NDNS indicated that foods rich in starchy carbohydrates contributed 21% and 20% to TDEI in children aged 18 to 47 months, and aged 48 to 60 months, respectively.

10.33 Data from NDNS indicated that non-dairy sources of protein contributed 9% to TDEI in children aged 18 to 60 months, with unprocessed meat making the largest contribution (8% of TDEI).

10.34 Data from NDNS indicated that foods high in (total) fat, salt and free sugars contributed 27% of TDEI for children aged 18 to 47 months. This increased to 34% of TDEI for children aged 48 to 60 months.

10.35 There is **limited** evidence from SRs that higher fruit juice consumption is associated with increased BMI when unadjusted for TDEI, and **limited** evidence that fruit juice consumption is not associated with BMI following adjustment for TDEI (see Chapter 5, paragraph 5.34).

10.36 There is **moderate** evidence from SRs of no association between total milk intake in children aged 1 to 5 years and BMI in later childhood.

10.37 There is **limited** evidence from SRs that dietary patterns classified as 'unhealthy' are associated with higher body fat measures in children aged 1 to 5 years.

Eating and feeding behaviours

10.38 There is **moderate** evidence from SRs that feeding practices (including repeated exposure and pairing vegetables with positive stimuli) can increase children's vegetable consumption in the short term (up to 8 months).

10.39 There is **moderate** evidence from SRs that repeated taste exposure (around 8-10 times) 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).

10.40 There is **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.

10.41 There is **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

10.42 Data from NCMP shows that the combined prevalence of overweight and obesity (85th and 95th centiles, respectively) for children living in England in Reception year (aged 4 to 5 years) was fairly stable at around 22% to 23% from the collection years 2006/2007 to the collection year preceding the COVID-19 pandemic (2019/2020). In the latest collection year 2020/2021, the combined prevalence of overweight and obesity increased substantially to approximately 28%. In addition, there were large increases in the proportion of children aged 4 to 5 years categorised as obese (including severely obese) (from 10% to 14%) and severely obese (from 3% to 5%). The prevalence of obesity was highest for children categorised as Black African (23.5%) and Black other (22.7%). The gap in obesity prevalence between children living in the most and least deprived areas also increased from 5.3% in 2006/7 to 7.3% in 2019/20, and this gap increased further during the first year of the COVID-19 pandemic to 12.4% (2020/21). The overall increase in prevalence of overweight and obesity may in part be explained by the reported decrease in physical activity levels leading up to and during the first national lockdown in the UK due to the pandemic. In Scotland, the latest available survey data from 2019 showed that the combined prevalence of overweight and obesity (85th and 95th centiles, respectively) for children aged 2 to 6 years was 30%. The 85th and 95th centiles used are intended for population monitoring use only, and do not provide the number or percentage of individual children clinically defined as overweight or obese.

10.43 There is **limited** evidence from SRs that adiposity rebound occurring before the age of 5 years is associated with a higher risk of obesity in adulthood.

10.44 There is **adequate** evidence from SRs 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.

10.45 There is **moderate** evidence from SRs of no association between BMI at age 6 years and under and adult coronary heart disease.

10.46 There is **moderate** evidence from SRs of no association between BMI at age 6 years and under and adult stroke.

Oral health

10.47 Dental caries in children remains a major public health problem despite reductions in prevalence of dental caries since the 1970s. National dental health surveys showed that 40% of children aged 5 years in Northern Ireland (2013), 34% in Wales (2015/6), 27% in Scotland (2020), and 23% in England (2019) had tooth decay.

10.48 There is **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 later childhood.

10.49 There is **moderate** evidence from SRs that breastfeeding beyond 12 months protects against the development of malocclusion.

