Dietary Reference Values for Energy

sacn
Scientific Advisory Committee on Nutrition

2011

Scientific Advisory Committee on Nutrition

Dietary Reference Values for Energy

Scientific Advisory Committee on Nutrition

2011

London: TSO

Any enquiries regarding this publication should be sent to the SACN Secretariat: www.sacn.gov.uk/contact_us/index.html

This document is available from the SACN website at: www.sacn.gov.uk

ISBN: 9780108511370

Printed in the UK by The Stationery Office Limited

ID 2485597 04/12 15707 19585

Printed on paper containing 75% recycled fibre content minimum.

Preface

In 1991, the Committee on the Medical Aspects of Food Policy (COMA) provided estimates of energy requirements for the UK population in their report *Dietary Reference Values for Food Energy and Nutrients for the United Kingdom*. The dietary reference values (DRVs) for energy were based on estimating the total energy expenditure (TEE) for groups of people. TEE provides a measure of the energy requirement at energy balance i.e. when energy intake matches energy expenditure. In this way, an energy requirement can be predicted as the rate of TEE plus any additional needs for growth, pregnancy and lactation.

Since the publication of the report in 1991, the methodology to measure TEE – the doubly labelled water (DLW) method – has advanced and as a result, the evidence base on TEE in a wide variety of population groups has expanded considerably. In addition, the Food and Agriculture Organization of the United Nations, World Health Organization, and United Nations University (FAO/WHO/UNU) and Institute of Medicine (IoM) have updated their recommendations on energy requirements. With the high levels of overweight and obesity currently seen in the UK and the wealth of new data now available, it was considered timely for the Scientific Advisory Committee on Nutrition (SACN) to review recommendations for the UK population.

The present report details the evidence and approaches SACN have considered in order to update the DRVs for energy. The DRVs for energy are based on the estimated average requirements (EARs) of infants, children, adolescents and adults. After much deliberation, SACN agreed that the *factorial approach* was the most appropriate way to derive energy requirements, whereby TEE is expressed as a multiple of the basal metabolic rate (BMR) and the physical activity level (PAL). Hence, TEE or EAR is equal to BMR x PAL. The PAL is best estimated from measures of DLW, which give higher values than previously estimated by COMA.

SACN noted that in populations like the UK, with a high and increasing proportion of overweight and obese individuals, if energy requirements are estimated at current levels of energy expenditure and body weights, many groups in the population would continue to carry excess weight. This is not desirable since excess body weight is associated with long-term poor health and increased mortality. To address this issue SACN chose a *prescriptive* approach to estimating energy reference values. That is, suitable reference body weight ranges consistent with long-term good health were used to calculate energy reference values. Thus, BMR values were predicted using healthy reference body weights. For the purposes of calculation, this equates to the 50th centile of UK-WHO growth standards for infants and pre-school children, the 50th centile of UK 1990 reference for school-aged children and for adults at a Body Mass Index (BMI) of 22.5 kg/m^2 at the current height of the UK adult population. Using this approach, if overweight groups consume the amount of energy recommended for healthy weight groups, they are likely to lose weight, whereas underweight sections of the population should gain weight towards the healthy body weight range. This approach represents a significant departure from the method used by COMA.

SACN has derived new energy reference values. For most population groups, except for infants and young children, the values have increased. This change reflects the more accurate methods used to assess energy expenditure; the evidence base available to COMA was more limited and as a result energy requirements were underestimated for some age groups. It is important to note that DRVs should be used to assess the energy requirements for large groups of people and populations, but should not be applied to individuals due to the large variation in physical activity and energy expenditure observed between people. Despite SACN's best efforts to base their recommendations on the most up to date evidence, it should be noted that there is less DLW data available for infants, younger adults (18-30 years) and those aged 80 years. The Committee hopes that this will be addressed in the future.

I would like to thank those who provided comments on the draft version of this report during the public consultation. The process assisted the Committee in refining its approach to setting energy requirements for the UK. This has been a challenging and large undertaking for SACN and I would like to thank the Energy Requirements Working Group and the Secretariat for their great commitment in producing this report. Particular thanks to Professor Joe Millward for his substantial contribution to this report. I would also like to extend my gratitude to the principal investigators of the Beltsville and OPEN studies for allowing the Committee to use their data.

Professor Alan Jackson
Chair of the Energy Requirements Working Group

Table of contents

1 Summary

Introduction

S1. The Dietary Reference Values (DRVs) for food energy provide a best estimate of the food energy needs of the UK population and its subgroups and present criteria against which to judge the adequacy of their food energy intakes (Department of Health [DH], 1991). The DRVs for food energy are defined as the Estimated Average Requirement (EAR). In adults, the EAR for energy has previously been set at the level of energy intake required to maintain weight i.e. an energy intake which matches energy expenditure. During infancy and childhood, the requirement also has to meet the needs for healthy growth and development, while during pregnancy and lactation the requirement must meet the needs for development of a healthy baby and supporting adequate lactation. Any sustained imbalance between energy intake and expenditure will lead to progressive gain or loss in weight.

S2. The National Diet and Nutrition Survey (NDNS) series shows average reported energy intakes to be consistently below the level indicated by the EAR for food energy defined in the 1991 report of the Committee on Medical Aspects of Food and Nutrition Policy (COMA) (DH, 1991). In reality, average habitual energy intakes in the United Kingdom (UK) are more likely to exceed energy needs, as evidenced by the increasing number of people classified as overweight or obese. Under-reporting of food intake may explain this paradox as it is known to largely account for a commonly reported discrepancy between measured total energy expenditure (TEE) and the apparent low energy intakes reported in NDNS and other dietary surveys.

S3. These observations, the expanding evidence base on TEE in a wide variety of population groups, together with the publication of the Food and Agriculture Organization of the United Nations, World Health Organization, and United Nations University (FAO/WHO/UNU) updated recommendations for energy intake from the expert consultation on Human Energy Requirements (FAO, 2004), led the Food Standards Agency and the Department of Health to request the Scientific Advisory Committee on Nutrition (SACN) to re-evaluate of the DRVs for food energy. Subsequent to this, the US Institute of Medicine (IoM) published revised Dietary Reference Intakes (DRIs) for energy (IoM, 2005).

Terms of reference

S4. The Terms of Reference for the Energy Requirements Working Group were to:

- Review and agree on the interpretation of the methods, definitions and assumptions used by COMA (DH, 1991) and FAO/WHO/UNU expert consultation on Human Energy Requirements (FAO, 2004) to agree energy requirements.
- Agree a framework by which to arrive at energy requirements for the UK population and its subgroups.

- Agree population-based Dietary Reference Values for energy, and provide recommendations taking into account age, body size, levels of activity, gender and physiological state (i.e. growth, pregnancy and lactation).

- Consider the implications of these recommendations on the requirements for other nutrients[1].

S5. In addressing these Terms of Reference, the report considers: components of and factors affecting energy expenditure; the measurement and estimation of energy expenditure; energy expenditure in representative populations of children and adults; and energy requirements for the UK population in health.

Background

Components of energy expenditure

S6. The TEE of an individual can be divided into a number of discrete components that can be determined separately. These are basal metabolic rate (BMR), the energy expended in physical activity, and other components such as the thermic effect of food (TEF) and growth.

Basal metabolic rate

S7. Basal metabolic rate (BMR) is a standardised measure of an individual's metabolism in a basal state: i.e. while awake and resting after all food has been digested and absorbed, and at thermal neutrality. For most individuals, BMR is the largest component of energy expenditure and requirements, ranging from 40-70% depending on age and lifestyle. BMR can be estimated from body size, age and gender. A variety of BMR prediction equations have been described in the literature, with the Schofield equations being primarily used in the COMA (DH, 1991) and FAO/WHO/UNU (FAO, 2004) reports.

Physical activity

S8. Physical activity includes a wide range of behaviour and encompasses sitting, standing, walking, and planned and structured exercise which may have the objective of maintaining or improving physical fitness or health. Physical activity-related energy expenditure (PAEE) is quantitatively the most variable component of TEE usually accounting for 25-50% of energy expenditure, up to a maximum of 75% in some unusual circumstances.

The physical activity level (PAL)

S9. TEE can be expressed as a multiple of BMR, the physical activity level (PAL). Hence, TEE or EAR is equal to BMR x PAL. PAL is theoretically independent of those factors influencing BMR (weight, age and gender), at least as a first approximation, and consequently, for any PAL value, TEE can be predicted for any group from estimates of the BMR. During growth, pregnancy and lactation the energy cost of tissue deposition also needs to be taken into account.

1 In most cases, energy intake has increased and therefore it was felt that there was no need to undertake this.

S10. PAL values are best estimated from direct measures of 24 hour TEE and BMR. Such measurements in free-living populations have indicated that PAL can range from <1.3 in immobile subjects to between 3-4.7 for limited periods of time in, e.g. soldiers on field exercises or elite endurance athletes. Within the general population, however, the overall range of PAL values for individuals in energy balance, leading sustainable lifestyles, is between 1.38 for the most sedentary to 2.5 for the most active.

S11. In previous reports, including COMA (DH, 1991), the duration and energy cost of individual activities (as physical activity ratio (PAR) values) has been summed to provide factorial estimates of PAL. The factorial estimation of PAL assumes that overall energy expenditure within the general population can be predicted using information derived from activity diaries or lifestyle questionnaires. However, there is little evidence that this can be done with sufficient accuracy for two main reasons:

- For most lists of activities used to identify lifestyle categories, individual activities are only defined in general qualitative terms and consequently there can be large inter-individual variations in the energy expended for each activity.
- Factorial estimates take no account of variations in spontaneous physical activity (SPA), a term used to describe all body movements associated with activities of daily living, change of posture and 'fidgeting'. SPA accounts for a between-individual variation in energy expenditure of ±15%. The potential for variable SPA throughout the range of defined activities adds to the uncertainty that their energy costs can be predicted.

S12. In this report, factorial predictions of PAL values for specific lifestyle categories have not been attempted due to the likely error in estimating actual TEE from listed PAR values of activities and the phenomenon of behavioural phenotypes which exhibit very different rates of energy expenditure. Instead, values for PAL for children, adolescents and adults have been identified from an analysis of the available TEE literature judged to be appropriate for the UK population.

Definition of energy requirements

S13. The energy requirement and associated descriptive terminology must be defined with particular care in the context of a population which includes many individuals not defined as healthy because they are overweight or exhibit habitually low levels of physical activity. In 1991, COMA defined requirements in general terms only i.e. intakes of nutrients which were likely to "meet the needs" of some or all within population groups. COMA did not offer a specific definition of the energy requirement in relation to specific body weights within the healthy range or prescribed levels of energy expenditure. COMA set EAR values for population groups calculated for a wide range of physical activity levels and adult body weights which in practice would maintain the status quo in terms of existing body weights. In populations like the UK, with a high and increasing proportion of overweight and obese individuals, application of such energy reference values would therefore maintain body weights in excess of healthy reference body weights. In contrast, the FAO/WHO/UNU reports of 1985 and 2004 used a 'normative' or 'prescriptive' approach[2] to calculate energy requirements. That is, suitable reference body weight ranges consistent with long-term good health were used to calculate energy reference values. Adoption of these prescriptive values by groups with body weights below or above such ranges would tend to mediate weight change towards the healthier, more desirable body weight range as opposed to maintaining body weights which exceed healthy reference values.

S14. In this report, the requirements for energy for all population groups, with the exception of pregnant women, have been set at the level of energy intake required to maintain a healthy body weight in otherwise healthy people at existing levels of physical activity. Allowances are made for any additional physiological needs (i.e. growth, pregnancy and lactation). Thus, the report recognises the increasing prevalence of overweight and obesity in the current UK population, and has adopted the prescriptive terminology and principle (i.e. relating to a desirable standard as distinct from *status quo*) for body weight, with physical activity set at best estimates of existing levels. Consequently, energy reference values defined in this report are derived for infants, children and adults in relation to body weights which, on the basis of current evidence, are likely to be consistent with long-term good health. This means that for people who are overweight or obese, energy intakes at the reference levels should enable the transition towards the healthy body weight range (i.e. Body Mass Index (BMI) 18.5-24.9kg/m^2 for adults). For pregnant women who are overweight or obese a precautionary approach has been adopted and it is recommended that energy requirements for this group are estimated based on actual preconceptional body weights.

S15. As with previous reports, the importance of adequate physical activity is recognised and this report includes advice on desirable physical activity consistent with long-term health. The likely impact of such advice on energy requirement values is also described.

Variability

S16. Measurements of energy needs that are used to predict DRVs for a particular population group will, for a number of reasons, exhibit variability.

S17. For most nutrients the population reference value is identified as the Reference Nutrient Intake (RNI)[3]. However, for dietary energy the DRV is defined differently i.e. it is equal to the average reference value (EAR). The RNI for dietary energy is not used because it represents an excess energy intake for the majority of the population. Energy intakes that consistently exceed requirements lead to weight gain and obesity in the long term. An intake equal to the average reference value for a population group on the other hand is, in theory, associated with similar probabilities of excessive and insufficient energy intakes for any individual within the population.

2 The terms 'normative' and 'prescriptive' can be used interchangeably.

3 The reference nutrient intake for a nutrient is the amount of the nutrient that is enough, or more than enough, for about 97% of people in a group.

S18. In the UK and similar countries, the population is characterised by sedentary lifestyles at all ages, and thus rates of energy expenditure and intakes that maintain energy balance at these levels of energy expenditure are unlikely to be normally distributed. Measurements of energy expenditure within adult populations indicate that the overall distribution is skewed towards sedentary behaviour. For the high proportion of individuals who are relatively inactive, energy expenditure and intakes that maintain energy balance will cluster at the lower end of the range. A small proportion will be more active, with a few individuals exhibiting the highest rates of expenditure that are sustainable. Statistical considerations, therefore, dictate that the appropriate descriptor of the midpoint of the distribution is the median, which is likely to be somewhat lower than the mean, thus the median is used in this report.

S19. Evidence that energy expenditure varies between individuals with similar lifestyles over a wider range than would be expected (at least in adults) presents a second difficulty in estimating energy reference values. This is particularly important since in the past, attempts have been made to define energy reference values in terms of specific physical activity levels for specific lifestyle population groups. Consequently, with the probable exception of those at the extremes of the activity range, lifestyle predictions of energy expenditure and resultant energy reference values cannot be made with the certainty often assumed by users of previous reports such as COMA (DH, 1991).

Approaches used to set energy reference values

S20. Measurement or prediction of TEE provides a physiological measure of the energy requirement at energy balance. This is because in most circumstances energy intake must match energy expenditure to achieve energy balance. Thus, the energy requirement can be predicted specifically as the rate of TEE plus any additional needs for growth, pregnancy and lactation.

Measurement of total energy expenditure

S21. The *doubly labelled water* (DLW) method is the most accurate practical means of measuring TEE in free-living individuals over a period of several days to several weeks. In this method, the TEE of individuals is computed from estimates of CO_2 production, which are calculated from the loss of the isotopes ^{18}O and ^{2}H from the body over time following administration of a known dose.

Utilising total energy expenditure measurements to derive energy requirements

S22. In recent years, EAR values for populations have been estimated from DLW-derived measures of TEE in reference populations together with estimates of deposited energy. Two potential analytical approaches can be employed.

Predicting TEE with regression equations

S23. TEE can be modelled against different anthropometric characteristics of the reference population (such as age and body weights) using regression equations and the regression equations can then be used to predict TEE and resulting EAR values for any population group based on their anthropometric variables. A major limitation to this approach has been the inability of TEE prediction models to account for variation in Physical Activity Energy Expenditure (PAEE), both between-individual and between-group), an important source of variation in TEE, in a transparent way.

S24. Previous reports which have used regression models of TEE to predict energy requirements, the FAO/WHO/UNU report (FAO, 2004)[4] and the US report on Dietary Reference Intake values for energy (IoM, 2005) have made special provision for the likely variation in PAEE within the population group described by the regression.

Predicting TEE from BMR and measured PAL values – the factorial approach

S25. The second analytical approach to the derivation of energy requirements employs a factorial approach[5] based on the assumption that TEE (or EAR) is equal to BMR x PAL. This involves the following steps:

- TEE values measured in a reference population are divided by measured or estimated BMR values from that population to extract PAL values. This means that the reference populations studied by DLW are described primarily by PAL values.
- For the population of interest, BMR values are then estimated from BMR prediction equations using relevant anthropometric data for the population.
- The PAL values derived from the reference population can then be used to estimate TEE and EAR values for the population of interest based on the latter's estimated BMR values.

S26. When utilising this factorial approach, suitable PAL values appropriate for specific groups and populations should be identified from measurements of DLW-derived TEE obtained from large population studies which are representative of the current UK population and which can be assumed to exhibit similar activity patterns. Once suitable PAL values have been established, their distribution can be evaluated for the population as a whole in order to identify the medians and centile ranges. This approach allows energy reference values to be framed against such distributions, in effect substituting PAL distributions for PAL values defined in terms of lifestyle. Thus energy reference values can be defined as a population EAR (i.e. from the median PAL value) with additional values appropriate to those who are more or less active than the average (i.e. from the 25th and 75th centiles).

4 Regression equations were used to derive energy requirements for infants, children and adolescents. The energy requirements of adults were calculated from factorial estimates of habitual TEE.

5 This approach has also been referred to as the BMR multiple approach and the PAL model.

Summary of approach used by SACN to determine revised energy reference values

S27. The SACN Framework for the Evaluation of Evidence has been used as the basis to identify and assess published evidence of TEE from which to guide derivation of energy reference values. Only studies using the DLW method to measure TEE were considered. No large-scale population studies of any age group have been conducted to determine TEE in the UK population, and TEE data from the NDNS series are as yet insufficient for the determination of energy reference values. Other data sources (reference populations) have therefore been used to derive the revised EAR values. Priority was given to studies in well-characterised populations (e.g. by age, gender, weight, height or BMI) and where BMR had been measured directly rather than estimated from prediction equations. The majority of studies identified were cross-sectional studies in healthy human populations of infants, children and adults. Those identified as being the most likely to be representative of the UK population were as follows:

- For infants, a longitudinal study of healthy American infants comprising similar numbers of breast fed and breast milk substitute-fed infants (n=76 individuals).
- For children and adolescents, a compilation of all identified mean study values for specific ages for boys and girls (n=170) representing a total of approximately 3500 individual measurements.
- For adults, two large data sets of individual TEE values (n=929) were obtained from the USA, the OPEN and Beltsville studies.
- For older adults, no specific representative data set has been identified.

S28. The limitations of these data sets (reference populations) for deriving estimated EARs for the UK population include the small numbers of participants in the infant and at some ages in the child cohorts and the lack of younger adults aged 18-30 years and older adults aged >80 years in the larger adult data set.

S29. For adults aged 19-65 years and children aged between 3-18 years, the different approaches to setting EARs, namely regression modelling and the factorial approach, were examined. Regression modelling was explored, but the approach was not pursued because the inability of TEE prediction models to account for variation in PAEE in a transparent way was considered a major limitation. A factorial model was therefore adopted in which TEE is predicted from BMR x PAL, following the steps outlined in paragraph S25.

Prescriptive energy reference values for healthy body weights

S30. The increasing prevalence of overweight and obesity at all ages in the population raises a clear difficulty in the definition of energy reference values for population groups. Such values, calculated to match TEE, will, for many of the population, maintain overweight and will therefore not be consistent with long-term good health. Even for subjects exhibiting desirable physical activity, excess body weight may be associated with increased risk of mortality. Given this report's objective of defining prescriptive reference values, healthy reference body weights have been identified and applied.

Factorial approach to setting EARs

S31. As described above (paragraphs S11 and S12), in contrast to the approach taken by COMA in 1991 (DH, 1991), the summation of the duration and energy cost of individual activities to derive estimates of PAL has not be used here. Instead, the distribution of PAL values within the reference population has been used to indicate a population average value (median) for PAL as well as the extent to which it is lower (25th centile) or higher (75th centile) for less or more active population groups. The PAL values for adults, as median, 25th and 75th centiles, are those calculated directly from individual TEE values reported in the OPEN and Beltsville data sets. For children over one year of age, the PAL values are those calculated directly from a data set of published DLW studies which were aggregated on the basis of study mean values. Estimates of the likely increase in PAL associated with varying increases in activity level are also given. Due to a lack of substantial new data, the reference values stated in the FAO/WHO/UNU report (FAO, 2004) were used for infants. Pregnant and lactating women were considered separately.

S32. In this report, BMR for children and adults is estimated using the Henry equations applying healthy body weights as indicated by the 50th centile of the UK-WHO Growth Standards (ages 1–4 years), the 50th centile of the UK 1990 reference for children and adolescents aged >4 years, and at weights equivalent to a BMI of 22.5kg/m^2 at the appropriate height of the adult population group (see paragraph S36).

S33. COMA reported EAR values for a range of body weights and PAL values for adolescents and adults (DH, 1991). However, the most widely cited values from that report are aggregated EAR values for these groups which were based on low PAL values thought to be in keeping with the sedentary lifestyle of the UK population: i.e. 1.56 for boys, 1.48 for girls and 1.4 for adults. An analysis of the range and distribution of PAL values shows that COMA is likely to have underestimated the PAL values of general sedentary populations because of an under-appreciation of the influence of routine activities of daily living on energy expenditure. Values used by COMA are lower than those observed for 90% of the subjects in the reference adolescent and adult populations examined for this report. Thus, median PAL values identified for adolescents and adults in this new report are 1.75 and 1.63 respectively, with 1.63 representing the median PAL value of a reference adult population in which, like the UK, approximately 60% are overweight or obese.

Energy reference values for infants, children and adolescents

Energy cost of growth

S34. TEE measured using the DLW method includes the energy expended in tissue synthesis. Therefore, only the cost of energy deposited in newly synthesised tissues should be added when calculating the energy reference values for infants, children and adolescents.