Table 10.1 Exposure-outcomes relationships graded moderate or adequate

Exposure (children aged 1 to 5 years)	Outcome[1]	Direction of effect or association[2]	Certainty of evidence
Portion sizes	Food and energy intake (short term, up to 3 months)[3]	↑	Moderate
Sugars-sweetened beverages	Odds of overweight BMI or body weight	↑	Adequate Moderate
Total protein intake	BMI	↑	Moderate
Total milk intake[4]	BMI	Null	Moderate
Feeding practices	Vegetable consumption (short term, up to 8 months)	↑	Moderate
Repeated taste exposure	Vegetable consumption (short term, less than 8 months)	↑	Moderate
Child BMI or weight status	Adult BMI or risk of adult overweight or obesity	↑	Adequate
Child BMI[5]	Adult coronary heart disease	Null	Moderate
Child BMI[5]	Adult stroke	Null	Moderate
Breastfeeding beyond 12 months	Malocclusion	↓	Moderate
Free sugars intake	Dental caries	↑	Adequate

[1] Mostly aged over 5 years through to adolescence.
[2] Direction of association for reported outcomes: ↑increase; ↓inverse.
[3] In preschool settings.
[4] Often unspecified, presumed cows' milk.
[5] Children aged under 6 years.

Overall conclusions

10.50 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.

10.51 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), while acceptable in many respects, does not meet current dietary recommendations for several nutrients.

Energy and macronutrients

10.52 Evidence from DNSIYC and NDNS indicated that:

- mean intakes of energy for children aged 12 to 35 months are above the Estimated Average Requirement (EAR) for dietary energy; the percentage above the EAR decreases with increasing age and is less than half by age 5 years
- mean intakes of protein exceed the RNI
- mean intakes of free sugars for children aged 18 to 60 months exceed current recommendations of no more than 5% of TDEI
- mean intakes of saturated fats exceed the population recommendation of no more than 10% TDEI (it should be noted that this recommendation currently does not apply in full until age 5 years)
- foods high in (total) fat, salt and free sugars provide over a quarter of TDEI in children aged 18 to 47 months and over a third of TDEI in children aged 48 to 60 months
- mean intakes of dietary fibre for children aged 18 to 60 months are below the recommended intake of 15 grams per day.

10.53 Evidence identified from SRs indicates that:

- increasing portion sizes in preschool settings increases food and energy intakes in the short term (interventions lasting for up to 3 months)
- higher total protein intake in children aged 1 to 5 years is associated with increased BMI in later childhood
- higher childhood BMI is associated with adult overweight or obesity

10.54 Evidence from SRs also indicates that a higher intake of free sugars (from food and drinks) in children aged 1 to 5 years is associated with dental caries development in later childhood.

10.55 These findings are of concern in relation to wider evidence on:

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

Micronutrients

10.56 Certain groups of children, including children from lower socioeconomic status households (as measured by the Index of Multiple Deprivation) and ethnic minority groups, may be at risk of inadequate intakes of iron, zinc and vitamin A, and inadequate dietary vitamin D intakes and vitamin D status. There is evidence of low uptake of government advice that children aged up to 5 years should be given vitamin supplements.

10.57 Current intakes of vitamin C exceeded the RNI across all age groups.

Vegetables and fruit

10.58 Currently there are no UK government recommendations on portion sizes for vegetables and fruit for young children. Findings from NDNS indicate that in all age groups fruit made a greater contribution to intakes than vegetables. Vegetables and fruit consumption appears to decrease with increasing deprivation. Maintaining consumption of vegetables as children grow up and develop more independence around food is important to ensure diet quality and adherence to population dietary guidelines.

10.59 Evidence identified from SRs indicates 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.

Drinks

10.60 The available evidence indicates that continued breastfeeding beyond the age of 1 year is protective against malocclusion.

10.61 Substitution modelling using data from DNSIYC indicates 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.

10.62 Evidence identified from SRs indicates that higher sugars-sweetened beverage (SSB) consumption in children aged 1 to 5 years is associated with greater odds of overweight or obesity in later childhood.

10.63 Conclusions on the consumption of plant-based drinks (soya, oat and almond) placeholder (pending COT/SACN findings).

Risks of chemical toxicity

10.64 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 that there is potential concern are described in chapter 9.

General limitations in the evidence base

10.65 Several limitations were identified in the evidence base including:

- there was no or insufficient systematic review evidence for a number of health outcomes of interest for this risk assessment, including paediatric cancers, allergy and autoimmune diseases, and bone or skeletal health
- most of the evidence identified from SRs that was specific to children aged 1 to 5 years was observational (from prospective cohort studies) or from non-randomised intervention studies and may therefore have been subject to confounding and selection bias
- many of the identified SRs had a broad search strategy that included population groups outside the age group of interest for this draft report (children aged 1 to 5 years) and it was difficult to determine whether their search strategy for the target age group was comprehensive
- risk of publication bias was not formally assessed in many of the identified SRs
- the evidence base for most 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
- few primary studies that were conducted in high income countries considered different ethnic minority groups; the majority were conducted in white participants only
- 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

- SRs without MAs reported findings from primary studies with varying levels of detail. Several SRs did not report quantitative results making it difficult to assess effect sizes and the certainty of findings
- the number of children that provided blood samples for status measures in NDNS was small and extrapolation to the wider population should be done with caution
- there was evidence of underreporting of dietary intakes in NDNS.

11 Recommendations

11.1 SACN supports the existing dietary recommendations for young children (which are presented in Table 11.1). In addition, SACN has made the following new recommendations outlined in paragraphs 11.2 to 11.13 below.

11.2 It is recommended that government:

- gives consideration to strategies that support parents who choose to breastfeed to continue for as long as they and their child want, including into the second year of life
- gives consideration to strategies to help improve uptake of advice on feeding children aged 1 to 5 years an appropriate and diverse diet
- gives consideration to strategies to help ensure the uptake of advice on micronutrient supplements in children aged 1 to 5 years, especially in at-risk groups such as children from lower socioeconomic status households and some ethnic minority groups
- continues to monitor the prevalence of both overweight and obesity and the extent of overfeeding in children aged 1 to 5 years.

The following recommendations are suitable for children who are able to consume a varied diet and are growing appropriately for their age.

Diversification of the diet

11.3 Current population dietary guidelines apply from age 5 years. It is recommended that government considers advising that these should be applied from age 2 years.

11.4 Between 1 to 2 years of age, children's diets should be diversified in relation to foods, dietary flavours and textures. Diversification should proceed incrementally, taking into account the variability between young children in developmental attainment and the need to satisfy nutritional requirements.

11.5 Vegetables should be presented on multiple occasions to children. This may require frequent presentation (as many as 8 to 10 times for each vegetable) before acceptance. Pairing vegetables with liked foods or additional flavours or energy may also increase acceptance.

11.6 It is recommended that government considers:

- developing and communicating age-appropriate portion sizes for food and drinks, including for vegetables and fruit, for children aged 1 to 5 years
- strengthening advice on limiting the consumption of high energy density foods and drinks, particularly discretionary/snack foods and sugars-sweetened beverages for children aged 1 to 5 years.

Macronutrients

11.7 Current population guidelines on average intake of free sugars apply from age 2 years. It is recommended that government considers advising these should be applied from age 1 year.

11.8 It is recommended that government consider approaches to:

- reducing excess protein intakes towards the Dietary Reference Values (DRVs) for children aged 1 to 5 years
- support children aged 1 to 5 years to consume a diet that does not exceed energy requirements

Drinks

11.9 From 1 year of age, semi-skimmed cows' milk can be given as a main drink. As currently recommended, skimmed and 1% cows' milk should not be given as a main drink until 5 years of age.

11.10 Sugars-sweetened beverages should not be given to children aged 1 to 5 years.

11.11 Advice on the consumption of plant-based drinks (soya, oat and almond) placeholder (pending COT/SACN findings)

Risks of chemical toxicity

11.12 Government should keep the risk from acrylamide and arsenic under review. Efforts to reduce the levels of inorganic arsenic in food and water, and levels of acrylamide in commercially produced and home-cooked foods should continue.

11.13 Additional compounds of potential concern identified included: excess retinol consumption, percholroate and chlorate; monochloropropanediol, its fatty acid, and glycidol; furan and methylfurans, aflatoxin; citrinin and polybrominated diphenyl ethers.