Infants aged 0 – 12 months

S35. Following the approach of the FAO/WHO/UNU Expert Consultation (FAO, 2004), the energy reference values for infants are calculated from the energy deposited in new tissue plus TEE. TEE was predicted from a simple equation expressing TEE as a function of weight which was derived from a longitudinal study in which TEE was measured by the DLW method in healthy, well-nourished, non-stunted infants, born at full term with adequate birth weight, and growing along the trajectory of the UK-WHO Growth Standard (Royal College of Paediatrics and Child Health [RCPCH], 2011). Costs of tissue deposition were calculated from an analysis of the body composition of a population of healthy US infants during normal growth. They were then applied to weight increments observed in the WHO Multicentre Growth Reference Study. See Tables 5 and 14 for revised EAR values of infants 1-12 months (pages 51 and 83).

Children and adolescents aged 1 – 18 years

S36. The energy reference values for boys and girls aged 1-18 years were calculated as TEE plus deposited energy costs using a factorial model BMR x PAL. The energy value of tissue deposited was assumed to be equivalent to a 1% increase in PAL. The population EAR values are calculated at median PAL values[6] for best estimates of healthy body weights i.e. the 50th centiles of the UK-WHO Growth Standards (ages 1–4 years) and the UK 1990 reference for children and adolescents for children aged over four years of age. These reference weights are about 15% lower than current UK weights and thus for those children who are overweight and for any underweight children, energy intakes at these levels will be associated with weight change.

S37. EAR values have also been calculated for less active or more active children by the 25th and 75th centile PAL values. See Tables 8 and 15 for revised EAR values for children aged 1–18 years old (pages 56 and 84).

Energy reference values for adults

S38. Energy reference values for adults were derived by the factorial calculation of TEE from BMR x PAL. BMR values are calculated using the Henry equations at weights equivalent to a BMI of 22.5kg/m^2, which can be considered to be a healthy body

6 where median PAL =1.4 for children aged 1-<3 years, 1.58 for children aged 3-<10 years, and 1.75 for children aged 10–18 years.

weight[7], and at the relevant height of the population. For illustration, current mean heights are used for England and Scotland; representative data for Wales and Northern Ireland were not available. PAL values were identified from an analysis of suitable DLW measures of TEE.

Identifying PAL values

S39. The approach taken to determine PAL values was to identify a data set of DLW measures of TEE which could serve as a reference distribution of TEE and PAL values from which energy reference values for the UK adult population could be estimated.

S40. An initial survey of all published and other available DLW measures of TEE in healthy adults identified published studies which could be utilised in terms of study means. Two sets of individual data points were also considered:

- a DLW data set of individual values drawn from UK national dietary surveys (the NDNS (n=156) and Low Income Diet and Nutrition Survey (n=36)).
- the DLW data set (n=767) assembled for the US DRI report which includes most of the UK studies published up to the writing of that report.

S41. With the exception of the NDNS data sets, subjects were not recruited to these studies explicitly as a representative sample of the UK or any other adult population; NDNS randomly selects participants while recognising that there may be some recruitment bias. In contrast, the DLW data assembled for the DRI report "were not obtained in randomly selected individuals ... and do not constitute a representative sample of the United States and Canada." While the NDNS data sets are most appropriate, these are currently too small to serve as a reference.

S42. Since publication of the DRI report in 2005, two large population-based studies from the United States, the OPEN study (n=451) and the Beltsville study (n=478) have been published, in which TEE was measured using DLW. The combined OPEN and Beltsville cohorts contained similar number of men (48%) and women (52%), with levels of overweight and obesity very similar to current levels reported in the Health Surveys for England and Scotland. Mean BMI values were 27.0 kg/m^2 (F), 27.5 kg/m^2 (M) compared with current UK values of 27 kg/m^2 (F and M); 39% were classified as overweight and 25% obese, compared with 38% overweight and 23% obese in England. Demographic characteristics were also similar to the current UK population, although some ethnic differences may exist. The combined OPEN/Beltsville data set has been used as the reference population in this report from which a median PAL value has been identified. Revised EARs for the UK adult population have then been derived employing the factorial approach of BMR x PAL = TEE, in which PAL is that derived from the reference population and BMR is calculated from the Henry equations at healthy body weights equivalent to a BMI of 22.5kg/m^2 at the height of the population group (see paragraph S30 above).

7 A body weight equivalent to a BMI of 22.5kg/m^2 is at the lower end of the body weight range (BMI 22.5-25kg/m^2) associated with a minimum risk of mortality (based on a collaborative analysis of the influence of BMI on all-cause mortality in 57 prospective studies, n=900,000) and can be considered to represent a healthy body weight.

S43. The distribution of PAL values within the combined data set (n=929 individual measures) was 1.01 to 2.61. In healthy, mobile individuals, the overall range of PAL values is normally between 1.38 and 2.5; PAL values can fall as low as 1.27 and may exceed 2.5, but such low and high values are not thought to be sustainable. The combined data set was therefore trimmed for PAL values $\leq$1.27 or $\geq$2.5. After trimming, the median PAL value used to predict the population EAR was 1.63. The 25th and 75th centile PAL values used to estimate energy reference values for groups judged to be less or more active than average were 1.49 and 1.78 respectively. The report also provides estimates of the probable additional energy needs, defined as increases in PAL, associated with changes in habitual activity involving specific types of activity such as increased walking, running and participation in sport or high-level training regimes.

S44. The derivation of EAR values using PAL values from a predominately overweight and obese population was carefully considered given the report's objective of identifying prescriptive EAR values consistent with long-term good health i.e. values suitable for weight maintenance at a healthy body weight at existing levels of physical activity. For those subjects within the normal body weight range, the median PAL (1.61) was not significantly different from that of the overweight and obese men or women. Furthermore, investigation of the influence of BMI on PAL values within the combined data set by regression analysis indicated that PAL did not significantly vary with BMI. This indicates that the level of energy expenditure as indicated by the median PAL for the cohort (i.e. 1.63) is not a specific characteristic of overweight or obesity and its use in deriving prescriptive EAR values consistent with healthy body weights is justifiable.

S45. The revised population EAR values for all adults, calculated using a PAL value of 1.63 and BMR values calculated at weights equivalent to a BMI of 22.5kg/m^2 at current mean heights for age, are 10.9 MJ/d (2605kcal/d) for men and 8.7 MJ/d (2079kcal/d) for women. See Tables 11, 12 and 16 for revised EAR values for adults (pages 61, 61 and 85).

Energy reference values for older adults

S46. Age-related changes in lifestyle and activity are very variable and many older people exhibit relatively high levels of activity. For healthy, mobile older adults energy requirements are unlikely to differ substantially from younger adults and reference values can be described in the same way. However, for those individuals with much reduced mobility it can be assumed that PAL values are likely to be lower. For these individuals and for older people who are not in good general health, energy requirements can be based on the less active, 25th centile PAL value of 1.49, recognising that for some groups of older people with specific diseases or disabilities or for patient groups who are bed-bound or wheelchair bound, the PAL value may be consistently lower than this.

Effects of additional physical activity on energy requirements

S47. Although the prediction of PAL values associated with a specific lifestyle cannot be made with any certainty, predictions of the likely additional energy cost of well-defined specific activities can be made with reasonable confidence. Examples are given for increments in PAL associated with increased activities ranging from 0.15 for 30 minutes of moderate intensity activity on five or more days of the week, to 0.6 for an intense aerobic exercise programme associated with training for competitive sport daily. These values have been derived primarily from studies on adults but there is no reason to believe that they will be substantially different for children and adolescents.

Reference energy values during pregnancy and lactation

S48. The energy requirements for pregnancy and lactation are calculated as increments to be added to the mother's EAR. These are based on singleton pregnancies reaching term.

S49. Ideally, women should begin pregnancy at a healthy weight (BMI 18.5-24.9 kg/m^2) and the EARs for non-pregnant women identified in this report are set at amounts consistent with maintaining a BMI of 22.5kg/m^2. Women who are underweight or overweight at the beginning of pregnancy are at risk of poor maternal and fetal outcomes. Women who are underweight benefit from greater weight gain during pregnancy. For women who are overweight and obese, the consequences of weight change during pregnancy are not completely understood. Given this uncertainty, a precautionary approach has been adopted and weight loss during pregnancy is not advised. The EARs for pregnancy and lactation defined in this report are therefore estimates of the incremental energy intakes likely to be associated with healthy outcomes for mother and child, for women consuming energy intakes which match energy expenditure at the commencement of pregnancy. That is, for women who are overweight the incremental energy intakes should be added to EAR values calculated at preconceptional body weights, rather than at healthy body weights for non-pregnant women.

Energy reference values for pregnancy

S50. Energy reference values for pregnancy estimated by the factorial method in previous reports such as the FAO/WHO/UNU (FAO, 2004) and US DRI (IoM, 2005) reports exceed energy intakes observed in well-nourished populations with average birth weight in the healthy range. Adherence to these energy reference values may lead to inappropriate maternal fat gain, some of which may remain after lactation. Consequently, it was not considered necessary to amend the increment of 0.8 MJ/day (191kcal/day) in the last trimester previously recommended by COMA. Women entering pregnancy who are overweight may not require this increment but current data are insufficient to make a recommendation regarding this group.

Energy reference values for lactation

S51. Factorial calculation suggests that women with a healthy pre-pregnancy weight (BMI 18.5-24.9 kg/m^2) who exclusively breastfeed their infants throughout the first six months require an increment of 2.1 MJ/day (502kcal/day) above the pre-pregnant EAR. However, the factor of 0.8 applied to adjust for efficiency of conversion of maternal energy intake to milk is insecure and likely to overestimate the true synthetic cost. This could explain why the factorial estimate is appreciably greater than that measured in DLW experiments. It is recommended that the US Energy DRIs (IoM, 2005) (based on DLW measurements) of 1.38 MJ /day (330 kcal/day) in the first six months of lactation are applied. Thereafter, the energy intake required to support breastfeeding will be modified by maternal body composition and the breast milk intake of the infant.

Comparisons with reference values for energy in the COMA 1991 report

S52. The energy reference values detailed in this report derive from a methodology which differs to a greater or lesser extent from COMA, the US DRI report, and the FAO/WHO/UNU. As a result of these methodological differences, the revised EAR values differ from COMA in a non-uniform way. For pre-adolescent children (aged 3 months to 10 years) the revised EAR values are lower by between 2-22%. However, the revised EAR values are generally higher for adolescent boys (by 6-9%) and girls (by 15-18%). For adults, including older adults in whom general health and mobility are maintained, the new population EAR values for all men and women are higher by 3% for men and by 7-9% for women due to the combined use of higher PAL values with lower BMI and BMR values. See Tables 38 and 39 for details.

S53. Although for some population groups the revised population EAR values are higher than previous estimates, this should not be interpreted to mean that these groups have increased their activity and therefore need to eat more, but rather that the new values represent a closer approximation of energy needs at current activity levels, estimated using updated methodology.

S54. The revised EARs for dietary energy can be found in Tables 5 and 14 for infants, Tables 8 and 15 for children aged 1- 18 years old, and Tables 11, 12 and 16 for adults.

S55. Gaps in the evidence base and consequent recommendations for future research are detailed in paragraphs 203-206.

2 Introduction

Background

1. Dietary Reference Values (DRVs) for food energy describe the requirements for food energy of population groups, and provide criteria against which to judge the adequacy of the food energy intake for the UK population and its subgroups (Department of Health [DH], 1991). These DRVs apply to groups of healthy people and are not appropriate for the definition of requirements for individuals. The DRVs support the maintenance of health in a population and are derived with the assumption that the requirements for all other nutrients are met.

2. The DRVs for food energy are defined as the Estimated Average Requirement (EAR)[8]. They are used for various purposes which include informing the provision of food in clinical and institutional settings. In adults, the EAR for energy has been set at the level of energy intake required to maintain body weight i.e. an energy intake which matches energy expenditure. During infancy and childhood, the requirement also has to meet the needs for healthy growth and development, while during pregnancy and lactation the requirement must meet the needs for carrying a healthy baby and supporting adequate lactation. Any sustained imbalance between energy intake and expenditure will lead to progressive gain or loss in body weight.

3. The National Diet and Nutrition Survey (NDNS)[9] series[10] shows average reported energy intakes to be consistently below the level indicated by the EAR for food energy defined in the 1991 report of the Committee on Medical Aspects of Food and Nutrition Policy (COMA) (DH, 1991). However, these, and other surveys of the UK population (The NHS Information Centre [NHS IC], 2010; Reilly *et al.*, 2009), also show that the mean Body Mass Index (BMI) and number of people classified as overweight or obese[11] is increasing. The latter data indicate that average habitual energy intake does, in fact, exceed energy needs.

4. One explanation for this apparent paradox is that reported intakes of food energy assessed in the NDNS may be lower than intakes actually consumed (Stephen *et al*, 2007; Poppitt *et al*., 1998). Under-reporting of food intake, which is significant and particularly pronounced in people who are overweight and obese (Rennie *et al.*, 2007; Westerterp & Goris., 2002), largely accounts for the apparent low energy intakes observed in NDNS and other dietary surveys.

8 The Estimated Average Requirement (EAR) is an estimate of the average requirement for energy or a nutrient and assumes normal distribution of variability.

9 The National Diet and Nutrition Survey (NDNS) is a UK survey of the food consumption, nutrient intakes and nutritional status of people aged 1.5 years and older living in private households. The NDNS rolling programme is currently collecting data from 2008-2012. Previously it was a series of discrete cross-sectional studies.

10 See publications by Finch et al., 1998; Gregory et al., 1990; Gregory *et al.*, 1995; Gregory et al., 2000; Henderson *et al.*, 2002; Henderson *et al.*, 2003a; Henderson et al., 2003b; Hoare *et al.*, 2004; Nelson *et al.*, 2007; Ruston *et al.*, 2004; .

11 Body Mass Index (BMI) (kg/m^2) is often used as a convenient measure of adiposity. In adults, cut-off points for underweight, overweight and obesity are defined as BMI values of <18.5 kg/m^2, >25 kg/m^2 and >30 kg/m^2 respectively.

5. Another possible explanation for the apparent paradox is that the EAR for food energy which has been used to evaluate measured energy intakes in the UK is, in fact, set at too high a value.

6. The EARs for food energy published by COMA (DH, 1991) were based on limited available evidence at that time. More recent observations on the energy expenditure in a wide variety of population groups together with the publication of the Food and Agriculture Organization of the United Nations, World Health Organization, and United Nations University (FAO/WHO/UNU) updated recommendations for energy intake from the expert consultation on Human Energy Requirements (FAO, 2004) indicate that the EAR values proposed by COMA are unlikely to be too high, at least for some groups.

7. Even relatively sedentary populations are now thought to exhibit higher rates of energy expenditure than previously estimated (Black, 1996; Black *et al.*, 1996). Consequently, the revised energy reference values published by FAO/WHO/UNU in 2004 differ from those set by COMA in 1991, being generally higher. For these reasons, it was timely to review the evidence and the Food Standards Agency (FSA) and the Department of Health therefore requested the Scientific Advisory Committee on Nutrition (SACN) to re-evaluate the DRVs for food energy. Subsequent to this, the US Institute of Medicine (IoM) published revised Dietary Reference Intakes (DRIs) for energy (IoM, 2005).

Terms of reference

8. The Terms of Reference for the Energy Requirements Working Group were to:

- Review and agree on the interpretation of the methods, definitions and assumptions used by COMA (DH, 1991) and FAO/WHO/UNU expert consultation on Human Energy Requirements (FAO, 2004) to agree energy requirements.
- Agree a framework by which to arrive at energy requirements for the UK population and its subgroups.
- Agree population-based Dietary Reference Values for energy, and provide recommendations taking into account age, body size, levels of activity, gender and physiological state (i.e. growth, pregnancy and lactation).
- Consider the implications of these recommendations on the requirements for other nutrients[12].

Report content

9. The report considers:

- energy available from food and drink;
- components of and factors affecting total energy expenditure (TEE);
- the measurement and prediction of TEE;

12 In most cases, energy intake has increased and therefore it was felt that there was no need to undertake this.

- TEE in representative populations of children and adults; and
- energy requirements for the UK population in health.

Physical activity and energy balance, and the nature and extent of obesity in the UK population, are also covered.

Methodology

10. The working procedures for the preparation and finalisation of the report are described in Appendix 1.

Units of energy

11. The unit of energy in the International System of Units (SI) is the joule (J) and is the energy expended when an object is moved one metre by a force of one newton in the direction in which the force is applied. A newton is the SI unit of force and one newton will accelerate a mass of one kilogram at the rate of one metre per second. The units normally used to express energy are the kilojoule (kJ = 10^3 J) and the megajoule (MJ = 10^6 J). The thermochemical calorie is equivalent to 4.184 J (1 kcal = 4.184 kJ).

Energy available from food and drink

12. Ingested food and drink contains chemical energy in the form of carbohydrate, fat, protein, and alcohol. The maximum amount of energy potentially available from a food by an organism can be determined by measuring the heat released after its complete combustion to carbon dioxide and water. This is the gross energy (GE), but not all GE from food is available for human metabolism because of losses during food utilisation.

13. Available energy is defined as metabolisable energy (ME) and the ME value of a food or diet can be measured as the difference between energy intake and all losses (mainly in faeces and urine and a small amount in sweat). Energy losses in faeces largely represent incomplete digestion, while energy losses in urine and sweat are due primarily to the urea content which represents the incomplete catabolism of protein.

14. The ME content is the value quoted as the energy content of foods on food labels and in the UK food composition tables (FSA, 2002). The ME content of a given food can be calculated from the amounts of protein, fat, carbohydrate, and alcohol in the food (determined by chemical analysis) using energy conversion factors (see Table 1). These conversion factors (called the Atwater factors) are estimates of the energy content of each macronutrient and alcohol and have been rounded for practical purposes. Alternatively, UK food composition tables of the ME content of a wide variety of commonly consumed foods can be used; these are based on analytical data. More detail on the energy yielded from different nutrients can be found in Appendix 2.

Table 1 Metabolisable energy (ME) conversion factors for nutrients and alcohol[a]

	ME factors	
	kJ/g	kcal/g
Fat	37	9.0
Carbohydrate		
Available carbohydrate expressed as monosaccharide	16	3.8
Fermentable non-starch polysaccharides	8	1.9
Organic acids	13	3.1
Polyols	10	2.4
Protein	17	4.0
Alcohol	29	6.9

[a] Food Standards Agency, 2002

15. Some ME utilisation results in energy lost as heat i.e. the thermic effect of food (TEF) (Schutz *et al.*, 1984) (see paragraph 31). The extent of this varies with the type of food ingested, but specific amounts are associated with amino acid metabolism, alcohol metabolism and the microbial fermentation of otherwise unavailable carbohydrate. Energy lost via TEF may be subtracted from ME, resulting in an expression of the capacity of food energy to fuel metabolic work or for conversion to stored energy. This is termed the net metabolisable energy (NME) (FAO, 2003). While the ME and NME values are the same or similar for available carbohydrate and fat, NME values are lower for protein, alcohol and fermentable non-starch polysaccharides.

16. The human body is able to capture some of the chemical energy from food through cellular metabolism, resulting in the generation of an intermediary chemical form, adenosine triphosphate (ATP). ATP acts as an energy source for cellular processes mainly through phosphorylation of proteins and other intermediates. It is regenerated from adenosine diphosphate (ADP) using the energy in food. Cells require chemical energy for three general types of tasks: 1) to drive metabolic reactions that would not occur automatically; 2) for the transport of substances across cell membranes especially, against a concentration gradient; and 3) for mechanical work, e.g. muscle contraction. Energy is also released as heat both in the formation of ATP and during its use in these metabolic processes, and this maintains body temperature. Food energy can also be directly converted to heat if the oxidation pathway is uncoupled from the ATP-producing process.

17. FAO/WHO/UNU reviewed the case for the use of NME in place of ME values (FAO, 2003). While it was recognised that NME represents the biological ATP-generating potential of foods, it was recommended that, for the present, the ME system should be retained. The FAO/WHO/UNU recommendation (FAO, 2004) is endorsed by this report.

Components of energy expenditure

18. The total energy expenditure (TEE) of an individual can be divided into a number of discrete components, which can be determined separately. These are daily energy expended at rest, the *basal metabolic rate* (BMR), energy expended in physical activity, and other components such as thermogenesis resulting mainly from food intake, and growth. These components, and their variation as a function of body size and composition, and other factors are described in detail in Appendix 3 and are briefly summarised here.

Basal and resting metabolism

19. The basal metabolic rate (BMR) is a standardised measure of an individual's metabolism in a basal state: i.e. while awake and resting after all food has been digested and absorbed, in a thermoneutral environment. BMR represents the metabolic activity of cells and tissues and the physiological functions essential for life. For most individuals, BMR is the largest component of energy expenditure and requirements, ranging from 40 to 70% depending on age and lifestyle. BMR can be estimated from body size, age and gender; suitable equations are discussed in Appendix 4.

Physical activity

20. Physical activity includes a wide range of behaviour and encompasses sitting, standing, walking, planned exercise, and so on (Wareham & Rennie, 1998). Thus exercise, as planned and structured physical activity, is a subset of physical activity that may have an objective of maintaining or improving physical fitness and/or health. The degree of physical fitness of an individual can be measured with specific tests (Caspersen *et al.*, 1985). Other subsets of physical activity include spontaneous physical activity (SPA), a term used to describe all body movements associated with activities of daily living, change of posture and 'fidgeting' (Ravussin *et al.*, 1986).

21. Physical activity-related energy expenditure (PAEE) is quantitatively the most variable component of TEE usually accounting for 25-50% of energy expenditure and in some unusual circumstances up to a maximum of 75% (Westerterp, 1998). PAEE can have an impact on the metabolic rate beyond the period of a specific exercise for a few hours or even longer; this is described as excess post-exercise oxygen consumption (EPOC).