Table 11.1. Existing dietary recommendations for young children

Recommendation area	Recommendation wording	Source or reference
Breastfeeding	It is recommended that infants are breastfed exclusively for around the first 6 months of their life and, alongside appropriate complementary foods, and continue to be breastfed for at least the first year of life. Each period makes an important contribution to infant and maternal health.	World Health Organization webpage Breastfeeding (who.int) SACN Feeding in the first year of life (2018) SACN_report_on_Feeding_in_the_First_Year_of_Life.pdf (publishing.service.gov.uk)
Diversification of the diet	From around 6 months of age, children should be offered a variety of fruit and vegetables (including those with bitter flavours).	SACN Feeding in the first year of life (2018) SACN_report_on_Feeding_in_the_First_Year_of_Life.pdf (publishing.service.gov.uk)
Diversification of the diet	From around 1 year of age, children should eat the same (appropriately prepared) foods, as the rest of the family.	Start for Life https://www.nhs.uk/start4life/weaning/what-to-feed-your-baby/12-months/ NHS What to feed young children - NHS (www.nhs.uk)
Diversification of the diet	Once your child is 2, you can gradually introduce lower-fat dairy products and cut down on fat in other foods – provided your child is a good eater and growing well.	NHS What to feed young children - NHS (www.nhs.uk)

Recommendation area	Recommendation wording	Source or reference
Diversification of the diet	From about 6 months of age, gradual diversification of the diet to provide increasing amounts of whole grains, pulses, fruits and vegetables is encouraged.	SACN Carbohydrates and Health (publishing.service.gov.uk)
Carbohydrates and fibre	For children aged 2 years and older: • total carbohydrate intake should be maintained at a population average of approximately 50% of total dietary energy • the population average intake of free sugars should not exceed 5% of total dietary energy • the average population intake of dietary fibre for children aged 2 to 5 years should be approximately 15 grams per day	SACN Carbohydrates and Health report (2015) Carbohydrates and Health (publishing.service.gov.uk)
Saturated fat	It is recommended that the dietary reference value for saturated fats remains unchanged: the [population] average contribution of saturated fatty acids to [total] dietary energy be reduced to no more than about 10%. This recommendation applies to adults and children aged 5 years and older.	SACN Saturated fats and health: SACN report - GOV.UK (www.gov.uk)

Recommendation area	Recommendation wording	Source or reference
Protein	Young children need protein and iron to grow and develop. Try to give your toddler 1 or 2 portions from this group [beans, pulses, fish, eggs, meat and other proteins] each day.	NHS What to feed young children - NHS (www.nhs.uk)
Fish	Boys should eat no more than 4 portions of oily fish per week and girls no more than 2 portions of oily fish per week	NHS What to feed young children - NHS (www.nhs.uk)
Fish	Children under 16 should avoid eating shark, marlin and swordfish.	NHS Fish and shellfish - NHS (www.nhs.uk) COT statement on potential risks from methylmercury in the diet of infants aged 0 to 12 months and children aged 1 to 5 years cotstatementonmethylmercury.pdf (food.gov.uk)
Dietary fat	Advice to reduce fat intake does not apply to children under age 2 years. It applies in full from 5 years of age.	COMA Weaning and the weaning diet report (1994) Weaning_and_The_Weaning_Diet__1994_.pdf (publishing.service.gov.uk)
Fibre, including wholegrain	You can give your child wholegrain foods, such as wholemeal bread, pasta and brown rice. But it's not a good idea to only give wholegrain starchy foods to under 2s.	NHS What to feed young children - NHS (www.nhs.uk)

Recommendation area	Recommendation wording	Source or reference
	Wholegrain foods can be high in fibre and they may fill your child up before they have taken in the calories and nutrients they need. After age 2 you can gradually introduce more wholegrain foods.	
Vegetables and fruit	Try to make sure vegetables and fruit are included in every meal. Dried fruit should be given to your toddler with meals, rather than as a snack in between, as the sugar they contain can cause tooth decay.	NHS What to feed young children - NHS (www.nhs.uk)
Drinks	Feeding from a bottle is discouraged from 1 year. From the age of 6 months babies should be encouraged to drink from a free-flow cup (rather than one with a valve which requires a child to suck).	COMA Weaning and the weaning diet report (1994) Weaning_and_The_Weaning_Diet__1994_.pdf (publishing.service.gov.uk)
Drinks	Beyond 1 year, infant and follow-on formula are not needed, and toddler milks, growing-up milks, goodnight milks, comfort milks and specialised formula, for example hypoallergenic formulas for the management of cows' milk protein allergy (if not prescribed by a clinician) are also unnecessary as nutrient requirements can be met through consuming a healthy, balanced, and varied diet.	NHS Drinks and cups for babies and young children