22. The energy costs of different physical activities can be expressed as multiples of BMR, called physical activity ratios (PAR), to account for differences in body size. Thus, PAR is used for discreet time periods of several hours or less. The physical activity level (PAL) over 24 hours is used as a measure of daily TEE adjusted for BMR.

The physical activity level (PAL)

23. A detailed discussion of PAL is provided in Appendix 5: an overview is given here. Twenty four hour TEE is a complex function of weight, age, gender, lifestyle and behavioural phenotype, but can be simplified and expressed as a multiple of BMR;

this is defined as the physical activity level, PAL (sometimes called the physical activity index, PAI). Thus PAL = TEE/BMR and is an index of 24h TEE adjusted for BMR, and is theoretically independent of those factors influencing BMR (weight, age and gender), at least as a first approximation. In the present context, PAL is important because it can be used to predict TEE, and hence energy reference values, as PAL x BMR (see paragraph 79).

24. PAL values are best estimated from direct measures of BMR combined with measures of TEE over periods of 24 hours or longer. Such measurements in free-living populations have indicated that PAL can range from <1.3 in immobile subjects to between 3-4.7 for limited periods of time in, e.g. soldiers on field exercises, elite endurance athletes, or Antarctic explorers (Black, 1996; Hoyt & Friedl, 2006). Within the general population, however, the overall range of PAL values for individuals in energy balance, leading sustainable lifestyles, is between 1.38 for the most sedentary to 2.5 for the most active (Prentice, 1995; Schutz, 2000). A change in lifestyle will change an individual's PAL value by reasonably predictable amounts. For example, an additional 150 minutes of moderate intensity activity a week, as currently recommended for adults in the UK (CMOs, 2011), will raise PAL by about 0.15 units (see Appendix 5 for details).

25. In previous reports, the duration and energy cost of individual activities (as PAR values) has been summed to provide factorial estimates of PAL (DH, 1991; FAO, 2004; IoM, 2005; WHO, 1985). A comprehensive summary of all published PAR values for a wide range of different physical activities for adults was compiled by Vaz *et al* (2005).

26. The factorial estimation of PAL assumes that overall energy expenditure within the general population can be predicted using information derived from activity diaries or lifestyle questionnaires. However, there is little evidence that this can be done with sufficient accuracy for several reasons.

27. Firstly, for most lists of activities used to identify lifestyle categories, individual activities are only defined in general qualitative terms. Consequently, there can be large inter-individual variations in the energy expended for each activity. In part this is due to variation in activity intensity resulting from variation in fitness (Martins *et al.*, 2007). Also, such listed energy costs may or may not include any thermic effect of food (TEF) or excess post-exercise oxygen consumption (EPOC).

28. Secondly, factorial estimates generally assume that time not allocated to activities is occupied by basal energy expenditure and take no account of variation in those body movements associated with activities of daily living, change of posture and 'fidgeting' (i.e. SPA (Ravussin *et al.*, 1986): see Appendix 3). SPA accounts for between-individual variation in energy expenditure of $\pm$15%. The potential for variable SPA throughout the range of defined activities adds to the uncertainty that their energy costs can be predicted.

29. The extent of error in factorial estimates of PAL does not appear to have been systematically evaluated. However, an examination of the doubly labelled water (DLW)-derived TEE literature in Appendix 5 (paragraphs 283-286) identifies considerable evidence that classification of individual lifestyles is a poor predictor of PAL.

30. In this SACN report, factorial predictions of PAL values for specific lifestyle categories have not been attempted due to the likely error in estimating actual TEE from listed PAR values of activities and the phenomenon of behavioural phenotypes, which exhibit very different rates of energy expenditure. Instead, PAL values for children, adolescents and adults have been identified from an analysis of the available DLW-derived TEE literature judged to be appropriate for the UK population (see paragraphs 104-107 and 113-118). For detail on the DLW method, see paragraph 70 and Appendix 6.

Thermic effect of food (TEF)

31. The TEF or heat increment of feeding reflects the metabolic costs of eating, digestion, absorption and metabolism of food and nutrients. Although TEF varies with food composition it is usually assumed to equate to energy expenditure equivalent to 10% of energy intake (Kleiber, 1975).

Other components of energy expenditure

Growth

32. Physical growth involves an increase in both the size and complexity of body structure, occurring under genetic and endocrine regulation in the presence of adequate nutrient supply. During growth, organs and tissues do not grow at a uniform rate, e.g. in the full-term newborn the brain represents about 12% of body weight, but in the adult is about 2% (Stratz, 1904). Energy is deposited into new tissues, the major part of growth costs, and some is expended during the synthesis of growing tissues. The energy required for growth is highest in the first three months of life when it accounts for about 35% of energy requirements, by 12 months of age this falls to about 3% (Butte *et al.*, 2000a). The energy cost of growth remains low from one year of age to mid-adolescence, then increases slightly during the adolescent growth spurt; by the late teens the amount becomes very small (Tanner, 1990).

33. Changes in relative organ size influence both energy and protein metabolism. Protein synthesis per unit of body mass proceeds at a high rate in the neonate and declines throughout infancy. High protein turnover contributes to the relatively high energy requirement of the newborn. Gender differences in body composition become more apparent after puberty. The developmental aspects of body composition and whole body metabolism affect the energy cost of growth (Butte *et al.*, 1989; Butte *et al.*, 2000b).

Pregnancy and lactation

34. During pregnancy, energy is needed for placental and fetal growth and for the growth of maternal tissues, e.g. uterus, breast and adipose tissue. In addition to these growth costs, there are increased energy costs associated with maintaining the larger tissue mass, along with an increased energy cost of movement, particularly for weight bearing activities after 25 weeks gestation (Butte *et al.*, 2004). However, some of these costs may be offset by adaptive changes in activity and in maternal metabolism.

35. During lactation, energy is lost as secreted milk and expended in producing the milk. Fat reserves that accumulate during pregnancy provide a variable proportion of this requirement (Butte & King, 2005).

36. The energy content of any tissue laid down during growth, pregnancy and lactation or of milk produced is not accounted for in TEE measured at this time; these additional costs are estimated from analysis of tissue deposition and milk secretion. The TEE, however, does include the energy required for the metabolic costs of new tissue synthesis or milk production.

Factors affecting energy expenditure

37. A detailed discussion of factors affecting energy expenditure can be found in Appendix 3, a summary is given here. Physical activity is considered in Appendix 7.

Body size and composition

38. Basal and total energy expenditure are related to body size with larger people having more tissue mass and a higher BMR. Both height and weight are determinants of the BMR although the independent influence of height is much less than that of weight. In infants, children and adolescents, there is an increase in BMR with age, due to growth and increasing tissue mass, although the changing relative organ sizes in early life complicates the relationship between body weight and BMR.

39. After adjustment for body size, inter-individual variation in BMR is mainly determined by variation in the most metabolically active tissue mass of the body, termed fat-free mass (FFM; the non-fat component of body composition comprising muscle, bone, skin and organs). The fat component of the body is termed fat mass (FM) and varies considerably between individuals in terms of the absolute amount. The differences observed in BMR with gender, age and ethnicity, after adjustment for body size, are mainly accounted for by differences in body composition, i.e. FFM (see Appendix 3 for further discussion).

40. The increasing body size of overweight and obese individuals increases both FM and FFM and the absolute BMR (Das *et al.*, 2004; Prentice *et al.*, 1996a) although the relationship is not linear (Prentice *et al.*, 1996a). Despite this, prediction equations developed for BMR as a function of weight, age, gender and height, provide a reasonably accurate estimate of BMR over quite a wide range of BMI; this is indicated by the similar relationships between BMI and measured and estimated

BMR in the adult DLW cohorts used to derive PAL values in this report (see Appendix 8). However, in this report, because prescriptive EAR values are defined (see paragraph 59), BMR values are only calculated for healthy body weights at the height of the population group, and potential errors in estimating BMR values for body weights markedly outside the healthy body weight range will not influence EAR values.

41. In individuals with higher percentages of body fat, a decrease in the mechanical efficiency of movement can increase the energy expenditure associated with certain types of activity. On a population basis, for adults up to a moderate level of fatness (i.e. overweight, but not obese), such influences of fatness on activity-specific energy expenditure are generally ignored (Durnin, 1996). With obesity these changes may become important because, as discussed in Appendix 3 (paragraphs 233 and 235), PAEE appears to decline only slightly with increasing BMI, and PAL does not vary with BMI; this suggests that any reduction in activity with increasing obesity is offset by the increasing energy cost of such activities with increasing BMI.

Genetic variation

42. While many studies have investigated the role of genetic variation on energy expenditure there have, as yet, been no clearly established relationships between specific gene variants and energy expenditure. For example, a number of studies have investigated the association between the mitochondrial uncoupling protein gene variants and energy expenditure, but results have been equivocal.

Hormones

43. Several hormones, e.g. sex hormones, thyroid hormone, adrenaline and leptin, may affect energy expenditure and have been implicated in the regulation of energy balance. Pharmacological agents, such as glucocorticoids, amphetamines and some anti-obesity drugs have all been shown to increase energy expenditure, while opiates and barbiturates can decrease it. Smoking acutely increases resting energy expenditure to a small extent (e.g. a 3.3% increase in resting metabolic rate (RMR) over a three hour measurement period (Collins *et al.*, 1994)).

Illness

44. The effect of illness on energy expenditure is discussed in more detail in Appendix 9. In patients with a range of conditions (sepsis, degenerative diseases, malignancy, trauma, congenital conditions and others), TEE is usually normal or reduced, partly because of a reduction in body weight and FFM as a result of disease-related malnutrition or neurological causes of wasting, and partly because of reduced physical activity. The latter compensates for any increase in BMR, which is common in acute and many chronic diseases. There are some exceptions, such as subgroups of patients with cystic fibrosis, anorexia nervosa, and congenital heart disease, where TEE has been reported to be increased (Elia, 2005). Specialist clinical advice is essential when considering energy requirements for people with disease.

Ambient temperature

45. Body temperature is tightly regulated in order to maintain cell function. A component of this regulation relies on variation in energy expenditure. Differences in environmental temperature affect energy expenditure and have been shown to account for about 2-5 percent of the variation in TEE. Indoor temperatures, however, are typically controlled to remain relatively constant and individuals adjust their clothing to create a relatively constant thermal microenvironment, so in reality, ambient temperature has a minimal effect on energy expenditure.

Energy balance and storage

46. Energy balance is achieved when ME intake is equal to TEE, plus the energy cost of growth in childhood and pregnancy or the energy cost of milk production during lactation. A positive energy imbalance occurs when energy intakes are in excess of these requirements, while negative energy imbalance occurs when energy requirements are not met by intake.

47. Energy intake should exceed TEE during growth, pregnancy and lactation when new tissues are being laid down or milk produced. In other circumstances, energy intake which exceeds expenditure is stored. Triglycerides within adipose tissue act as the body's major energy store. Some energy is also stored in the liver and skeletal muscle as glycogen. The amount of energy stored in the adipose tissue of a healthy adult of normal weight is equivalent to approximately one month's energy requirements (Schutz & Garrow, 2000).

48. Short-term, day to day energy imbalances are accommodated by the deposition and mobilisation of glycogen and fat. Positive and negative energy imbalances occur in the short term in free-living individuals, so, in terms of weight regulation, it is important to consider the overall energy balance over a prolonged period of time.

49. Chronic negative energy imbalance results in the utilisation of stored energy from triglyceride in adipose tissue and protein in muscle and viscera, since glycogen stores are limited and are rapidly exhausted. Chronic positive energy imbalances are mostly accommodated by the deposition of adipose tissue triglycerides, together with a small but fixed ratio of lean tissue (Schutz & Garrow, 2000). Thus, an individual in chronic positive energy imbalance stores excess food energy mainly as triglyceride and to a lesser extent protein. Muscle and liver glycogen stores are modest and have a limited capacity; whereas the capacity of the body to store triglycerides in adipose tissue is substantial.

Obesity

50. Obesity results from a long-term positive energy imbalance. The increasing prevalence of obesity must reflect temporal lifestyle changes, since genetic susceptibility remains stable over many generations, although inter-individual differences in susceptibility to obesity may have genetic determinants (Maes *et al.*, 1997). Definitions of obesity and current prevalence in the UK population are discussed in Appendix 10.

51. Obesity increases the risk of a number of diseases (see Table 2 for an overview). In 2009, a collaborative analysis of the influence of BMI on all-cause mortality in 57 prospective studies (900 000 adults), identified a U shaped relationship with minimum risk associated with a BMI of about 22.5-25kg/m^2 in men and women (Prospective Studies Collaboration *et al.*, 2009). This relationship and how it has informed the approach used to derive energy reference values in this report will be discussed in more detail in Section 3.

Table 2 Summary of associations observed in prospective studies between obesity and subsequent ill health[a]

Association for increased risk	Disease outcome
Relative risk >3	Type 2 diabetes
	Insulin resistance
	Hypertension
	Dyslipidaemia
	Breathlessness
	Sleep apnoea
	Gall bladder disease
Relative risk about 2-3	Coronary heart disease or heart failure
	Osteoarthritis (knees and hip)
	Hyperuricaemia and gout
	Complications of pregnancy, e.g. pre-eclampsia
Relative risk about 1-2	Cancer, e.g. oesophagus (adenocarcinoma), colorectum, breast (postmenopausal), endometrium and kidney
	Impaired fertility/polycystic ovary syndrome
	Low back pain
	Increased risk during anaesthesia
	Fetal defects associated with maternal obesity

[a] based on Correa et al., 2008; Haslam *et al.*, 2006; Key *et al.*, 2004; Renehan *et al.*, 2008

The influence of physical activity and diet on the regulation of body weight

52. The influence of physical activity and energy intake in relation to body weight regulation is discussed in detail in Appendix 7 and 11; a summary of the main considerations is given here.

53. Body weight is gained when energy intake exceeds TEE over time. PAEE is the most variable component of TEE and is amenable to modification, so changes in PAEE may affect risk of weight gain.

54. Methodological constraints are a severe limitation in defining the role of physical activity in the regulation of body weight, for example, studies and surveys rely mostly on subjective measures of reported physical activity. Proxy measures of population-level physical activity trends suggest that changes in activity in domestic life, work and travel have coincided with the increase in prevalence of obesity. A review of the available evidence from prospective cohort studies

suggested that, on balance, increased physical activity and decreased sedentary behaviour were associated with lower relative weight and fatness gains; however, the results were mixed and the identified associations were generally of a small magnitude (Wareham *et al.*, 2005). Evidence from studies using objective measures of physical activity, especially DLW-derived measures of PAEE, and trials of the primary prevention of weight gain, is inconsistent. Available data are insufficient to accurately define a level of energy expenditure or the required frequency, intensity and duration of physical activities required to reduce the risk of unhealthy weight gain.

55. Methodological constraints are also a severe limitation in defining the role of diet and dietary composition in the regulation of body weight, for example, limitations in the accurate assessment of dietary exposures and under-reporting of dietary intake (Bandini *et al.*, 1990a; Bingham *et al.*, 2001, 2003; Bingham & Day, 2006; Buhl *et al.*, 1995; Champagne *et al.*, 1998; Goris *et al.*, 2000; Lichtman *et al.*, 1992; Livingstone & Black, 2003; Rennie *et al.*, 2005). A prolonged excess energy intake is, however, fundamental to weight gain and the development of obesity. Household purchase data from the Family Food module of the Living Costs and Food Survey[13] suggest that there has been a decrease in average daily energy intakes, which seems to date from the 1960s, and coincides with the time period in which there has been a large increase in the prevalence of overweight and obesity. Since under-reporting of food intake is particularly pronounced in the overweight and obese (Rennie *et al.*, 2007), the proportion of the population likely to under-report increases as the population gains weight, thus exacerbating the problem of under-reporting of energy intakes (Bandini *et al.*, 1990a; Rennie *et al.*, 2005). The use of household purchases as a proxy for consumption in these surveys also limits the accuracy with which energy intakes can be estimated, and in addition, reported energy values do not take into account food waste. The NDNS, in which food consumption by individuals has been measured with seven day weighed diaries, a considerably more robust methodology than that used in Family Food, also shows average energy intakes in men, but not women, have decreased between 1986/7 and 2000/1. Under-reporting is also an issue in NDNS (estimated to be approximately 25% of energy needs in both sexes (Rennie *et al.*, 2005)) and there is some indirect evidence of a secular trend towards increasing under-reporting over time (Rennie *et al.*, 2005).

56. Energy flux or turnover is the rate at which energy in all its forms flows through the body on a daily basis (chemical, work, thermal). For an individual in energy balance, the energy flux is numerically the same as energy expenditure or energy intake. Provided energy intake matches energy expenditure then energy balance will be maintained whether the energy flux itself is at a higher or lower level. However, as the flux of energy might have important physiological relevance in its own right, the level of energy flux at which balance is achieved should be considered. It has

13 The Family Food module of the UK Living Costs and Food Survey was formerly known as the Expenditure and Food Survey (EFS). The EFS was formed in 2001 from the merger of the National Food Survey (NFS) and the Family Expenditure Survey. Under-reporting in Family Food is thought to be lower than in the NFS as it does not only focus on food purchases, but on household expenditure across the board and is largely based on till receipts. Family Food also provides better estimates of food eaten outside the home than did the NFS. The change in methodology when the EFS replaced the NFS, however, makes the estimate of year on year change unreliable between 2000 and 2001-02.

long been hypothesised that the mechanisms controlling energy balance may be more sensitive and effective in individuals with higher levels of physical activity and hence energy flux, while sedentary individuals may be below the threshold of physical activity at which these mechanisms become imprecise, leading to obesity (Mayer & Thomas, 1967). There is some indirect evidence for this hypothesis that the coupling between energy expenditure and energy intake may be less precise at low levels of physical activity (Prentice & Jebb, 2004; Schoeller, 1998).

Definition of energy requirement

57. The energy requirement, and associated descriptive terminology, must be defined with particular care in the context of a population which includes many individuals who are overweight or exhibit habitually low levels of physical activity. COMA (DH, 1991) defined requirements in general terms only i.e. intakes of nutrients which were likely to meet the needs of some or all within population groups. There was no specific definition of the energy requirement in relation to specific healthy body weight ranges or prescribed levels of energy expenditure. COMA reported EAR values calculated for a wide range of physical activity levels and adult body weights, which in practice, would maintain the *status quo* in terms of existing body weights of such population groups. The COMA report did include a widely quoted summary table[14] for sedentary younger and older adult men and women at specific body weights which were within the healthy range, albeit within the upper part of this range[15].

58. The definition offered by FAO/WHO/UNU (WHO, 1985; FAO, 2004), in the context of a diet which contains adequate amounts of all essential nutrients, was '*the amount of food energy needed to balance energy expenditure in order to maintain body size, body composition and a level of necessary and desirable physical activity consistent with long-term good health. This includes the energy needed for the optimal growth and development of children, for the deposition of tissues during pregnancy, and for the production of milk during lactation consistent with the good health of mother and child*' (WHO, 1985; FAO, 2004). Thus this definition includes both healthy body weights and desirable physical activity levels, with the term "normative" used in the 1985 report and "prescriptive" in the 2004 report. Each report calculated energy requirements based on suitable reference body weight ranges, with the 1985 report noting that application of such requirements to groups with body weights below or above such ranges would tend to mediate weight change towards the median. Neither report, however, calculated values in relation to a "desirable" physical activity level.

59. In this SACN report, the requirements for energy for all population groups have been set at *the level of energy intake required to maintain a healthy body weight*[16]

14 Table 2.8 *Estimated Average Requirements (EARs) for energy for groups of men and women with physical activity level of 1.4* (DH, 1991).

15 These were median body weight values from a 1980 survey of adult heights and weights in Great Britain, and were equivalent to BMI values of 24.1 and 24. 9 kg/m^2 for men and 23.1 and 24.7 kg/m^2 for women.

16 Although the healthy body weight range is usually defined as a BMI between 18.5-24.9kg/m^2, for the purposes of calculating the revised EAR values, in this report healthy body weights have been identified as those associated with a body mass index of 22.5 kg/m^2 which represents the lower end of the minimum mortality range (Prospective Studies Collaboration *et al*., 2009) (see paragraph 51).

in otherwise healthy people at existing levels of physical activity and to allow for any special additional needs (growth, pregnancy and lactation). Thus, the prescriptive[17] terminology and principle (i.e. relating to an desirable standard as distinct from *status quo*) have been adopted, but only for body weight, with physical activity set at best estimates of existing levels. However, as with previous reports, the importance of adequate physical activity is recognised and this report includes advice on desirable physical activity consistent with long-term health (Chief Medical Officers [CMOs], 2011). The likely impact of such advice on energy requirement values is also described.

60. Energy requirements described here are identified from sufficient measurements of energy expenditure on healthy, well-nourished individuals to demonstrate how energy expenditure varies according to age, gender, body size and composition, pregnancy, lactation and physical activity. These characteristics are used to describe population groups for whom EARs are defined, with values calculated for best estimates of healthy body weights at the heights of each population group and at respective current levels of physical activity.

Variability

61. Measurements of energy needs that are used to predict DRVs for a particular population group will, for a number of reasons, exhibit variability. Consequently, there will be a distribution of energy reference values for each population group and the level set for the DRV should take this into account.

62. For most nutrients the DRV is identified as the Reference Nutrient Intake (RNI) from the upper bound of the reference ranges i.e. two notional standard deviations above the average reference value (DH, 1991). However, for dietary energy the DRV is defined differently i.e. it is equal to the average reference value (EAR). The RNI for dietary energy is not used because it represents an excess energy intake for the majority of the population. Energy intakes that consistently exceed requirements lead to weight gain and obesity in the long term. An intake equal to the average reference value for a population group on the other hand is, in theory, associated with similar probabilities of excessive and insufficient energy intakes for any individual within the population.