Recommendation area	Recommendation wording	Source or reference
Drinks	Large volumes of milk (more than 600ml per day) should be discouraged as these will stop appetite for other foods.	COMA Weaning and the weaning diet report (1994) Weaning_and_The_Weaning_Diet__1994_.pdf (publishing.service.gov.uk)
Drinks	Whole cows' milk can be given as a main drink from the age of 1 year.	COMA Weaning and the weaning diet report (1994) Weaning_and_The_Weaning_Diet__1994_.pdf (publishing.service.gov.uk)
Drinks	Skimmed and 1% milk should not be given as a drink until 5 years of age. Skimmed or 1% fat milk can be used in cooking from the age of 1 year.	COMA Weaning and the weaning diet report (1994) Weaning_and_The_Weaning_Diet__1994_.pdf (publishing.service.gov.uk)
Drinks	Young children should limit consumption of 100% fruit and vegetable juices or smoothies to no more than a combined total of 150ml per day	NHS Drinks and cups for babies and young children
Drinks	Children aged 1 to 5 years should not be given rice drinks as they may contain too much arsenic.	COT Statement on potential risks from arsenic in the diet of infants aged 0 to 12 months and children aged 1 to 5 years

Recommendation area	Recommendation wording	Source or reference
		https://cot.food.gov.uk/sites/default/files/finalstatementonarsenic_0.pdf Editorial update
Drinks	Milk (including breast milk) or water should constitute the majority of drinks given.	COMA Weaning and the weaning diet report (1994) Weaning_and_The_Weaning_Diet__1994_.pdf (publishing.service.gov.uk)
Drinks	Squashes, flavoured milk and juice drinks, diet drinks and no added sugar drinks, tea and coffee and fizzy drinks should not be given to babies and young children.	NHS Drinks and cups for babies and young children - NHS (www.nhs.uk)
Micronutrients	All infants from birth to 1 year of age who are being exclusively or partially breastfed should be given a daily supplement containing 8.5 to 10µg of vitamin D (340 to 400IU per day). Infants who are fed infant formula should not be given a vitamin D supplement unless they are consuming less than 500ml (about one pint) of infant formula a day, as infant formula is fortified with vitamin D. Children aged 1 to 4 years should be given a daily supplement containing 10µg (400 IU) of vitamin D.	SACN Vitamin D and Health (2016) SACN_Vitamin_D_and_Health_report.pdf (publishing.service.gov.uk)

Recommendation area	Recommendation wording	Source or reference
Micronutrients	It's recommended that all children aged 6 months to 5 years are given vitamin supplements containing vitamins A, C and D every day. Babies who are having more than 500ml (about a pint) of infant formula a day should not be given vitamin supplements. This is because formula is fortified with vitamins A, C and D and other nutrients.	NHS Vitamins for children - NHS (www.nhs.uk)
Micronutrients	A wide range of iron-containing foods should be introduced in an age-appropriate form from around 6 months of age, alongside continued breastfeeding, at a time and in a manner to suit both the family and individual child. Healthy infants and young children do not require iron supplements unless advised by their health professional	SACN Feeding in the first year of life (2018) SACN_report_on_Feeding_in_the_First_Year_of_Life.pdf (publishing.service.gov.uk)
Oral Health	For oral health, in addition to reiterating breastfeeding recommendations: • 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	Summary guidance tables for dental teams (2021) Chapter 2: Summary guidance tables for dental teams - GOV.UK (www.gov.uk)

Recommendation area	Recommendation wording	Source or reference
	• minimise amount and frequency of consumption of sugars-containing foods and drinks • avoid sugars-containing foods and drinks at bedtime when saliva flow is reduced and buffering capacity is lost	
Salt	Children aged 1 to 3 years should eat no more than 2g salt a day (0.8g sodium) and children aged 4 to 6 years should eat no more than 3g salt a day (1.2g sodium)	SACN Salt and Health (2003) 6216 Salt and Earth Report 6th (publishing.service.gov.uk)

12 Research recommendations

[To be added post-consultation]

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