63. Appetite regulation allows humans in good health to match broadly their energy intake to their requirement so the chances of energy deficiency are low unless food supply is limited. However, this mechanism is not always sufficiently sensitive to prevent small excesses leading to inappropriate weight gain in the long term.

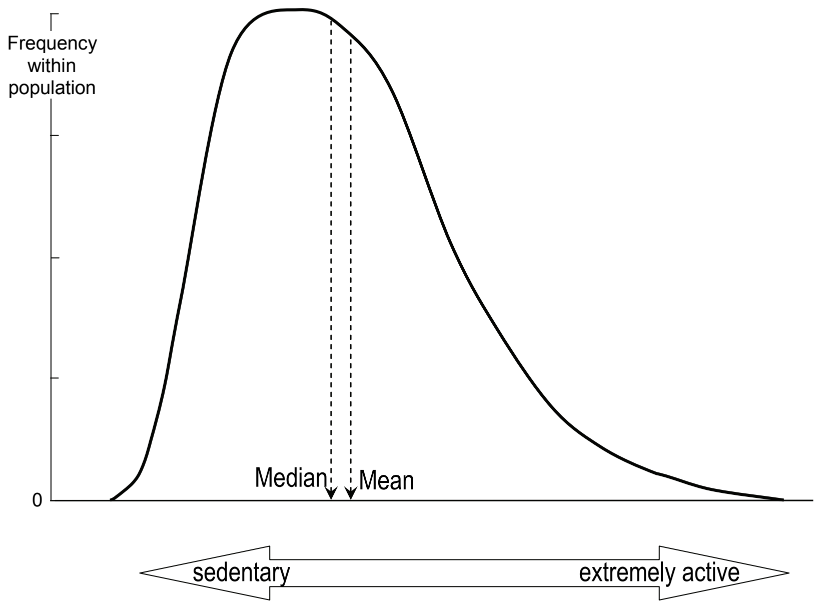
64. In countries like the UK, the population is characterised by sedentary lifestyles at all ages. As a result, rates of energy expenditure and intakes that maintain energy balance at these levels of energy expenditure are unlikely to be normally distributed. Measurements of energy expenditure within adult populations[18] indicate that the overall distribution is skewed towards sedentary behaviour. For

17 Sometimes used interchangeably with 'normative'.

18 See Appendix 6 for a description of the methodology for measuring energy expenditure and the uncertainties that exist in its application and interpretation.

the high proportion of individuals who are relatively inactive, energy expenditure and intakes that maintain energy balance will cluster at the lower end of the range. A small proportion will be more active, with a few individuals exhibiting the highest rates of expenditure that are sustainable. A schematic diagram of the probable distribution within the adult population is shown in Figure 1. Statistical considerations, therefore, dictate that the appropriate descriptor of the midpoint of the distribution is the median, which is likely to be somewhat lower than the mean, thus the median is used in this report.

Figure 1 – Schematic of the likely distribution of energy expenditure (expressed as a multiple of Basal Metabolic Rate – BMR) and hence reference intakes within the adult population



65. Evidence that energy expenditure varies between individuals with similar lifestyles over a wider range than would be expected (at least in adults) presents a second difficulty in estimating energy reference values. This is particularly important since in the past, attempts have been made to define energy reference values in terms of specific physical activity levels (as multiples of basal metabolic rate, see paragraphs 25 and 26) for specific lifestyle population groups. Consequently, with the probable exception of those at the extremes of the activity range, lifestyle predictions of energy expenditure and resultant energy reference values cannot be made with the certainty assumed in previous reports such as COMA (DH, 1991) (see Appendix 5 for a detailed consideration).

66. Finally, given the high prevalence of overweight and obesity at all ages in the current population (see Appendix 10), prescriptive energy reference values which are consistent with long-term good health need to be based upon suitable reference body weights, as distinct from existing body weights. If this principle is not adopted, application of such energy reference values would maintain body

weights, which are in excess of the healthy reference weights for many of the population. Thus, in contrast to COMA's report (DH, 1991), energy reference values defined in this report will be derived for infants, children and adults in relation to body weights which, on the basis of current evidence, are likely to be associated with long-term good health. This means that for people who are underweight, overweight or obese, energy intakes at the reference levels should enable the transition towards the healthy body weight range (BMI 18.5-24.9 kg/m^2) over time.

Approaches used to estimate energy reference values

67. Energy reference values are defined as the Estimated Average Requirement (EAR) for food energy for specific population groups, not for individuals, and are set using two basic approaches:

1) Measurement of energy intake (EI) of healthy reference populations in energy balance, growing appropriately or achieving successful pregnancy and lactation, provides, in theory, a direct estimate of the energy requirement. However, it has not proved possible to make such measurements with sufficient accuracy for them to be useful, especially for groups where under-reporting or failure to report 'usual' food and drink intake may be an issue (e.g. overweight adults) (Black *et al.*, 1993; Livingstone, 1995; Prentice *et al.*, 1986). In addition, EI is not a physiological measure of the energy requirement since it is not adequately regulated through the appetite mechanism to match TEE precisely and allow exact energy balance, and EI can be consciously altered by the subject. Also, there is no independent check on whether the measured EI matches TEE and is therefore appropriate for the subject's need. Thus, in practice, this approach has been largely abandoned in favour of approaches measuring TEE.

2) Measurement or prediction of TEE provides a physiological measure of the energy requirement at energy balance. This is because for energy balance, EI must equal TEE. Thus, the energy requirement can be predicted specifically as the rate of TEE plus any additional energy needs for growth, pregnancy and lactation.

Measurement of total energy expenditure (TEE)

68. Several approaches are used to measure TEE. Short-term measurements under highly defined conditions can be made by calorimetry. Direct calorimetry which measures the rate of heat loss from the subject to the calorimeter is the most accurate method. Indirect calorimetry, the most commonly used approach, measures oxygen consumption and/or carbon dioxide production from which TEE is calculated using standard formulae such as the Weir equation (Mansell & Macdonald, 1990).

69. The components of TEE (e.g. BMR, PAEE) can be measured separately using direct and indirect calorimetry. TEE can also be measured with large walk-in calorimeters, although the necessary confinement of subjects limits this to short periods, most often 24 hours. The information provided by these approaches is accurate and has provided the energy costs of different physical activities (see paragraph 22 and Appendix 5) and minimal daily rates of TEE. Free-living activities cannot usually

be measured by these techniques. Some non-calorimetric techniques can be used to predict free-living TEE by extrapolation from physiological measures, the best example being heart rate monitoring (Levine, 2005). These methods need first to be calibrated against direct or indirect calorimetry before TEE can be calculated.

70. The DLW method (International Atomic Energy Agency [IAEA], 2009) is generally recognised as the most accurate measure of free-living TEE currently available and is discussed in detail in Appendix 6. DLW measures the rate of carbon dioxide production, and hence TEE, in free-living subjects over a period of several days to several weeks providing more accurate measures of TEE than other non-calorimetric methods, e.g. heart rate monitoring (Levine, 2005).

71. Whilst the DLW method is the best available, a series of assumptions are made which can affect the predicted TEE values. There are also concerns regarding recruitment bias, since people who are willing to participate in DLW studies may not be representative of the population as a whole. Nevertheless, the DLW method has proved to be the most useful approach. It remains the method of choice and is more accurate than others available. TEE measurements obtained by this method are judged to be representative of the current TEE of the UK population and form the basis of estimates of energy reference values in this report.

Utilising TEE measurements to derive energy requirements

72. The 1985 FAO/WHO/UNU report (WHO, 1985) was the first to use a factorial approach to estimate energy requirements based on the prediction of TEE as BMR x PAL (see paragraphs 23-30). This approach was adopted by COMA in 1991 (DH, 1991) and by the most recent FAO/WHO/UNU report (FAO, 2004) for calculating energy requirements of adults, although not used in the US DRI report (IoM, 2005) (see paragraph 86). The calculations made in these reports estimated BMR from anthropometric measures (as described in paragraphs 80 and 81) and were based on the assumption that values for PAL can be estimated from time-allocated lists of daily activities expressed as PAR values (see paragraph 22). With information about PAL values for specific lifestyles and types of daily activities, appropriate PAL values can then be assigned to particular population groups.

73. Reservations have been expressed regarding the prediction of PAL through the summation of PAR values (see paragraphs 26-28 and Appendix 5). Determination of energy reference values using *measured* values of TEE is therefore preferable. There are two potential analytical approaches that can be employed to derive energy reference values from measured TEE:

- predicting TEE using regression equations (see paragraphs 74-78);
- employing a factorial approach based on the assumption that TEE is equal to BMR x PAL (see paragraph 79).

Predicting TEE with regression equations

74. The first approach is to use measured TEE directly to derive regression equations which describe how TEE varies as a function of anthropometric variables (such as weight and height) for defined population groups. The regression equation can

then be used to predict TEE and resulting energy reference values for any group on the basis of their anthropometric variables (see Appendix 5 for details).

75. The analysis of TEE as a function of its potential predictor variables (weight, age and gender) by multiple regression techniques would seem a logical progression from the accumulation of reliably estimated measures of free-living TEE by the DLW method (Goran, 2005). In practice, however, a major limitation to this approach has been the inability of TEE prediction models to account for variation in PAEE, an important source of variation in TEE, in a transparent way.

76. Variation in PAEE, at least in terms of its duration and intensity, is not physiologically related to anthropometric variables in the same way as the BMR, and any such variation will be lost in a regression model involving just anthropometric variables. The regression equation will, in fact, predict values for TEE which contain a PAEE component comparable to the mean value observed at any given weight or age within the data set used to generate the regression.

77. Between-individual variability in PAEE in a regression of TEE on anthropometric variables is to some extent indicated by the residuals of the regression. Such residuals (the difference between observed TEE values and those predicted by the regression) will include all TEE not accounted for by the regression, of which variation in PAEE will be the major part. However, any variation in the basal energy expenditure not accounted for by body weight, age and gender, will also be included. Thus, use of residuals in terms of predictors of the likely range of variation in PAEE within population groups is limited and residuals have not been used as such in any previous report (see Appendix 5 for more detail).

78. Instead, previous reports which have used regression models of TEE to predict energy requirements (FAO/WHO/UNU (FAO, 2004)[19] and the US DRI values for energy (IoM, 2005), made special provision for the likely variation in PAEE within the population group described by the regression. The methodology employed, is examined below (paragraphs 85 and 86).

Predicting TEE from BMR and PAL values extracted from measured TEE – the factorial approach

79. The second analytical approach to the derivation of energy requirements from measured TEE employs the factorial approach[20] in the following steps:

- TEE values measured in a reference population are divided by measured or estimated BMR to extract PAL values. This means that the reference populations studied by DLW are described primarily by PAL values.
- For the population of interest, BMR values are then estimated from BMR prediction equations using relevant anthropometric data for the population.

19 Regression equations were used to derive energy requirements for infants, children and adolescents. The energy requirements of adults were calculated from factorial estimates of habitual TEE.

20 This approach has also been referred to as the BMR multiple approach and the PAL model.

- The PAL values derived from the reference population can then be used to estimate TEE and EAR values for the population of interest based on the latter's estimated BMR values.

Whilst some criticism of the PAL x BMR approach has been expressed (Goran, 2005) (see Appendix 5), no satisfactory alternative has yet been identified.

Estimating BMR

80. A variety of BMR prediction equations are described in the literature and different authors favour different equations in published work especially in the US and Europe. Within the UK COMA report (DH, 1991) and for FAO/WHO/UNU reports (FAO, 2004; WHO, 1985), the Schofield equations have primarily been used (Schofield *et al* 1985). The Schofield equations are a series of predictive equations for BMR based on height, body weight, age and gender, although simplified equations based on just body weight, age and gender are used in these reports. The equations are derived from an analysis of a compilation of calorimetric measures of BMR values and anthropometric data. These predictive equations, slightly modified, form the basis for FAO/WHO/UNU energy requirements for adults (FAO, 2004) and modified versions were used by COMA for the previous UK EAR for energy in children aged 3-18 years and adults (DH, 1991).

81. The data set upon which the Schofield prediction equations for BMR were based was compiled mainly from results in West European and North American subjects, with almost half of the subjects being Italian in whom BMR was measured using a closed circuit method[21] in the 1930s and 1940s (Henry, 2005). The use of the closed circuit method has been queried as it may overestimate oxygen consumption and consequently energy expenditure (FAO, 2004). Furthermore, the applicability of the Schofield equations to all population groups has been questioned and an alternative more comprehensive database has been assembled and analysed from which a new set of equations has subsequently been derived, i.e. the Henry equations (Henry, 2005). Although estimates of BMR made by the Schofield or Henry prediction equations differ only slightly in children or adults (i.e. the Henry equations are 3-4% lower), an assessment of the validity of different predictive equations for BMR in adults found the Henry prediction equations to be the more accurate (Weijs, 2008). In this report the BMR prediction equations published by Henry (2005) are therefore used to derive EAR values which, because they are prescriptive values, require the identification of reference values for healthy body weights for which BMR is calculated. A more detailed discussion on the BMR equations is provided in Appendix 4, with suitable values for reference body weights described below in paragraphs 90-93.

21 The methods available to measure BMR may be divided into two types; closed and open circuit methods. In the closed circuit indirect calorimetry method, the subject breathes in and out of a closed system, commonly a spirometer, which is sealed to room air. Oxygen, or a mixture of oxygen and nitrogen, is supplied to the spirometer at the rate at which it is consumed. Thus the rate at which oxygen is delivered is the same as oxygen consumption. Heat production may be estimated from oxygen consumption alone, in which case the carbon dioxide produced by the subject is absorbed, for example by soda lime, within the closed breathing circuit. Alternatively, weight gain of the carbon dioxide absorber may be used to derive carbon dioxide production using this method. (From: Green, 1994).

Calculation of energy requirements

82. There are two major considerations in the practical application of the factorial approach to calculate energy requirements using PAL and BMR values from appropriate DLW data sets: 1) identifying suitable PAL values appropriate for specific groups and populations; and 2) utilising this information to derive energy reference values.

83. PAL values can be calculated from DLW-derived measurements of TEE largely obtained from studies conducted in UK populations and comparable populations in the US and other developed countries over the last 20 years, and from measurements or predictions of the BMR. With few exceptions, published DLW studies involve relatively small numbers of healthy subjects who may or may not have been engaged in activities representative of the general population at that time. Although data from these studies can be combined to derive best estimates of PAL values for the whole population based on age and gender, the degree to which these values are representative of the current UK population is uncertain. For example, the data set assembled for the US DRI report (IoM, 2005) (n=767) included many individual studies with subjects exhibiting high levels of physical activity (see Appendix 5). An alternative is to make use of large population studies of randomly selected subjects which can be assumed to exhibit similar activity patterns to that of the current UK population (see paragraph 116 and Appendix 8).

84. Suitable PAL values can be used to update and improve tables of PAL value ranges for the various ages and lifestyle groups identified in previous reports, allowing energy reference values to be calculated for such groups (see paragraphs 25-30). However, the marked between-individual variation in PAL (which seems to occur independently of any predictable lifestyle) makes such a selection of an appropriate PAL value unreliable. The alternative approach is to evaluate the distribution of PAL values, with medians and centile ranges identified for the population as a whole. This approach allows energy reference values to be framed against such distributions, in effect substituting PAL distributions for PAL values defined in terms of lifestyle. Thus, energy reference values can be defined as a population EAR[22] (i.e. from the median PAL value) with additional values appropriate to those who are less or more active than the average (i.e. from the 25th and 75th centiles). Only three activity groups within the population are identified by PAL values using this method, but it is considered unrealistic to judge PAL values more finely. However, additional information on the change in PAL with specified additional activity will allow calculation of the probable additional energy intakes required to support such activities.

22 The word average in the EAR (Estimated Average Requirement) term is used here as a general term embracing both median and mean.

Approaches employed to calculate energy requirements in other reports

FAO/UNU/WHO (FAO 2004)

85. The development of regression equations for TEE from measured values forms the basis of the FAO/WHO/UNU (FAO, 2004) report on energy requirements for infants, children and adolescents (but not for adults). Data sets were developed using TEE values from DLW studies for infants, and DLW and heart rate monitoring studies for children and adolescents. Mean study values weighted by the number of subjects were then used to derive the regression equations. For children and adolescents, the predicted TEE values were not used directly but used to predict PAL values. This prediction was done by dividing TEE values from the regression with BMR values predicted as a function of age and weight. The calculated PAL values were assumed to represent activity levels for populations with "average" or "moderate" physical activity. Values for more ("vigorous") or less ("light"), active lifestyles were calculated as average ± 15%. This approach allowed calculation of energy requirements for the three lifestyles as BMR x PAL. For adults, FAO/WHO/UNU (FAO, 2004) adopted a factorial model (BMR x PAL) in place of regression equations with PAL values identified for lifestyle categories.

US DRI Institute of Medicine (2005)

86. The US Dietary Reference Intake (DRI) values for energy (IoM, 2005) were calculated from age-range and gender-specific prediction equations for TEE derived by regression analysis of a large (n=767) DLW data set of individual TEE values. These were obtained, with ancillary data, directly from the investigators of each study. However, unlike the FAO/WHO/UNU report (FAO, 2004), variation in physical activity was accommodated within the regression. This involved an activity constant, which could be one of four values, that the user must choose. Each of these four activity constants was identified as equivalent to a range of PAL values appropriate for sedentary, low active, active, and very active lifestyles. Thus, the user of the regression equations is instructed to estimate which activity/lifestyle category the subject or population group belongs to, and assign the appropriate activity term within the regression, together with age, weight, and height, to calculate the EAR value. Separate regression equations were defined for each gender and age range. In practice these equations differ little from prediction equations of the form BMR x PAL with four values of PAL identified for the four activity ranges.

3 Estimated average requirements – approaches used and values derived

Summary of approach used to determine EARs

87. The SACN Framework for the Evaluation of Evidence (SACN, 2002) was used as the basis to identify and assess published evidence of total energy expenditure (TEE) from which to guide derivation of energy reference values. Only studies using the doubly-labelled water (DLW) method to measure TEE were considered, as this represents the most accurate method for assessing TEE in free-living populations. No large-scale population studies of any age group have been conducted to determine DLW-derived TEE in the UK population and although TEE values for subjects of all ages obtained from the UK NDNS series were examined, this data set alone (n=156 adults) was not large enough to serve as a sole reference for the determination of energy reference values. Consequently, other data sources (reference populations) were sought. Priority was given to studies in well-characterised populations (e.g. by age, gender, weight, height or BMI) and where BMR had been measured directly rather than estimated from prediction equations. Studies of extreme energy expenditure, such as those involving elite athletes, and studies based on populations which are not commonly represented in the UK (such as ethnic groups not widely seen in the UK) were given less emphasis. The majority of studies identified were cross-sectional studies in healthy human populations of infants, children and adults and provided mean TEE values for the study population. Two large data sets of individual TEE values for adults were subsequently obtained from the USA, the OPEN and Beltsville studies (as described in paragraph 116 and Appendix 8). Some prospective cohort studies were also found and any potential link between energy intake and risk of ill health has been mainly drawn from these.

88. For adults aged 19-65 years and children aged between 3-18 years, the different approaches to setting EARs described above (see paragraphs 72-86) were examined. Regression modelling was explored and mean TEE values from the data set of DLW studies were used to develop regression equations of TEE against age, weight and gender for different age groups. However, as discussed above (see paragraphs 74-78) this approach is limited because of the inability of TEE prediction models to account for PAEE, an important source of variation in TEE, in a transparent way. As a result, the regression approach was not pursued and a factorial model was adopted, in which TEE is predicted from BMR x PAL (see paragraph 79).

89. Thus, new EAR values have been derived for children and adults. However, for infants the reference values stated in the FAO/WHO/UNU report (FAO, 2004) were used due to a lack of substantial new data. Pregnant and lactating women were considered separately.

Prescriptive energy reference values for healthy body weights

90. The high prevalence of overweight and obesity at all ages in the UK population (see Appendix 10) raises a clear difficulty in the definition of energy reference values for population groups. Such values, calculated to match energy expenditure, will, for many of the population, maintain overweight and will therefore not be consistent with long-term good health. Given the objective of defining prescriptive reference values, such values need to be based on healthy body weights.

91. For infants and children, healthy body weights are difficult to define but the UK-WHO Growth Standards (Royal College of Paediatrics and Child Health [RCPCH], 2011) for infants and preschool children are considered to be appropriate as the pattern of growth represented is associated with favourable health outcomes. For school children, the UK 1990 reference values for child growth (Freeman *et al.*, 1995) indicate body weights which are on average about 15% lower than recent (2009) UK values (NHS IC, 2010). These body weights can reasonably be assumed to be a better indication of healthy weights than current values.

92. For adults, the normal body weight range is generally defined as a BMI between 18.5 and 24.9 kg/m^2 (WHO, 1998). As already indicated (paragraph 51), a collaborative analysis of the influence of BMI on all-cause mortality in approximately 900,000 adults (Prospective Studies Collaboration *et al.*, 2009) identified a U-shaped relationship with minimum risk associated with a BMI of about 22.5-25kg/m^2. Above this range, positive associations were recorded for several specific causes (vascular mortality, diabetic, renal, and hepatic mortality and neoplastic mortality) with each 5 kg/m^2 higher BMI associated with about 30% higher overall mortality. Below 22.5-25 kg/m^2, the overall inverse association with BMI was predominantly due to strong inverse associations for smoking-related respiratory disease (including cancer).

93. On this basis, energy reference values were calculated in this report at the 50th centile of the UK-WHO Growth Standards for children up to four years (RCPCH, 2011) and using the UK 1990 reference values for child growth for children aged over four years (Freeman *et al.*, 1995). In adults, the healthy body weights that equate to a BMI at the lower end of the minimum mortality range (as discussed in paragraph 92) i.e. BMI 22.5 kg/m^2, have been adopted. Thus, EAR values should be calculated for a body weight equivalent to a BMI of 22.5 kg/m^2 at the height of the population group. Illustrative EAR values are shown calculated from current estimates of heights of the UK population[23]. Energy intakes matching the revised EAR values will be less than those which would maintain weight for overweight groups and can therefore form the basis of intakes designed to achieve healthier body weights. In contrast, energy intakes matching the revised EAR values will be greater than intakes that would maintain weight for those who are underweight.

23 It is important to note that a weight equivalent to a BMI of 22.5 kg/m^2 does not represent a precise target body weight to which everyone should aspire, rather that a single figure was required for the purpose of calculating prescriptive EARs based on healthy body weights. A body weight equating to a BMI within the range of 18.5 – 24.9 kg/m^2 is generally considered 'normal' or 'healthy'.

Energy reference values for infants, children and adolescents

Energy cost of growth

94. TEE measured using the DLW method includes the energy expended in tissue synthesis. Thus, only the cost of energy deposited in growing tissues should be added when calculating the energy reference values for infants, children and adolescents. For infants, such costs are relatively high and change during the first year of life (see paragraphs 100 and 101 and Tables 3 and 4). For older children and adolescents, growth costs are much less and the energy deposited can be accounted for by a simple adjustment to the factorial prediction of TEE from BMR x PAL (see paragraph 108 and Table 7).

Infants aged 1 – 12 months of age

95. Following the approach of the FAO/WHO/UNU report (FAO, 2004), the energy reference values for infants (Table 5) were estimated from TEE (measured by the DLW method in healthy, well-nourished infants born at full term within the range of normal birth weight (Butte, 2005)), plus the energy deposited during growth, estimated from measured protein and fat deposits (Table 3). These energy deposition values were then applied to weight increments taken from the UK-WHO Growth Standards (RCPCH, 2011) (Table 5) to derive EARs for breastfed, breast milk substitute-fed and for those infants where feeding is mixed or unknown. These standards describe the growth pattern of healthy infants living in non-deprived circumstances who were exclusively or predominantly breastfed for at least four months and introduced to complementary foods at a mean of 5.4 months (WHO, 2006). Since this pattern of growth is associated with favourable health outcomes, it is considered an indicator of optimal growth applicable to all infants and young children. In 2009, standards for children aged 0-4 years of age were adopted by the UK (Scientific Advisory Committee on Nutrition & the Royal College of Paediatrics and Child Health [SACN/RCPCH], 2007) as the growth standards to be used for clinical monitoring and population surveillance.

Energy expenditure

96. The FAO/WHO/UNU report (FAO, 2004) describes several equations relating TEE to infant body weights. One simple prediction equation for TEE as a function of body weight was derived from a longitudinal study of TEE with DLW measures conducted at three month intervals for the first two years of life on 76 infants (40 breastfed and 36 breast milk substitute-fed) (Butte *et al.*, 2000a).

TEE (MJ/day) = 0.371 kg – 0.416 n = 320, r = 0.85, see = 0.456 MJ/day (109 kcal/day)

97. This was very similar to the relationship between TEE and weight derived from an analysis of 13 published studies with DLW performed on a total of 417 infants aged 0-12 months using the *mean* values for TEE and body weight (Butte, 2005).

98. Exclusive breastfeeding to the age of about six months with continued breastfeeding as part of a progressively varied diet is recommended for all healthy

infants (SACN/RCPCH, 2007). The energy expenditure of breast milk substitute-fed infants has been shown to be higher than that of breastfed infants (Butte *et al.*, 1990; Jiang *et al.*, 1998; Butte *et al.*, 2000a;), indicating differences in TEE between feeding groups over the first year of life that diminish thereafter.

99. When predicting TEE for breastfed and for breast milk substitute-fed infants from body weight, because of the differences described above, separate regression equations for TEE as a function of weight were derived for these two groups (Butte, 2005). The between individual *coefficient of variation* (CV) of TEE ranged from 15 to 21% (18% average) or from 13 to 17% for TEE/kg (15% average). The equations used to predict TEE from body weight are as follows:

TEE for breastfed infants (FAO, 2004):

TEE (MJ/day) = 0.388 Weight (kg) – 0.635; standard error of estimate (SEE) = 0.453 MJ/day (108kcal/day)

TEE (kcal/day) = 92.8 Weight (kg) – 152; SEE = 0.108

TEE for breast milk substitute-fed infants (FAO, 2004):

TEE (MJ/day) = 0.346 Weight (kg) – 0.122; SEE = 0.463 MJ/day (110kcal/day)

TEE (kcal/day) = 82.6 Weight (kg) – 29.0; SEE = 0.110

Weights substituted in these equations were derived from the WHO Child Growth Standards (WHO, 2006) discussed below (paragraph 101).

Energy deposition

100. The energy stored in new tissue was calculated as the deposited energy accrued during normal growth. These costs were estimated from a multi-component body composition model (total body water, total body potassium and bone mineral content) (Butte *et al.,* 2000b) based on a modified version of Fomon's term infant reference (Fomom *et al.*, 1982) describing changes in body composition during growth. Estimates of protein and fat gain over three month periods were used to predict energy accrued per gram of weight gain, which was then used to predict growth costs at monthly intervals.

Table 3 Energy content of tissue deposition of infants[a]

Age interval (months)	Protein gain (g/d)	Fat mass gain (g/d)	Energy deposited in growing tissues (kJ/g)
Boys			
0-3	2.6	19.6	25.1
3-6	2.3	3.9	11.6
6-9	2.3	0.5	6.2
9-12	1.6	1.7	11.4
Girls			
0-3	2.2	19.7	26.2
3-6	1.9	5.8	15.6
6-9	2.0	0.8	7.4
9-12	1.8	1.1	9.8

[a]Butte, 2005

Gross energy equivalents: 1g protein = 23.6kJ (5.65 kcal); 1g fat = 38.7kJ (9.25kcal)

101. Using this model, the estimate of energy deposited in new tissue fell from about 25.6 kJ/g (6.3 kcal/g) at 0-3 months to about 10.6 kJ/g (2.5 kcal/g) at 9-12 months (see Table 3). These values were applied to the weight velocities observed in the UK-WHO Growth Standards (RCPCH, 2011) to estimate the rates of energy deposition at monthly intervals (see Table 4 for weight velocities). These predictions of energy deposited during growth derive from a relatively small study by Butte *et al* (2000a) which was "validated" against other data sets (Butte, 2005). It is assumed that these values for the energy deposited in new tissue are appropriate for children growing according to the WHO weight velocity values, even though in the original study (Butte *et al,* 2000a) the pattern of breastfeeding followed was not fully described and the growth of infants did not fully reflect the WHO growth trajectory (WHO, 2006). In the study by Butte *et al* (2000a) no significant differences in the body composition of breastfed and breast milk substitute-fed infants were noted, but further information on this point is lacking.

Table 4 Weights, growth and energy deposition rates for infants 1–12 months of age

Age (months)	Weight (kg)[a]	Weight velocity (g/day)[b]	Energy deposition (kJ/g)	Energy deposition (kJ/day)
Boys				
1	4.47	37.1	25.1	931
2	5.56	35.8	25.1	899
3	6.37	26.6	25.1	668
4	7	20.7	11.6	240
5	7.51	16.8	11.6	195
6	7.93	13.8	11.6	160
7	8.3	12.2	6.2	76
8	8.61	10.2	6.2	63
9	8.9	9.5	6.2	59
10	9.16	8.5	11.4	97
11	9.41	8.2	11.4	93
12	9.65	7.9	11.4	90
Girls				
1	4.19	31.6	26.2	828
2	5.13	30.9	26.2	810
3	5.84	23.3	26.2	610
4	6.42	19.1	15.6	298
5	6.9	15.8	15.6	246
6	7.3	13.1	15.6	204
7	7.64	11.2	7.4	83
8	7.95	10.2	7.4	75
9	8.22	8.9	7.4	66
10	8.48	8.5	9.8	83
11	8.72	7.9	9.8	77
12	8.95	7.6	9.8	74

[a] 50th percentile weight for age of the UK-WHO Growth Standards (RCPCH, 2011)
[b] 50th percentile weight increment of the UK-WHO Growth Standards (RCPCH, 2011)

Table 5 Estimated Average Requirement (EAR) values for infants 0–12 months of age

Age (months)	Total energy expenditure (kJ/day)			EAR[e]					
				Breast-fed		Breast milk substitute-fed		Feeding mixed or unknown	
	Breast-fed[a]	Breast milk substitute-fed[b]	Feeding mixed or unknown[c,d]	kJ/day	kJ/kg per day	kJ/day	kJ/kg per day	kJ/day	kJ/kg per day
Boys									
1	1099	1425	1242	2030	454	2356	527	2173	486
2	1522	1802	1647	2421	435	2701	486	2546	458
3	1837	2082	1947	2505	393	2750	432	2615	411
4	2081	2300	2181	2321	332	2540	363	2421	346
5	2279	2476	2370	2474	329	2671	356	2565	342
6	2442	2622	2526	2602	328	2782	351	2686	339
7	2585	2750	2663	2661	321	2826	340	2739	330
8	2706	2857	2778	2769	322	2920	339	2841	330
9	2818	2957	2886	2877	323	3016	339	2945	331
10	2919	3047	2982	3016	329	3144	343	3079	336
11	3016	3134	3075	3109	330	3227	343	3168	337
12	3109	3217	3164	3199	332	3307	343	3254	337
Girls									
1	991	1328	1138	1819	434	2156	514	1966	469
2	1355	1653	1487	2165	422	2463	480	2297	448
3	1631	1899	1751	2241	384	2509	430	2361	404
4	1856	2099	1966	2154	336	2397	373	2264	353
5	2042	2265	2144	2288	332	2511	364	2390	346
6	2197	2404	2292	2401	329	2608	357	2496	342
7	2329	2521	2418	2412	316	2604	341	2501	327
8	2450	2629	2533	2525	318	2704	340	2608	328
9	2554	2722	2634	2620	319	2788	339	2700	328
10	2655	2812	2730	2738	323	2895	341	2813	332
11	2748	2895	2819	2825	324	2972	341	2896	332
12	2838	2975	2904	2912	325	3049	341	2978	333

[a] Total Energy Expenditure (TEE) (MJ/day) = 0.388 Weight (kg) – 0.635
[b] TEE (MJ/day) = 0.346 Weight (kg) – 0.122
[c] These figures should be applied for infants when the mode and proportions of feeding are uncertain
[d] TEE (MJ/day) = 0.371 Weight (kg) – 0.416
[e] Calculated as TEE + energy deposition (kJ/day) as in Table 4

Energy reference values for children and adolescents aged 1-18 years

102. The energy reference values for boys and girls aged 1-18 years were calculated as TEE plus deposited energy costs using a factorial model BMR x PAL, with PAL adjusted for growth in terms of a 1% increase (see paragraph 108).

Estimating the BMR

103. The BMR values for children and adolescents were estimated from the Henry prediction equations (Henry, 2005) (see Appendix 4 for details), using weights and heights indicated by the 50^{th} centiles of the UK-WHO Growth Standards (RCPCH, 2011) for ages 1 to 4 years and the UK 1990 reference for children and adolescents (Freeman *et al.*, 1995) for ages 5 to 18 years.

Identifying PAL values

104. PAL values were identified from an analysis of DLW measures of TEE. The objective of the analysis was to identify specific age ranges of children within which variation with age was less than variation between individuals. This approach would allow PAL to be defined for these age-range groups in terms of its distribution: i.e. as the median, 25^{th} and 75^{th} centile range values. The analysis also examined the need to identify gender-specific PAL values.

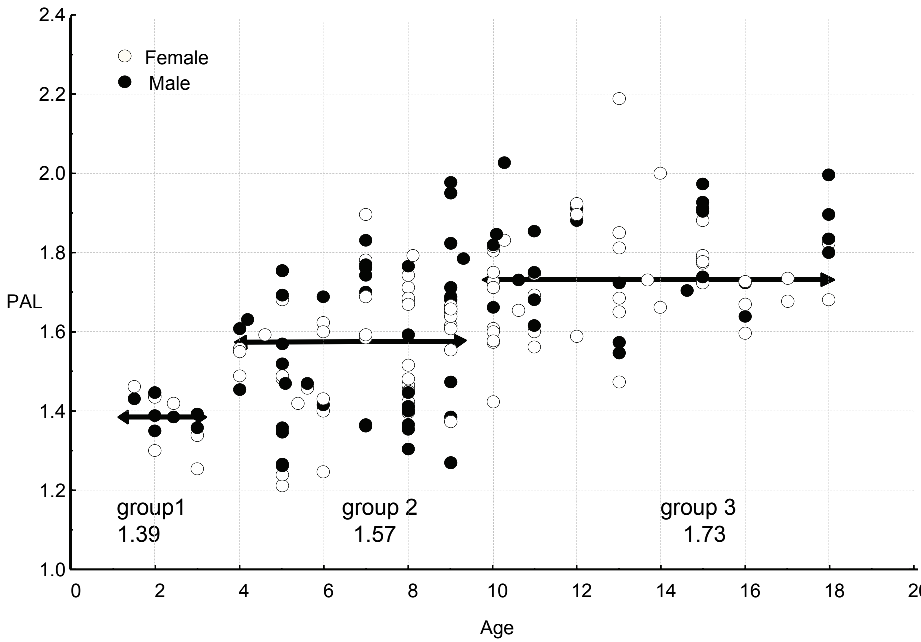
105. A data set was compiled of all published DLW studies of children aged over one year (see Appendix 12 for details). All studies were tabulated according to mean values for boys and girls for specific age groups. This resulted in 170 data points as study means representing a total of 3502 individual measurements (2082 females, 1420 males).

106. For all studies which did not report BMR, BMR values were calculated from the Henry equations for weight and height or weight alone if no height was reported (see Appendix 4). PAL values were calculated from TEE and either the reported or calculated BMR values.

107. The analysis revealed no influence of gender but an increase in PAL values with age as shown in Figure 2. From an early age, however, there was a wide range of study mean PAL values so that variation in PAL at any age was much greater than variation with age itself. Indeed, on the basis of the association of PAL values with activity levels (as shown in Table 22 Appendix 5), many of the study mean PAL values for younger school children (PAL<1.5) would imply very low activity levels compared with other children of the same age. Although one option was to exclude some of these studies, there was insufficient evidence to do this and all studies shown in Figure 2 were therefore included in the analysis. Thus, three age groups were identified within which the distribution of PAL values could be identified: 1-3 years, >3-<10 years and 10-18 years. To some extent, these age ranges reflect important periods of growth and development that could influence behaviour and energy requirements. These age ranges also correspond to the age ranges within which BMR prediction equations have been generated for both the Schofield and Henry

prediction equations. Median, 25th centile and 75th centile PAL values were identified for each of these groups and are shown in Table 6.

Figure 2 – Physical Activity Level (PAL) values for children and adolescents (aged 1-18 years) as a function of age.



Median values for the indicated age ranges are shown. Each point represents a single age group reported in a single publication or in publications which list mean values for each of several age groups. Boys and girls are shown separately. The publications from which these data points and the mean values in Table 6 were derived are detailed in Table 37, Appendix 12.

Table 6 Physical Activity Level (PAL) values for the age groups 1-3, >3-<10 and 10-18 years derived from the DLW data from studies of children aged >1 year[a]

Age group (years)	Age (years)					PAL								
	N	mean	sd	Min	Max	mean	sd	Min	10th centile	Q25	Median	Q75	90th centile	Max
1-3	14	2.3	0.54	1.5	3.0	1.39	0.06	1.26	1.30	1.35	**1.39**	1.43	1.45	1.46
>3-<10	85	7.0	1.87	4.0	9.3	1.56	0.14	1.21	1.35	1.42	**1.57**	1.69	1.77	1.98
10-18	71	13.0	2.38	10.0	18.0	1.75	0.13	1.42	1.58	1.66	**1.73**	1.85	1.91	2.19

[a] PAL values calculated from DLW-derived TEE and measured or estimated BMR (using Henry equations).

Adjusting for growth

108. The gross energy deposited in new tissue should be added to the energy expended as a cost of tissue deposition. Only the latter is part of the observed TEE. Deposited energy accounts for a relatively small overall proportion of the total energy needs of children at all ages after the first year of life (see Appendix 12). In the context of a factorial model of requirements, the simplest approach is an adjustment of PAL, as suggested by FAO/WHO/UNU (FAO, 2004), which in effect represents growth as a fixed proportion of the overall energy requirement. Growth costs calculated as a percentage of energy requirement are on average (1-16 years of age) 0.98% (min=0.4%, boys, 0.05%, girls; max= 1.37%, boys, 1.59%, girls). This indicates that a single average growth value calculated as 1% of energy requirements could be used throughout the age range. Use of this 1% value will result in underestimation of overall energy requirements during the peak growth phase for older children of up to 0.6% for girls or 0.4% for boys. There will be an overestimate by similar amounts as growth slows in late adolescence. Such errors were not viewed to be significant given the overall variation in PAL values between children. In this report growth costs were therefore accounted for by a 1% adjustment of PAL values for each age group. These adjusted values are shown in Table 7.

Table 7 Physical Activity Level (PAL) values for use in calculation of energy requirements of children and adolescents, adjusted for growth

Age group (years)		PAL[a]	
	Q25[c]	Median[b]	Q75[c]
1-<3	1.36	**1.40**	1.45
3-<10	1.43	**1.58**	1.70
10-18	1.68	**1.75**	1.86

[a] PAL adjusted for growth (=PALx1.01)

[b] Population Estimated Average Requirement (EAR) estimates for children and adolescents with average levels of physical activity

[c] 25th and 75th centile PAL values used to calculate energy requirements for children with lesser or greater activity levels

Calculating energy reference values for children and adolescents

109. Energy reference values can be calculated for boys and girls aged 1-18 years as adjusted PAL x BMR. BMR values are estimated from the Henry equations (see Appendix 4), using weights and heights indicated by the 50th centiles of the UK-WHO Growth Standards (RCPCH, 2011) (ages 1 to 4 years) and the UK 1990 reference for children and adolescents (Freeman *et al.*, 1995). Growth-adjusted PAL values representing the 25th (less active), median and 75th (more active) centiles of the PAL distributions for each age group (1-<3, 3-<10 and 10-18 years), are then used to calculate energy reference values for boys and girls, for each year (see Table 8). The reference weights used represent best estimates of healthy body weights and are about 15% lower than current UK weights (see Appendix 10). Thus, for those children who are overweight and for any underweight children, energy intakes at these levels will be associated with weight change.

110. Although it is difficult to predict and make judgements about the different activity patterns likely to be associated with the three PAL bands shown in Table 8, it can be assumed that the 75th centile "more active" PAL value will represent a more desirable level of physical activity for the maintenance of health and that the 25th centile "less active" PAL value will represent a less desirable level.

Table 8 Estimated Average Requirement (EAR) values for energy based on median weights and heights from the WHO growth standards[a] (ages 1 to 4) and the UK 1990 reference for children and adolescents[b] for children aged >4 years

	Boys			Girls			Energy requirements MJ/d					
							Boys			Girls		
Age (years)	Weight kg	height cm	BMR[c] MJ/d	Weight kg	height cm	BMR[c] MJ/d	less active[d]	Population[e]	more active[f]	less active[d]	Population[e]	more active[f]
1	9.6	76	2.29	9.0	74	2.12	3.1	**3.2**	3.3	2.9	**3.0**	3.1
2	12.2	87	3.02	11.5	86	2.78	4.1	**4.2**	4.4	3.8	**3.9**	4.0
3	14.4	97	3.46	13.9	96	3.23	4.7	**4.9**	5.0	4.4	**4.5**	4.7
4	16.3	104	3.67	16.0	103	3.43	5.3	**5.8**	6.3	4.9	**5.4**	5.8
5	18.6	110	3.89	18.2	109	3.63	5.6	**6.2**	6.6	5.2	**5.7**	6.2
6	21.0	117	4.14	21.0	117	3.88	5.9	**6.6**	7.1	5.6	**6.2**	6.6
7	23.0	123	4.34	23.0	123	4.07	6.2	**6.9**	7.4	5.8	**6.4**	6.9
8	26.0	129	4.61	26.0	129	4.32	6.6	**7.3**	7.9	6.2	**6.8**	7.4
9	29.0	134	4.87	29.0	134	4.57	7.0	**7.7**	8.3	6.5	**7.2**	7.8
10	31.5	138	4.83	32.0	139	4.63	8.1	**8.5**	9.0	7.8	**8.1**	8.6
11	34.5	143	5.09	35.9	144	4.84	8.5	**8.9**	9.5	8.1	**8.5**	9.0
12	38.0	149	5.38	40.0	149	5.05	9.0	**9.4**	10.0	8.5	**8.8**	9.4
13	43.0	155	5.77	46.0	154	5.34	9.7	**10.1**	10.8	9.0	**9.3**	10.0
14	49.0	164	6.26	51.0	160	5.60	10.5	**11.0**	11.7	9.4	**9.8**	10.4
15	55.5	170	6.75	53.0	162	5.70	11.3	**11.8**	12.6	9.6	**10.0**	10.6
16	60.2	173	7.09	55.3	163	5.80	11.9	**12.4**	13.2	9.7	**10.1**	10.8
17	64.0	175	7.36	57.0	163	5.87	12.3	**12.9**	13.7	9.8	**10.3**	10.9
18	66.2	176	7.52	57.2	163	5.88	12.6	**13.2**	14.0	9.9	**10.3**	11.0

[a] RCPCH, 2011

[b] Freeman *et al*., 1995

[c] Calculated from the Henry equations based on weight and height (see Appendix 4).

Because the Henry equations have overlapping age bands (0-3, 3-10, 10-18 years), choice of equation is ambiguous at the age boundaries. Here the BMR equation for 3-10 year olds is used for the 3 year olds and the equation for 10-18 year olds is used for those aged 10 years on the basis of a smoother transition in the plot of BMR/kg against age.

[d] Calculated with the 25th centile PAL value adjusted for growth shown in Table 7.

[e] Calculated with the median PAL value adjusted for growth shown in Table 7.

[f] Calculated with the 75th centile PAL value adjusted for growth shown in Table 7.

Energy reference values for adults

111. The approach adopted for the determination of energy reference values for adults was to utilise a factorial model based on BMR x PAL. BMR is estimated for healthy body weights, i.e. weights equivalent to a BMI of 22.5 kg/m^2 at the height of the population group (as discussed in paragraph 92). Values listed are the current mean heights (for England and Scotland[24]) at various ages. The PAL values are identified from an analysis of suitable DLW measures of TEE.

Estimating BMR

112. As already indicated there are several different equations currently used to estimate BMR (see paragraphs 80 and 81, and Appendix 4 for details). In this report, the prediction equations of Henry (2005) have been used.

Identifying PAL values

113. The approach taken to determine PAL values was to identify a data set of DLW measures of TEE which could serve as a reference distribution of TEE and PAL values from which energy reference values for the UK adult population could be predicted. An initial survey of all published and other available DLW measures of TEE in healthy adults identified published studies which could be utilised in terms of study means. Two sets of individual data points were also considered:

- a DLW data set of individual values drawn from the NDNS (n=66) (Henderson *et al.*, 2003a) and Low Income Diet and Nutrition Survey (n=36) (Nelson *et al.*, 2007), and values from the unpublished NDNS comparison study (n= 90 adult)
- the DLW data set (n=767) assembled for the US DRI report (IoM, 2005) which includes most of the UK studies published up to the writing of that report.

114. With the exception of the NDNS data sets, subjects were not recruited to these studies explicitly as a representative sample of the UK or any other adult population; NDNS randomly selects participants to be representative of the UK population while recognising that there may be recruitment bias into the DLW subsample (as discussed in paragraph 71). While the NDNS data sets are most appropriate, these are currently too small to serve as a reference. All of the published DLW studies included a wide range of BMI and a reasonable age distribution, but several involved investigations of physical activity measurement devices (e.g. accelerometers) and specifically recruited subjects following relatively high activity lifestyles. Although these studies illustrated the overall extent of the variation in PAL values, especially its upper and lower limits and its likely response to changes associated with specific activity programmes, the suitability of a combined data set was a cause for concern.

115. Subsequent to publication of the DRI report in 2005, two large population-based studies of energy expenditure measured using DLW have been published, the OPEN study (Subar *et al.*, 2003; Tooze *et al.*, 2007) and the Beltsville study

24 Representative data for Wales and Northern Ireland were not available.

(Moshfegh *et al.,* 2008) (see Appendix 8 for details). The OPEN study involved healthy volunteers (n=451; 245 men and 206 women) aged 40–69 years; about 85% of volunteers were white with the remainder mainly black or Asian. The Beltsville study involved healthy volunteers (n= 478) aged 30–69 years. The subjects were predominately non-Hispanic white and were distributed evenly by sex and approximately by age. Both studies comprised an urban population with subjects recruited from the Washington DC metropolitan area. The combined OPEN and Beltsville cohorts contained similar numbers of men (48%) and women (52%), with levels of overweight and obesity very similar to current UK levels reported in the UK Health Surveys (see Appendix 10). Thus, mean BMI values were 27.0 kg/m^2 (F), 27.5 kg/m^2 (M) compared with current UK values of 27kg/m^2 (M and F); 39% were classified as overweight and 25% obese, compared with 38% overweight and 23% obese in England in 2009 (NHS IC, 2010). From the perspective of ethnic mix and body weights, this population is therefore similar to the current UK population. However, no objective measures of physical activity were made in either study so it is not known how representative the distribution of TEE and PAL values is of the UK population. Individual PAL values from both studies were made available for this report[25]. BMR was measured in the Beltsville study but not in the OPEN study, so in the latter case BMR has been calculated using the Henry BMR prediction equations (Henry, 2005) based on weight and height. As shown in Figure 9 (Appendix 8), predicting BMR for an overweight/obese population did not appear to introduce bias since the regressions of PAL on BMI for the two cohorts were very similar.

Table 9 Distribution of Physical Activity Level (PAL) values in the Beltsville and OPEN studies[a,b]

Distribution boundaries	OPEN (n=451; age 40-69 years)	Beltsville (n=478; age 30-69 years)
Minimum	1.01	1.01
10^{th} centile	1.40	1.32
lower quartile	1.49	1.46
median	1.61	1.62
upper quartile	1.77	1.78
90^{th} centile	1.92	1.96
Maximum	2.61	2.34

[a] Beltsville data set (Moshfegh *et al*, 2008)
[b] OPEN data set (Subar *et al*., 2003; Tooze *et al*., 2007)

116. The distribution of PAL values within the combined data set (n=929 individual measures), was 1.01 to 2.61, as shown in Table 9. Investigation of the range of PAL values observed in healthy, mobile individuals with overall levels of physical activity which are sustainable, indicated a range of 1.38-2.5 (see Appendix 5) with a value of 1.27 representing a minimal survival requirement: i.e. minimal movement in waking hours as suggested by FAO/WHO/UNU in 1985 (WHO, 1985). Thus, values below 1.27 can be assumed to reflect either methodological error or to be non ambulatory

25 TEE data from the OPEN study were obtained from Amy Subar. TEE data from the Beltsville study was provided by Alanna Moshfegh.

individuals, highly dependent on others, or those with an unsustainable lifestyle. Similarly, subjects with PAL values >2.5 can be assumed to be participating in very high activity levels during the measurement period which are unrepresentative of usual activity. The combined data set was therefore trimmed for PAL values ≤1.27 or >2.5. This removed one subject with a PAL value >2.5 and 38 subjects with PAL values <1.27. However, the trimming had a minimal influence on the distribution characteristics increasing the median PAL value from 1.62 to 1.63 and the mean PAL value from 1.64 to 1.66 (see Table 10). The skewed distribution with subjects clustered at the lower end of the range is clearly apparent in Figure 3. The PAL values selected to predict energy reference values for the adult population are highlighted. The use of the 25th and 75th centile values is discussed below (see paragraph 119-122).

117. The derivation of EAR values using PAL values from a predominantly overweight and obese population was carefully considered given the objective of identifying prescriptive EAR values consistent with long term health: i.e. values suitable for weight maintenance at a healthy body weight and at existing physical activity levels. For those subjects within the normal body weight range i.e. BMI 18.5 to 24.9kg/m^2 (n=322, 36% total), the median PAL (1.61) was not significantly different from that of the overweight or obese subjects for men or women (see Table 30, Appendix 8). Furthermore, investigation of the influence of BMI on PAL values within the combined data set by regression analysis indicated that PAL did not significantly vary with BMI (p=0.64 for slope: R^2 less than 0.1%: see Appendix 8 for detail). This implies that the rate of energy expenditure, as indicated by the median PAL for the cohort (i.e. 1.63), is not a specific characteristic of overweight or obesity and its use in deriving prescriptive EAR values consistent with healthy body weights is justifiable.

118. Regression analysis did show that PAL values decrease slightly with age. However, age explained <1% of the variance (R^2 =0.004), and since the slope is shallow, age has a minor influence on PAL, i.e. PAL = 1.69 at 30 years and 1.63 at 70 years. This indicates that energy reference values can be defined independently of age at least to the age of 70 years.

Table 10 Physical Activity Level (PAL) value statistics for the combined Beltsville and OPEN data sets[a,b]

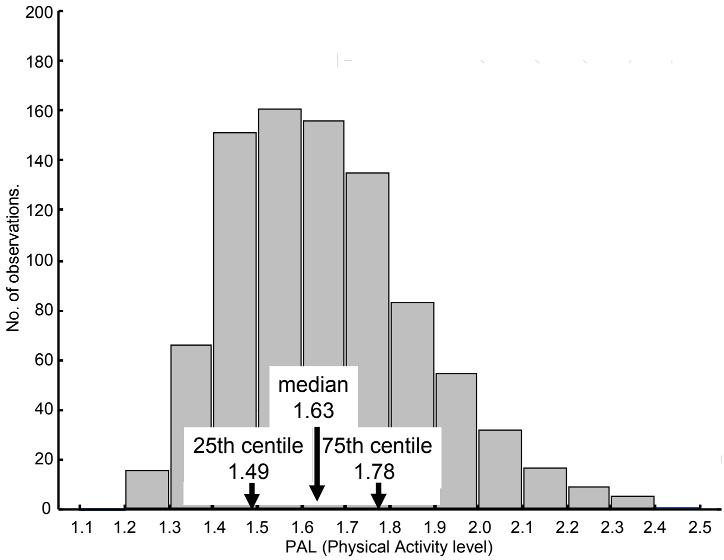
	N	Mean	SD	Min	10th centile	25th centile	Median	75th centile	90th centile	Max
All	929	1.64	0.23	1.01	1.36	1.48	1.62	1.78	1.95	2.61
Trimmed[c]	890	1.66	0.21	1.27	1.40	**1.49**	**1.63**	**1.78**	1.96	2.50

[a] Beltsville data set (Moshfegh *et al*, 2008)

[b] OPEN data set (Subar et al., 2003; Tooze *et al*., 2007)

[c] Subjects with PAL values < 1.27(n=38) and >2.5 (n=1) excluded

Figure 3 – Distribution of Physical Activity Level (PAL) values for trimmed combined OPEN and Beltsville data sets[a,b]



[a] Beltsville data set (Moshfegh *et al*, 2008)
[b] OPEN data set (Subar et al., 2003; Tooze *et al*., 2007)

Calculating energy reference values

119. Tables 11 and 12 show energy reference values for men and women, respectively. Specific values of TEE and the consequent EAR are calculated from BMR x PAL as a function of age, gender, height and BMI. BMR is calculated with the Henry equations for healthy body weights equivalent to a BMI of 22.5kg/m^2. For illustrative purposes, current mean heights of the population are shown for the population at the various age ranges as indicated by the Health Surveys for England (data for 2009) (NHS IC, 2010). PAL is independent of gender and the change with age is too small to have any significant influence. Thus, a single set of PAL values calculated as the median (1.63), 25th (1.49) and 75th (1.78) centile boundaries of the reference population is used (see Table 31 and Appendix 8).

120. In the absence of other information on activity, the population EAR values are those derived using the median PAL (1.63) as the assumed population activity level. Reference values for population groups of men and women, thought to be less or more active than average, are those calculated from the 25th (1.49) and 75th (1.78) centile boundary PAL values and the BMR values shown in Tables 11 and 12. For population groups of different heights, weights can be calculated at a BMI of 22.5 kg/m^2, as weight = 22.5 x height^2. BMR can be calculated from the Henry (2005) equations (see Appendix 4), and population EAR values can be calculated as BMR x PAL (see Tables 11 and 12).

Table 11 Estimated Average Requirement (EAR) values for energy for groups of men at various ages, weights and physical activity levels[1], at current mean height for age and a Body Mass Index (BMI) of 22.5 kg/m^2

Age range (years)	Height[a] cm	Weight[b] kg	BMR[c] MJ/day	EAR MJ/d less active[d]	EAR MJ/d population[e]	EAR MJ/d more active[f]
19-24	178	71.5	7.1	10.6	11.6	12.6
25-34	178	71.0	7.1	10.5	11.5	12.6
35-44	176	69.7	6.7	10.0	11.0	12.0
45-54	175	68.8	6.7	9.9	10.8	11.8
55-64	174	68.3	6.6	9.9	10.8	11.8
65-74	173	67.0	6.0	9.0	9.8	10.7
75+	170	65.1	5.9	8.8	9.6	10.5
all adults	175	69.2	6.7	10	10.9	11.9

[a] Values for illustration derived from mean heights reported in the Health Survey for England 2009 (NHS IC, 2010)

[b] At BMI= 22.5 kg/m^2: i.e. weight = 22.5 x height^2

[c] Calculated from the Henry prediction equations based on weight and height, where BMR = coefficient x weight (kg) + coefficient x height (m) + constant (see Appendix 4)

[d] 25th centile Physical Activity Level (PAL) =1.49

[e] Median PAL= 1.63,

[f] 75th centile PAL=1.78

Table 12 Estimated Average Requirement (EAR) values for energy for groups of women at various ages and physical activity levels, at current mean height for age and a Body Mass Index (BMI) of 22.5 kg/m^2

Age range (years)	Height[a] cm	Weight[b] kg	BMR[c] MJ/day	EAR MJ/d less active[d]	EAR MJ/d population[e]	EAR MJ/d more active[f]
19-24	163	59.9	5.6	8.4	9.1	10.0
25-34	163	59.7	5.6	8.3	9.1	10.0
35-44	163	59.9	5.4	8.1	8.8	9.7
45-54	162	59.0	5.4	8.0	8.8	9.6
55-64	161	58.0	5.3	7.9	8.7	9.5
65-74	159	57.2	4.9	7.3	8.0	8.7
75+	155	54.3	4.7	7.0	7.7	8.4
all adults	162	58.7	5.4	8	8.7	9.5

[a] Values for illustration derived from mean heights reported in the Health Survey for England 2009 (NHS IC, 2010)

[b] At BMI= 22.5 kg/m^2: i.e. weight = 22.5 x height^2

[c] Calculated from the Henry prediction equations based on weight and height, where BMR = coefficient x weight (kg) + coefficient x height (m) + constant (see Appendix 4)

[d] 25th centile Physical Activity Level (PAL) =1.49

[e] Median PAL= 1.63,

[f] 75th centile PAL=1.78

121. The EAR values shown in Tables 11 and 12 have been calculated for subjects with healthy body weights equivalent to a BMI of 22.5kg/m^2, the lower end of the body weight range associated with minimum mortality (see paragraph 92). For subjects with BMI values greater than 22.5kg/m^2, energy intakes at these EAR levels would be associated with weight change towards a healthier body weight.

122. As with the EAR reference values for children, although it is difficult to predict and make judgements about the different activity patterns likely to be associated with the three PAL bands shown in Tables 11 and 12, it can be assumed that the 75th centile "more active" PAL value will represent a more desirable level of physical activity for the maintenance of health and that the 25th centile "less active" PAL value will represent a less desirable level.

Energy reference values for older adults

123. Age-related changes in lifestyle and activity are very variable and there is little evidence that older people have decreased physical activity while they remain mobile and in good health. In a cohort (n= 302) of community-dwelling US older adults (aged 70-82 years) (Manini *et al.*, 2006) who are described as high-functioning, able to independently perform activities of daily living, and with no evidence of life-threatening illnesses, a wide variation of PAL values was observed. The mean PAL values for tertiles of PAEE were 1.48, 1.68 and 1.94. The overall mean PAL value for this group, 1.70, was slightly higher than that of the combined OPEN and Beltsville cohort of younger adults (mean PAL =1.64).

124. In advanced age, PAL values can be very low. In a group of 21 Swedish men and women aged 91-96 years of age, all free- and independently-living, and termed healthy, all living a quiet life (some not having been out of doors for years), PAL values were on average 1.38 (1.13-1.65 after trimming for PAL values <1.1 on the basis of comments that BMR used as the divisor of TEE to calculate PAL may have been overestimated in some cases (Rothenberg *et al.*, 2000)). Another study in free-living British men (n=23; all over 75 years of age) observed a mean PAL value of 1.5 (Fuller *et al.*, 1996).

125. Taken together, these data indicate that it is difficult to generalise about the energy requirements of older adults other than the requirements are unlikely to differ from younger adults whilst general health and mobility are maintained. However, in advanced age for those individuals with much reduced mobility it can be assumed that PAL values are likely to be lower. For these individuals and for older people who are not in good general health, energy requirements can be based on the less active, 25th centile PAL value of 1.49, recognising that for some groups of older people with specific diseases or disabilities or for patient groups who are bed-bound or wheelchair bound, the PAL value may be consistently lower than this.

Energy reference values for children, adolescents and adults outside the expected range of habitual activities

126. The PAL values identified in the preceding sections have not been derived in relation to any specific level of physical activity or lifestyle because, as discussed

in Appendix 5, it has proved very difficult to predict the PAL values of individuals as a function of lifestyle or even measured physical activity. The median PAL values for children (see Table 7) and adults (see Table 10) represent the midpoint of the distribution observed in the reference population and, as such, provide the best estimate of the average activity level for the population. In the absence of any other information on activity this value is the assumed population level and is used to calculate the population EAR. Based on the literature (as summarised in Table 22, Appendix 5), the median PAL values can probably be judged to represent a light activity lifestyle for school children and adults. The 25th and 75th centile PAL values have been identified for subject groups who are less or more active than the average and probably represent a sedentary, less desirable or moderate activity, more desirable lifestyle. The difference between these centile values and the median is 9% for adults and for children aged >3-<10 years, 5% for adolescents and only 3% for the youngest children. These values can be compared with the slightly greater range of ± 10% identified as the basis for the higher or lower than average activity levels by FAO/WHO/UNU in their analysis of energy requirements of children and adolescents (FAO, 2004). Judging when to assign subjects with the 25th less active and 75th more active centile values as opposed to the median population PAL values poses a difficult problem. As shown in Table 13, the difference of ±0.15 PAL units from the median for adults corresponds to ±30 minutes of moderate intensity activity five times per week, all other activities being the same. However, to date, it has not proved possible to identify from questionnaires or activity logs the actual behavioural differences which determine such variation in TEE in terms of identifiable exercise activities. Hopefully this will improve with the increasing use of objective methods of physical activity assessment (e.g. accelerometers) which can measure and record a much wider range of human movement including 'incidental' movement not captured by questionnaires.

Effects of additional physical activity on energy requirements

127. Although the prediction of PAL values associated with a specific lifestyle cannot be made with any certainty, predictions of the likely additional energy cost of well defined specific activities can be made with reasonable confidence (see Appendix 5). Estimates of the likely changes in PAL for a range of activities derived from theoretical calculations and from observed effects to the change in PAL for ten minutes and one hour of activity, or following the adoption of a high level physical activity training programme, are summarised in Appendix 5. Some examples are shown in Table 13. These examples are the likely increases in TEE (and hence requirements) expressed as the change in PAL for the listed activities for individuals leading lives with little physical activity, but maintaining body weight, and provided that no compensatory reduction in other activities or increase in energy intake occurs. These values have been derived primarily from studies on adults, but there is no reason to believe that they will be substantially different for children and adolescents.

Table 13 Examples of changes in Physical Activity Level (PAL) associated with increased activity (IoM, 2005)[a,b]

Change in PAL	Activity
0.15	30 minutes of moderate intensity activity on 5 or more days of the week[c]
0.2	60 minutes brisk walking (brisk =>6<7.5kmph, (=>4<5mph)) daily
0.3	60 minutes of active sport, (i.e. jogging at 9km/hr (6mph)), 5 times per week
0.4	60 minutes jogging at 9km/hr (6 mph) daily
0.6	An intense aerobic exercise programme associated with training for competitive sport daily

[a] More examples are shown in Appendix 5

[b] IoM, 2005

[c] Equivalent to previous recommendations from the Chief Medical Officer for England regarding physical activity in adults (DH, 2004)

Reference energy values during pregnancy and lactation

128. The energy requirements for pregnancy and lactation are calculated as increments to be added to the mother's EAR. These are based on singleton pregnancies reaching term.

129. Ideally, women should begin pregnancy at a healthy body weight (BMI 18.5-24.9 kg/m^2); the EARs for non-pregnant women identified in this report are set at amounts consistent with maintaining a BMI of 22.5kg/m^2 (see paragraphs 90-93). Women who are overweight or underweight at the beginning of pregnancy are at risk of poor maternal and fetal outcomes (Han *et al.*, 2011; McDonald *et al.*, 2010; March of Dimes, 2002; Stothard *et al.*, 2009). For such women, the relationships between weight change during pregnancy and fetal outcome are not completely understood. Given this uncertainty, a precautionary approach has been adopted and weight loss during pregnancy is not advised (NICE, 2010). The EARs for pregnancy and lactation defined in this report are therefore estimates of the incremental energy intakes likely to be associated with healthy outcomes for mother and child. These incremental energy intakes should be added to EAR values calculated at actual preconceptional body weights, rather than at healthy body weights for non-pregnant women.

130. The energy requirements for pregnancy also need to take into account the protection of vulnerable groups. Adolescents who become pregnant must meet the dietary requirements imposed by growth, in addition to the demands of pregnancy and lactation. This is a complex issue. For example, consuming the extra energy needed to cover the costs of pregnancy does not in itself guarantee a better outcome (Kramer & Kakuma, 2003). Also, those under 18 years of age are at greater risk than older women of giving birth to infants who are of low birth weight by virtue of pre-term delivery or small size for gestational age (FAO, 2004). Thus, pregnancies up to 18 years are qualitatively different and must be considered differently.

Energy costs of pregnancy

131. The energy costs associated with the maintenance of a normal pregnancy arise from increases in maternal and feto-placental tissue mass, the rise in energy expenditure attributable to increased basal metabolism and changes in the energy cost of physical activity. Additional energy is also required to ensure energy stores are sufficient to support adequate lactation following delivery (Butte & King, 2005).

Gestational weight gain

132. Gestational weight gain (GWG) is the major determinant of the incremental energy needs during pregnancy. GWG determines not only energy deposition, but also the increase in BMR and the increase in TEE resulting from the energy cost of moving a larger body mass. The WHO Collaborative Study on Maternal Anthropometry and Pregnancy Outcomes (WHO, 1995) reported birth weights and maternal weight gains associated with lower risk of fetal and maternal complications. Birth weights between 3.1 and 3.6 kg (mean, 3.3 kg) were associated with the optimal ratio of maternal and fetal health outcomes. The range of GWG associated with birth weights greater than 3 kg was 10–14 kg (mean, 12 kg).

133. The tissue deposited during pregnancy includes the products of conception (fetus, placenta, and amniotic fluid), maternal tissues (uterus, breasts, blood, and extracellular fluid) and maternal energy reserves as fat. A theoretical model has previously been used to estimate the energy requirements during pregnancy (Hytten & Chamberlain, 1991). This assumed an average GWG of 12.5 kg (0.9 kg protein, 3.8 kg fat, and 7.8 kg water), an efficiency of energy utilization of 90% and a mean birth weight of 3.4 kg. The total energy cost of pregnancy was estimated to be about 330 MJ (80,000 kcal) (WHO, 1985). Since publication of this model in 1991, a number of longitudinal studies in developed and developing countries have facilitated the revision of these theoretical estimates.

134. Longitudinal studies of body composition during pregnancy in well-nourished[26] women from the UK, USA, Netherlands and Sweden have observed a mean GWG of 11.9 kg at 36 weeks gestation. Extrapolating the calculations to 40 weeks of gestation suggests a total mean weight gain of 13.8 kg (Butte & King, 2005).

Basal metabolism in pregnancy

135. In studies of healthy, well-nourished women with adequate weight gain during pregnancy who gave birth to infants with adequate weights, average increases in BMR over pre-pregnancy values have been observed to be around 5, 10 and 25% in the first, second and third trimesters, respectively (Butte & King, 2005). There is, however, considerable variation in the cumulative increase in BMR (Prentice *et al.*, 1996b).

136. In under-nourished populations such as The Gambia, adaptive changes in BMR, and reduction in the amount of additional maternal fat stored during gestation,

26 The terms 'well-nourished' and 'under-nourished' are generally not defined in the original studies which inform reference energy values during pregnancy and lactation.

can make a profound difference to the overall energy needs of pregnancy (Prentice & Goldberg, 2000). The extent of adaptive changes in BMR that occur in well-nourished populations is unclear, but the increase in BMR during pregnancy has been observed to vary in response to pre-pregnancy body fat content. Larger increases in BMR have been observed in those having a higher percent fat mass, while increased energetic efficiency in the basal state (i.e. a depressed BMR) has been observed in thinner women (Butte *et al.*, 2004; Prentice *et al.*, 1996b).

Total energy expenditure in pregnancy

137. The TEE of pregnancy has been measured longitudinally using DLW techniques in well-nourished, free-living women in Sweden, the UK and the USA (Butte *et al.*, 2004; Forsum *et al.*, 1992; Goldberg *et al.*, 1991a, 1993; Kopp-Hoolihan *et al.*, 1999). TEE increased throughout pregnancy in proportion to the increase in body weight. TEE increased by about 1, 6 and 19%, and weight increased by 2, 8, and 18% over baseline in the first, second and third trimesters, respectively. The estimated increments in TEE (0.1 (25), 0.4 (95) and 1.5 (360) MJ/day (kcal/day) in the first, second and third trimesters, respectively) are similar to the increments observed by 24-hour calorimetry (Butte & King, 2005). The average GWG was 13.8 kg, but for a mean GWG of 12 kg (observed in the WHO collaborative study (WHO, 1995)) the corresponding values would be 0.08 (20), 0.35 (85) and 1.30 (310) MJ/day (kcal/day).

138. In the latter half of pregnancy, increases in body weight result in increased energy costs for activities; however, women may compensate for this by reducing the pace or intensity with which the activity is performed (Butte & King, 2005; Prentice *et al.*, 1996b). The extent to which women are able to modify habitual physical activity patterns during pregnancy will be determined by socioeconomic and cultural factors specific to the population; women who are sedentary prior to pregnancy will have little flexibility to reduce their level of physical activity further.

139. Changes in PAEE (TEE–BMR) during pregnancy are highly variable, but when measured longitudinally by DLW in well-nourished women averaged -2, +3 and +6% in the first, second and third trimesters, respectively, relative to pre-pregnancy values (Butte & King, 2005). Because of the larger increment in BMR, PAL declined from 1.73 prior to pregnancy to 1.60 in late gestation in well-nourished women (Butte & King, 2005).

140. In another study of women of varying pre-pregnancy BMI[27], significant reductions in PAEE and PAL were also observed in all BMI groups as pregnancy progressed, while BMR increased gradually throughout pregnancy (Butte *et al.*, 2004). The same study found that excessive GWG was mainly due to fat mass gain and not protein accretion (Butte *et al.,* 2003a): GWG within the IoM recommendations (Institute of Medicine Food and Nutrition Board, 1990) was associated with appropriate birth weights and moderate postpartum fat retention; whereas women who gained weight above the IoM recommendations had significantly higher excessive

27 Participants were grouped into *healthy* underweight (BMI<19.8), normal weight (BMI 19.8-26.0) and overweight (BMI >26.0) according to 1990 Institute of Medicine recommended GWG ranges (Institute of Medicine Food and Nutrition Board, 1990).

fat retention at 27 weeks postpartum. Calculation of total energy costs indicate that due to the higher GWGs, maternal fat deposition, and increments in BMR, increases in dietary energy intakes are required as pregnancy progresses; increases in BMR and energy deposited in maternal and fetal tissues are not fully offset by reductions in physical activity. Studies such as this highlight the problems which can arise when energy costs of pregnancy are based on the sum of TEE and energy deposition, since the latter may reflect excessive fat deposition which is retained postpartum.

Calculation of energy requirements for pregnancy

141. As discussed above, the energy cost of pregnancy is not evenly distributed over the gestational period and this must be considered when calculating the energy requirements for pregnancy (Butte & King, 2005). Most weight is gained in the second and third trimesters, at rates of 0.45 kg per week and 0.40 kg per week respectively, compared with a GWG of 1.6 kg in the whole first trimester. The increases in BMR and TEE are most pronounced in the second half of pregnancy.

142. The total energy cost of pregnancy can be estimated both from the increment in BMR and energy deposition, and from the increment in TEE and energy deposition. These two factorial approaches provide slightly different distributions, but when applied to the WHO Collaborative Study on Maternal Anthropometry and Pregnancy Outcomes mean GWG of 12 kg (WHO, 1995), the estimated extra energy cost of pregnancy is 321 MJ (77,000 kcal). This is divided into approximately 0.35 (85), 1.2 (285) and 2.0 (475) MJ/day (kcal/day) for the first, second and third trimesters, respectively (FAO, 2004). This is based on the assumption that increments in BMR and TEE are proportional to gestational weight gain.

143. Most dietary studies in well-nourished women, however, have revealed no or only minor increases in energy intake that only partially covered the estimated energy cost of pregnancy. An analysis of available data from longitudinal studies in populations with average birth weights greater than 3 kg revealed a cumulative reported intake of only 85 MJ (around 20,000 kcal; or 0.3 MJ/day, 72kcal/day) over the whole of pregnancy (Prentice *et al.*, 1996b). This is only 25% of the estimated needs or about 40% if allowances are made for under-reporting of energy intake[85].

Approaches taken in other reports

144. The FAO/WHO/UNU report (FAO, 2004) recommends an increase in food intake of 1.5 MJ/day (360 kcal/day) in the second trimester and 2.0 MJ/day (475 kcal/day) in the third, based on a GWG of 12 kg and specific for women in societies with a high proportion of non-obese women who do not seek prenatal advice before the second and third month of pregnancy. Energy deposited as fat represents 45% of these energy costs.

145. The 1.5 MJ/day (360kcal/day) increment for the second trimester represents a summing and rounding for ease of calculation of 1.2 MJ/day (285 kcal/day) and 0.35MJ/day (85 kcal/day) (for first trimester) on the grounds that women may not

know they are pregnant until the second trimester. This is pragmatic, but there is no evidence that retrospective energy supplementation of this type alters outcome and in circumstances of unconstrained food intake, increased requirements may have already been achieved through an increase in appetite.

146. The US energy DRI for pregnancy (IoM, 2005) was established using a DLW database of individual energy expenditure measures of pregnant women with pre-pregnancy BMI values of 18.5 up to 25 kg/m^2; the measures were obtained from studies in British (Goldberg *et al.*, 1991b, 1993) Swedish (Forsum *et al.*, 1992) and North American (Kopp-Hoolihan *et al.*, 1999) women. GWG was between 11.6 and 13.5 kg. The median change in TEE was 33.5 kJ (8 kcal) per week of gestation with a large range of -238 kJ (-57 kcal) to +448 kJ (+107 kcal) per week. The value of 33.5 kJ (8 kcal) per week was supported by a subsequent study (Butte *et al.*, 2004) which reported that TEE, as measured by DLW, increased linearly at a similar mean rate of 31 ± 42.7kJ (7.4 ± 10.2 kcal) per week of gestation in women with pre-pregnancy BMI values of 19.8 to 26.0 kg/m^2. The total energy deposition during pregnancy was estimated to be 753 kJ/day (180 kcal/day). Thus, the estimated requirement for energy during pregnancy was derived from the sum of the TEE of the woman in the non-pregnant state plus a median change in TEE of 33.5 kJ/week (8 kcal/week) plus the energy deposited during pregnancy. For the first trimester, no increase in energy intake was recommended, on the basis of only small changes in TEE and weight gain. For the second and third trimesters, the energy deposition rate of 753 kJ/day (180 kcal/day) was added to the increase in TEE expected at 20 weeks (672 kJ; 160 kcal) and 34 weeks (1142 kJ; 272 kcal) to give the incremental values of 1.43 MJ/day (340 kcal/day) and 1.9 MJ/day (452 kcal/day), respectively. As with the FAO/WHO/UNU recommendations (FAO, 2004), maternal fat deposition accounts for a considerable part of these recommendations.

147. The COMA DRV report (DH, 1991) set an increment in EAR of 0.8 MJ/day (191 kcal/day) above the pre-pregnant EAR only during the last trimester, which is equivalent to an extra total intake of 75 MJ (around 18,000 kcal). It was noted that women who were underweight at the beginning of pregnancy, and women who did not reduce activity, may have a higher EAR.

148. An increasing proportion of women in the UK enter pregnancy at a weight exceeding the healthy range and, especially for those who are obese, this may place them and their babies at increased risk (McDonald *et al.*, 2010; March of Dimes, 2002; Stothard *et al.*, 2009). In 2009, between 40% (16-24 years) and 53% (35-44 years) of all women of child-bearing age were either overweight or obese in the UK (NHS IC, 2010) and corresponding mean BMI values (kg/m^2) were 24.8 and 27.2 (see Appendix 10). A study of UK pregnant women (Heslehurst *et al.,* 2007) found that the incidence of maternal obesity at the start of pregnancy had increased from 9.9% to 16.0% between 1990 and 2004, and predicted this would rise to 22% by 2010 if the trend continued.

Energy reference values for pregnancy

149. Current evidence suggests that, in general, it is unlikely that women in the UK require extra energy in the first trimester of pregnancy and compensatory reductions in

PAEE during the second and third trimesters are likely to reduce the demand for extra energy intake at this time. Furthermore, although careful studies of energy expenditure and deposition indicate an apparent need for additional energy (Butte *et al.,* 2003a), such calculations include fat deposition. This is not a determinant of birth weight (Butte *et al.,* 2003a) and some fat deposition remains six months postpartum. Such fat deposition may contribute to weight gain in women through successive pregnancies and could be undesirable, though there is uncertainty about this. On the other hand, there is strong evidence that inadequate GWG is associated with decreased birth weight and fetal growth (small for gestational age) (Siega-Riz *et al.*, 2009). Recommendations for constraining weight gain may also be inappropriate for pregnancies in vulnerable groups such as teenagers. Taken together and in the absence of sufficient evidence to revise the recommendation made by COMA (DH, 1991), in this SACN report it was considered prudent to retain the EAR for pregnancy set by COMA i.e. an additional intake of 0.8MJ/day (191 kcal/day) during the last trimester.

Energy costs of lactation

150. The amount of milk produced and secreted, the energy content of the milk and the energetic efficiency of milk synthesis theoretically determine the energy cost of lactation. There is little evidence of energy conservation, i.e. changes in BMR, thermogenesis or the energy cost of certain physical activities, compensating for these energy costs during lactation in well-nourished women (Butte & King, 2005). Fat stores that accumulate during pregnancy may cover part of the additional energy needs in the first few months of lactation.

151. Variation in the energy content of human milk is principally attributable to fluctuation in milk fat concentration which shows complex diurnal, within-feed and between-breast changes. Twenty-four hour milk sampling schemes have been developed which minimally interfere with the secretion of milk flow and capture the diurnal and within-feed variation (Garza & Butte, 1986). The mean gross energy content of representative 24-hour milk samples analysed in a number of studies of well-nourished women was 2.80 kJ/g or 0.67 kcal/g from 1 to 24 months (Butte & King, 2005).

152. The biological efficiency of converting dietary energy into human milk has been conservatively estimated to be about 80 to 85% (Butte & King, 2005). Based on a WHO-sponsored review (Butte & King, 2002), mean milk intakes through six months postpartum measured by the test-weighing technique were 769g/day for women exclusively breastfeeding. This value is an indicative average for infants of both sexes over the whole six months of exclusive breastfeeding. Correction of the mean milk intakes for the infant's insensible water loss during a feed (assumed to be equal to 5%) gives a mean milk intake over the first six months postpartum of 807g/day (FAO, 2004) for exclusive breastfeeding.

Calculation of energy requirements for lactation

153. The average total energy requirements associated with lactation can be estimated by the factorial approach, whereby the cost of milk production (estimated from

the amount of milk produced, energy density of milk and the energetic efficiency of milk synthesis is added to the energy requirements of non-pregnant women, with an allowance made for energy mobilisation from tissue stores, if replete. In well-nourished women it has been estimated that on average 0.72 MJ/day of tissue stores may be utilised to support lactation during the first six months postpartum (Butte & King, 2002), based on a rate of weight loss of -0.8kg per month (Butte & Hopkinson, 1998). This will vary depending on the amount of fat deposited during pregnancy, lactation pattern and duration.

154. For exclusive breastfeeding during the first six months of life, the mean energy cost of lactation over the six month period is 2.8 MJ/day (675 kcal/day) based on a mean milk production of 807g/day, energy density of milk of 2.8kJ/g (0.67kcal/g), and energetic efficiency of 0.80. This may be subsidised by energy mobilisation from tissues in the order of 0.72 MJ/day (172 kcal/day) (Butte & King, 2002), resulting in a net increment of 2.1 MJ/day (505 kcal/day) over pre-pregnancy energy requirements.

155. The estimated average energy requirement of breastfed infants in the first six months of life is 2.28 MJ/day (545kcal/day) (see Table 5). If this is met by maternal supply and the energetic efficiency of milk synthesis is assumed to be 0.8, this also equates to a maternal energy cost of about 2.8 MJ/day (669kcal/day).

156. The total energy requirements may also be estimated from the sum of TEE plus milk energy output, minus the energy mobilised from tissues. The measurement of TEE by DLW techniques circumvents any assumptions regarding the energetic efficiency of milk synthesis or activity energy expenditure, since they are included in TEE. This approach was taken in four studies between one and six months postpartum of well-nourished women who exclusively breastfed their infants (Butte *et al.*, 2001; Forsum *et al.*, 1992; Goldberg *et al.*, 1991b; Lovelady *et al.*, 1993). Milk energy output averaged 2.15 MJ/day (514kcal/day).

157. The US energy DRI for lactation (IoM, 2005) is based on this approach and uses a DLW database of individual energy expenditure measures of lactating women with a pre-pregnancy BMI value of 18.5 up to 25 kg/m^2. The measured TEE, milk energy output and estimated energy mobilisation from tissue stores are used to estimate energy requirements. Based on a milk energy output rounded to 500kcal/d and an average weight loss of 0.8kg/month, which is equivalent to 170kcal/day, the recommendation is for an increment of 1.38MJ/day (330kcal/day; 2.1MJ/day - 0.72 MJ/day (500 - 170kcal/day)) for the first six months. After the first six months, the energy cost of lactation will depend on the amount of breast milk, which is likely to be diminishing.

158. The FAO/WHO/UNU report (FAO, 2004) employs the factorial approach and recommends an increase in food intake by 2.1MJ/day (505kcal/day) for the first six months of lactation in well-nourished women. It notes that energy requirements for milk production in the second six months are dependent on rates of milk production that are highly variable among women and populations.

Energy reference values for lactation

159. Having reviewed the available evidence, it was agreed that the approach outlined above (paragraph 157) adopted for the US Energy DRI report (IoM, 2005), should be adopted for this report, i.e. an increment of 1380kJ/day (330kcal/day) for the first six months during which time exclusive breastfeeding is recommended. Thereafter, energy intake required to support breastfeeding will be modified by maternal body composition and the breast milk intake of the infant.

Comparisons with reference values for energy in the 1991 COMA report

160. The energy reference values detailed in this SACN report derive from a methodology which differs from the COMA report (DH, 1991). In contrast to the COMA report:

- Energy reference values have been calculated from rates of TEE assessed by the DLW method. This has been used either directly as a measure of TEE for infants or, in all other cases, to identify suitable PAL values which have been employed within a factorial calculation as BMR x PAL.

- BMR has been estimated using the Henry prediction equations, resulting in slightly lower BMR values than would have been estimated from the modified Schofield equations.

- The revised population PAL values are higher. In this report, a value of 1.63 has been used as the population PAL value for adults compared to a PAL value of 1.4 used by COMA in their commonly quoted summary table (Table 2.8; DH, 1991).

- A prescriptive approach has been adopted, calculating energy reference values on the basis of healthy body weights which are slightly lower than those used by COMA (DH, 1991).

The details of these differences compared with previous reports are outlined below. The use of the Henry BMR prediction equations is discussed in Appendix 4; the use of PAL values to determine energy reference values is discussed further in Appendix 5; and a comparison of actual EAR values in this and the 1991 COMA report (DH, 1991) is made in Appendix 13.

161. As a result of these methodological differences, the revised EAR values differ from COMA in a non-uniform way for the various age groups. Compared to mean summary values reported by COMA (see Tables 2.6 and 2.8 (DH, 1991)), the revised EAR values are higher by 6-9% and 15-18% for adolescent boys and girls respectively, and for adults are 3% higher for men and 7-9% higher for women. Table 39 (Appendix 13) provides a comparison of the SACN EAR values against the COMA EAR values presented at the same body weight and PAL values as those used by SACN.

162. In the COMA report (DH, 1991) insufficient DLW data were available to enable calculation of energy reference values for any age group. Such data that were available for infants up to 30 months were used together with energy intake data

to derive EAR values for infants up to 36 months. Energy intake data alone were used for all pre-adolescent children. A factorial approach was adopted for children aged 10-18 years, predicting TEE from BMR x PAL, and assigning PAL values of 1.56 for boys and 1.48 for girls with small additions made for growth costs. In this SACN report a median PAL value of 1.75 has been used for both boys and girls aged 10-18 years, which includes the growth costs. This is reflected in the higher energy reference values for adolescents, especially girls, in this report.

163. For adults, COMA (DH, 1991) discussed a matrix of PAL values varying by occupational and leisure activities from 1.4-1.9 for men and 1.4-1.7 for women, on the basis that judgements could be made about lifestyles of population groups. EAR values were also defined for various weights at nine PAL values between 1.4 and 2.2 (in Table 2.7, DH, 1991). A better understanding of the extent and nature of the variation in TEE within populations and lifestyle groups has clearly indicated the impracticality of predicting lifestyle-dependent PAL values with any precision. For adults in this SACN report, PAL values of 1.49, 1.63 and 1.78 equate to the 25th, median and 75th centile. These values represent the less active, typically active, and the more active, and can only be equated to lifestyles in very general categories: i.e. sedentary, low and moderate activity. However, simple recommendations for the likely influence of changes in activity on PAL values can be made. This approach, adopted here for children and adults, is similar in principle to that recommended for children and adolescents by FAO/WHO/UNU (FAO, 2004): i.e. identifying average PAL values from the DLW data with a ±15% variation for more or less active children. However, here, PAL values for broad age ranges (1-3, >3-<10, 10-18 years) have been aggregated, whereas FAO/WHO/UNU (FAO, 2004) predicted PAL values for each year of age by regression analysis of the DLW data.

164. Although not discussed as such, COMA (DH, 1991) identified weight-maintaining energy reference values. The present SACN report has taken into account the high proportion of the population who are overweight and obese and the generally low levels of physical activity in the UK, and has adopted a prescriptive approach (see paragraphs 90-93). This approach is similar in principle to that taken in the FAO/WHO/UNU report (FAO, 2004) which was also prescriptive (or "normative"), recommending that reference EAR values should be identified in relation to height and listing values which would maintain BMI between 18.5 and 24.9 kg/m^2.

165. COMA recommended that the general adult population could be assumed to have an inactive lifestyle. Accordingly, the population EAR was based on a PAL value of 1.4, which is lower than the 25th centile PAL identified here (i.e. 1.49) but at the time was thought to be an appropriate value. The updated EAR values, estimated from the published DLW-derived TEE data which have become available since 1991 and calculated using a population PAL value of 1.63, better reflect the energy requirements of the current UK population. Despite being calculated using a PAL value about 16% higher than that used by COMA, the revised all-adult energy reference values reported here are 2.8% higher for men and 7-9% higher for women. This is explained by the use of 5-7% lower average body weight for the all-age category of adult men and women and the slightly lower BMR values (see

Table 40 in Appendix 13 for details). It should be noted that these revised values are within the range of measurement error/uncertainty.

166. The energy reference value identified in this report for pregnancy, a single daily increment of 0.8 MJ/day (191kcal/day) in the last trimester, is the same as previously recommended by COMA (DH, 1991), but considerably lower than that recommended by either FAO/WHO/UNU (FAO, 2004) (1.5MJ/day (359kcal/day) during the second trimester and 2.0MJ/day (478kcal/day) during the third trimester), or in the US DRI report (IoM, 2005) (1.43MJ/day (340kcal/day) during the second trimester and 1.9MJ/day (454kcal/day) during the third trimester) (see paragraphs 144-148). As stated above (paragraphs 148-149) there are cogent arguments for avoiding excessive weight gain, especially fat gain, in pregnancy and evidence for adaptive changes in energy expenditure (paragraph 138) which reduce the need for additional food energy intake. Although such arguments were not taken into account in the previous reports, they are considered to be valid and sufficiently important because of the increasing prevalence of overweight and obesity to guide the recommendation in this SACN report.

167. The energy reference value identified here for lactation, an increment of 1.38MJ/day (330kcal/day) for the first six months, is the same as previously recommended in the US DRI report (IoM, 2005), but considerably lower than that recommended for the first six months by either COMA (DH, 1991), an increment of 1.9-2.4MJ/day (454-574kcal/day) or by FAO/WHO/UNU (FAO, 2004) (2.1MJ/day (502kcal/day). In all cases the values derive from estimated milk energy contents less the energy mobilised from maternal stores, with different calculations for the magnitude of these two components. The higher value recommended by FAO/WHO/UNU (FAO, 2004) is mainly due to the inclusion of a scaling factor for milk energy content of +25% to take into account an assumed inefficiency of dietary energy utilisation to provide for milk energy. Since such energy costs would appear as increased TEE in lactating women and because such increases are not observed in practice, the inclusion of such a scaling factor is unwarranted.

4 Conclusions and recommendations

168. The requirement for energy for all population groups was previously set at the level of energy intake required to maintain body weight in otherwise healthy people at their existing levels of physical activity and to allow for any special additional needs (i.e. growth, pregnancy and lactation). Measurements of total energy expenditure (TEE) together with estimates of deposited energy provide the basis for estimating energy reference values. In the absence of growth, an individual's TEE is the sum of daily energy used to maintain basal metabolic rate (BMR; metabolism at rest), energy expended in physical activity, and thermogenesis deriving mainly from food intake. TEE can be expressed as a multiple of BMR, the physical activity level (PAL); hence TEE or EAR is equal to BMR x PAL. BMR is predictable as a function of age, weight and gender, while PAL, which is a descriptor of lifestyle and/or behaviour as determinants of energy expenditure, is independent of these factors, at least as a first approximation. Thus for any PAL value, TEE can be predicted for any group from estimates of the BMR. The BMR is calculated for each population group from their heights and reference body weights at these heights, which are specifically chosen as those likely to be associated with long term good health. During growth, pregnancy and lactation the energy cost of tissue deposition also needs to be taken into account.

169. The most accurate practical means of measuring TEE in free-living individuals integrated over days and weeks is the doubly labelled water (DLW) method. In this method the TEE of individuals is computed from estimates of CO_2 production, which are derived from the rate of loss of the isotopes ^{18}O and ^{2}H from the body over a period of days following the administration of a known dose. The computation relies on a series of assumptions (e.g. about water losses and metabolic fuel use) the validity of which varies between and within individuals, but the limits of such variability are known and considered generally acceptable for the purpose of estimating EARs.

170. Energy requirements for populations have been estimated from DLW-derived measures of TEE in reference populations together with estimates of deposited energy. There are two basic approaches: TEE can be modelled against different anthropometric characteristics of the reference population (e.g. age, healthy body weights) using regression equations which can then be used to predict TEE and EAR values for population groups with defined anthropometric values. Alternatively, measured TEE values and measured or predicted BMR values from the reference populations can be used to derive PAL values. These PAL values can then be used to estimate TEE and EAR values for population groups on the basis of their predicted BMR values at healthy body weights. This is a factorial approach to setting EARs. Both approaches are limited by the validity of the DLW method and, to a larger extent, by the representativeness of the study participants compared with those

groups to whom the EAR will be applied. Nevertheless, the DLW method remains the method of choice and is more accurate than others available.

171. As no large randomised population-based UK studies of any age group have been conducted using DLW, and with measurements from the National Diet and Nutrition Survey as yet insufficient to serve as a reference population, a variety of other data sources (reference populations) have been used to derive the revised EAR values. Those identified as being the most likely to be representative of the UK population were as follows:

- For infants, a longitudinal study of healthy American infants comprising similar numbers of breast fed and breast milk substitute-fed infants (n=76 individuals) (Butte *et al*., 2000a).
- For children and adolescents, a compilation of all identified mean study values for specific ages for boys and girls (n=170 mean study values and about 3500 individual measurements) (Appendix 12).
- For adults, two large population-based studies (Moshfegh *et al*., 2008; Tooze *et al*., 2007) of adult urban populations in the US (n = 890 individuals; Appendix 8). These have similar demographic and anthropometric characteristics as the current UK population although some cultural differences may exist.
- For older adults, no specific representative data set has been identified (see paragraphs 123-125 for discussion).

172. The limitations of these data sets (reference populations) for deriving estimated EARs for the UK population include the small numbers of participants in the infant and at some ages in the child cohorts and the lack of younger adults aged 18-30 years and older adults aged >80 years in the larger adult data set.

173. The application of these data sets to the determination of EAR values is as follows:

- *For infants from birth to 12 months*, FAO/WHO/UNU energy requirement recommendations (Butte, 2005; FAO, 2004) were recalculated on the basis of more recent infant growth data from the WHO Multicentre Growth Reference Study (WHO Multicentre Growth Reference Study Group, 2006).
- *For pregnancy and lactation*, the COMA DRV (DH, 1991) and US DRIs (IoM, 2005) have been used respectively, and reference values given as incremental energy requirements for pregnancy and lactation.
- *For children, adolescents and adults*, revised reference values have been determined using a factorial method of estimating TEE from BMR x PAL, with BMR predicted for reference body weights which can be assumed to be associated with long term health.
- *For older adults*, evidence indicates EAR values estimated for adults should also be applied whilst general health and mobility are maintained (see paragraphs 124-126).

174. The distribution of PAL values within the reference population has been used to indicate a population average value for PAL, as well as the extent to which it is lower or higher for less or more active population groups. Thus, three PAL values are identified for age bands of children, adolescents, and for adults. These are the median which represents the best estimate of the average level of activity for the population, and the 25th and 75th centiles which represent those who are less active and more active than average. In addition, estimates of the likely increase in PAL associated with varying increases in activity levels (for example, increased walking, running and participation in sport or high-level training regimes) are given. The median PAL values identified for adolescents and adults in this report are 1.75 and 1.63 respectively, with 1.63 representing the median PAL value of a reference population in which, like the UK, approximately 60% are overweight or obese. When energy expenditure is impaired due to immobility or chronic illness (e.g. in extreme old age), energy requirements can be based on the less active, 25th centile PAL value of 1.49. For some groups of older people with specific diseases or for patient groups who are bed-bound or wheelchair bound, the PAL value may be consistently lower than this (Tables 11 and 12).

175. BMR values have been calculated on the basis of healthy body weights as indicated by the 50th centile of the WHO Child Growth Standards (RCPCH, 2011) (ages 1 to 4 years), the 50th centile of the UK 1990 reference for children and adolescents (Freeman *et al*., 1995) aged over four years, and equivalent to a BMI of 22.5kg/m^2 at current mean heights for adults, which are consistent with long term health[28]. This approach was adopted in the context of the high prevalence of overweight and obesity in the UK and consequently, in contrast to the COMA EARs, the revised EAR values for both children and adults are prescriptive. The exception to this is pregnant women, where EARs are based on actual pre-conceptual body weights rather than prescriptive body weights.

176. Due to changes in the methodology for determining both PAL values and BMR, the revised EAR values in this report differ from those of COMA in a non-uniform way. The median PAL values identified in this report are higher than those used by COMA in their summary tables i.e. 1.56 for boys, 1.48 for girls and 1.4 for adults. An analysis of the range and distribution of PAL values shows that COMA was likely to have underestimated the PAL values of generally sedentary populations because of an under-appreciation of the influence of routine activities of daily living on energy expenditure. The use of prescriptive body weights which are lower than those used by COMA in their summary tables means that the BMR values used in the SACN calculation of the revised EARs are correspondingly lower. This is in addition to the slightly lower BMR values predicted by the Henry prediction equations used here compared with the Schofield equations used by COMA and by FAO/WHO/UNU (FAO, 2004). A detailed comparison of the revised SACN values with those from COMA can be found in Appendix 13.

28 In adults, the healthy body weight range is defined as a body mass index (BMI) between 18.5-24.9kg/m^2. For the purposes of calculating the revised EAR values, healthy body weights have been identified as those associated with a BMI of 22.5kg/m^2.

177. The reference population identified for estimation of the adult energy reference values comprises predominantly overweight and obese individuals (mean BMI=28 kg/m^2: range 18-43 kg/m^2), and is similar to the current UK population in this respect. It is important to recognise that this reference population was used to identify activity levels (as PAL values), rather than TEE values, and equivalent energy intakes. In fact, the median PAL value, 1.63, is at the upper end of the sedentary-light activity range identified by WHO/FAO/UNU (FAO, 2004) (1.4-1.69). The similarity in distribution of BMI to that in the current UK population enables it to serve as a reference in terms of energy expenditure. However, the derived population PAL value should not be assumed to specifically represent that of overweight and obese subjects; this is because no relationship was observed between the distribution of BMI and physical activity levels within the reference population group. Also, those subjects with healthy body weights exhibited the same average PAL value as those overweight and obese subjects. This observation indicates that the risk of overweight and obesity i.e. a positive energy imbalance, is independent of measured PAL values (see Appendix 8, Table 30). Nevertheless, the importance of proposals for increased physical activity for the general health of the population, referred to in paragraph 181 and 187 below, should not be underestimated.

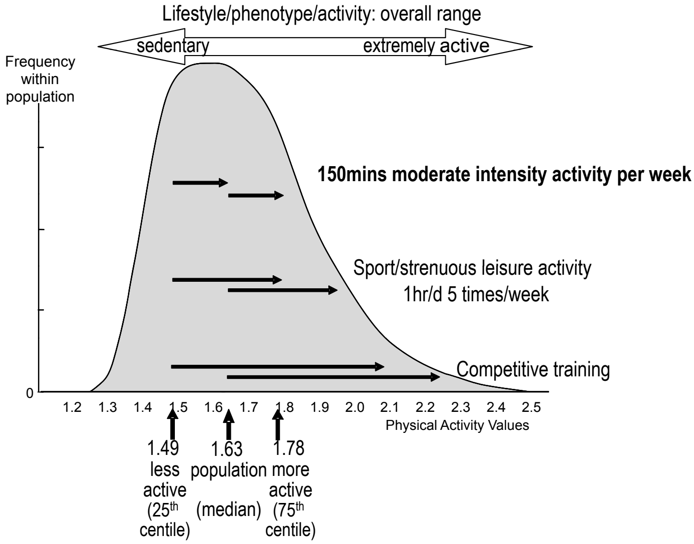
178. These new population EAR values are prescriptive and calculated for body weights thought to be associated with health benefits i.e. weights calculated at current average heights equivalent to a BMI of 22.5kg/m^2 which represents the lower end of the minimum mortality range. Consequently, EAR values will be less than energy intakes which would maintain weight for those who are overweight. For such individuals, reference energy intakes will be associated with weight loss. For adults with a BMI of 30 kg/m^2 with average levels of physical activity, intakes at the new population EAR values for energy would be less than their energy intakes for weight maintenance by on average 1.8MJ/day (426Kcal/day) for men and 1.1MJ/day (260Kcal/day) for women.

179. On the basis of current evidence, it is not possible to define the extent or nature of the impact of diet and physical activity on the risk of weight gain in any detail (see Appendix 11) and as indicated above (see paragraph 177), observed variation in physical activity levels in populations are not directly related to variation in levels of overweight or obesity. Nevertheless, it is clear that both physical activity (CMOs, 2011) and nutrient composition of the diet have key roles in maintaining good health (SACN, 2008). Weight gain is only possible when energy is consumed in excess of requirements, so it is important that individuals are aware of their energy intakes relative to their energy expenditure. Higher physical activity energy expenditures, balanced by a higher energy intake in the form of a nutritionally balanced diet could be expected to yield health benefits, by both increasing fitness and overall nutrient intakes. The EAR values calculated for a healthy body weight and with desirable physical activity consistent with long term health can be assumed to be those based on the "more active" PAL value (i.e. 75th centile). For overweight individuals who undertake this increased activity, energy intakes at this level should mediate weight change towards a more desirable weight.

180. The following general statement describes the energy requirements of adults, with the principle illustrated by Figure 4. The statement should, however, be viewed with caution given the limitations of the data (outlined in paragraph 182). The population estimated average energy requirement is an energy intake from a nutritionally-balanced diet which, in subjects with healthy body weights, balances a TEE of about 1.63 (PAL) x BMR, with BMR varying with body weight, age and gender; this value can be assumed to be maintained in old age as long as subjects remain mobile and in good health. Rates of energy expenditure and consequent requirements vary markedly between individuals, mainly as a result of lifestyle, but also through individual behavioural characteristics. Within the population the overall range is normally between about 1.38-2.5 (PAL) x BMR; PAL values can fall as low as 1.27 (PAL) x BMR and may exceed 2.5, but such low and high values are not thought to be sustainable. Although it is inherently difficult to relate rates of energy expenditure of individuals or small population groups to identifiable activity levels, the population (median) PAL value of 1.63 can be used. For those judged to be either less or more active than average, representative PAL values of 1.49 and 1.78 are appropriate.

181. For maintenance of healthy body weights, individuals may require less or more than these reference values, but individual appetite generally helps match intakes with expenditure. For those leading lives with little physical activity, but maintaining body weight, health benefits would follow from increasing energy expenditure and hence requirements by about 0.15 (PAL) x BMR. This can be achieved by 150 minutes of moderate intensity exercise per week (CMOs, 2011); a brisk walk of one hour/day would result in similar benefit. For very old people in whom activities become limited, energy expenditure and requirement are likely to be at the low end of the physiological range. For groups with average activity levels and body weights greater than values considered to be healthy, intakes at the population EAR level should mediate weight change towards a more healthy weight.

Figure 4 – Schematic representation of energy expenditure and consequent energy requirements of men and women as a function of the physical activity (PAL) value[a]



[a] The distribution of PAL values is that of the reference population discussed in Appendix 8. The likely increases in PAL (and consequent energy needs) shown for various activities are those identified in Table 13 and include the current UK recommendations that adults participate in at least 150 minutes of moderate intensity activity per week (CMOs, 2011). This would result in an increase in PAL of 0.15. These increases in activity are shown as they would affect initial PAL values at the 25th centile and median values, with the increases indicated by the length of the arrows. Although for those with PAL values at the 25th centile the recommendations for increased activity will only increase PAL to the median level, this can still be expected to have health benefits. As explained in Table 25, Appendix 5, the actual increase in PAL associated with a specific activity will vary to a small extent with the initial PAL, i.e. with the intensity of the activity replaced. This means that the actual increases will be slightly less than the values shown, especially for subjects with high initial PAL values.

182. Although these revised EAR values are based on a much larger body of evidence of measured TEE than was available to COMA in 1991 (DH, 1991), it is important to note the insecurities which remain. These include the applicability of the data sets and reference populations from which they are derived to the current UK population; the paucity of data for younger adults aged 18-30 years and for older adults aged >80 years; the methods used to extract PAL values from measured TEE values when BMR has not been measured; the accuracy of the BMR prediction equations; and the measurement of TEE itself (see Appendix 5), especially the possibility of methodological bias influencing the values for the 3-10 year old children (see Appendix 12). Individual activity patterns and consequent rates of TEE can vary considerably over time, so that measures collected over 1-2 week periods provide no more than a snapshot of habitual activity.

183. The approaches used in this report, and previously by COMA (DH, 1991), have produced reference values that 'band' population subgroups according to the mean EAR value for that group. This approach results in the reference value being too low for some people and too high for others. Caution is therefore required when applying these EAR values to groups since a mismatch between energy intake and energy expenditure (unlike most nutrients) has major public health implications; a

proportion of any group would gain weight inappropriately, while others would become under-nourished if everyone received the same energy intake.

184. The COMA EAR values (DH, 1991) of 10.6MJ/day (2550kcal/day) and 8.1MJ/day (1940kcal/day) as average values for all men and women respectively,[29] are commonly quoted, e.g. in healthy eating messages to consumers. These figures were rounded to obtain the commonly cited values of 2500kcal/day for men and 2000kcal/day for women. The continuation of use of these values will require thorough deliberation. The COMA values were derived using average weights and with a PAL value of 1.4. The revised EAR values would indicate higher average values for all men and women respectively: 10.9MJ/day (2605kcal/day) and 8.7MJ/day (2079kcal/day) (Table 16) i.e. 2.8% and 7-9% higher respectively. Given this relatively small difference, especially in the context of the uncertainty arising from the assumptions made in estimating these values, and given the concerns about increasing obesity, careful consideration should be given to the risks and benefits of updating the values particularly as the revised figures fall within the bounds of measurement error/uncertainty.

185. A question arises as to whether a reduction in population energy intake, as recommended in this report, has implications for intakes of other nutrients. The revised EAR values for energy are based on the assumption of both a healthy body weight and consumption of a balanced diet which will supply adequate intakes of all nutrients. For population groups with healthy body weights, these new EAR values should not pose any risk of nutrient inadequacy. For those population groups showing a high prevalence of overweight or obesity, the new EAR values indicate a need to reduce food energy intake if physical activity is unaltered. Whenever food energy intakes are reduced with the intention of weight change towards a healthier, lower body weight, particular care should be taken to maintain a balanced diet to minimise risk of nutrient deficiency; micronutrient deficiencies have been observed for some UK population groups with unbalanced diets (SACN, 2008). In the case of obese individuals the reductions in energy intakes implied by the new prescriptive EAR values will be considerable and recommendations for such changes should be accompanied by expert dietetic advice on dietary nutrient balance.

186. For adults, the revised EAR values are broadly similar to the COMA EAR values (DH, 1991) while for infants aged 0-3 months and adolescents they are higher. Consequently, the impact on the achievement of adequate nutrient intakes should be negligible. The revised EAR values for young children are lower than those previously set, but requirements for micronutrients in this group are generally low in comparison to energy requirements because the rate of growth slows considerably after the first year of life. Thus, the new recommendations should have minimal implications for this group. To meet micronutrient requirements, individuals should be encouraged to consume a healthy balanced diet containing

29 Table 2.8 Estimated Average Requirements (EARs) for energy for groups of men and women with physical activity level of 1.4 (DH, 1991).

a variety of foods including plenty of fruit, vegetables, starchy foods and some protein-rich and dairy foods.

187. Finally, it is important to recognise that where this new report indicates higher than previous population EAR estimates, i.e. for adolescent boys and especially girls and for adult women, this does not mean that these groups are considered to have increased their activity. Instead, the new values represent a closer estimation of energy needs at current activity levels for the UK population. In fact, for a considerable proportion of all population groups who are overweight, the new reference intakes calculated for desirable body weights will be less than their current intakes and should help in achieving a healthier body weight. There is consistent evidence to support current public health recommendations from the UK health departments regarding physical activity; adults are recommended to have at least 150 minutes of moderate intensity physical activity a week (which can be achieved by 30 minutes of moderate activity on five or more days of the week) (CMOs, 2011). For children and young people, a total of at least 60 minutes each day of at least moderate to vigorous intensity physical activity is recommended. If the UK population responds to such recommendations, PAL values would increase: i.e. closer to the current 75th centile identified for the "more active" population for whom an increase in EAR to maintain energy balance may be appropriate.

Recommendations

Rationale for recommendations

188. The Dietary Reference Values (DRVs) for food energy describe the requirements for food energy of the UK population and its subgroups in health. The Estimated Average Requirements (EAR) for energy for the UK population and its subgroups, have been calculated from measurements of total energy expenditure (TEE) in free-living people. In the absence of growth, TEE is primarily the sum of daily energy used to maintain basal metabolic rate (BMR; metabolism at rest) and the energy expended in physical activity. TEE can be expressed as a multiple of BMR, the physical activity level (PAL). Therefore,

$$\text{TEE (or EAR)} = \text{BMR} \times \text{PAL}$$

During growth, pregnancy and lactation, the energy cost associated with tissue deposition or milk secretion must also be taken into account.

189. Body weight will be maintained with energy balance i.e. when energy intake matches energy balance. Any sustained imbalance between energy intake and expenditure will lead to progressive weight gain or loss. In the UK, a substantial proportion of the population are overweight and obese. Obesity increases the risk for a number of diseases (such as type 2 diabetes, some cancers, hypertension and coronary heart disease) and adverse pregnancy outcomes, and represents a major public health problem for the UK.

190. In recognition of the high prevalence of overweight and obesity, the revised EAR values for all population groups (with the exception of pregnant women), are prescriptive in relation to body weight. This means that they are set at the level

of energy intake needed to maintain a healthy body weight. In adults, the healthy body weight range is defined as equivalent to a body mass index (BMI[30]) between 18.5 - 24.9 kg/m^2. For the purposes of calculating the revised EAR values, healthy body weights have been identified as those associated with a body mass index of 22.5 kg/m^2. The prescriptive nature of the revised EAR values means that for population groups who are overweight, the EAR values are lower than the energy intakes required to maintain weight. Consequently, matching energy intake to the EAR values will facilitate weight reduction towards the healthy body weight range. However, it should be noted that the revised EAR values are set at existing levels of physical activity, i.e. a prescriptive PAL has not been used. EAR values set in line with current public health recommendations regarding increased physical activity (see paragraph 201), would therefore be higher than the revised values.

191. The 1991 COMA EAR values for energy (DH, 1991) were based on limited available evidence. Since then, better measurements of TEE in a wide variety of population groups have substantially expanded and improved the evidence base.

192. Following a careful consideration of this evidence, SACN recommend a revision to the EAR values for food energy for infants, children, adolescents and adults. They are intended only for use in healthy populations and are not intended for individuals or groups that require clinical management.

193. BMR should be calculated using the Henry prediction equations (see Appendix 4).

194. Exclusive breastfeeding is recommended for about the first six months of life, however, recognising that infants are fed in a variety of ways, separate recommendations are therefore made for exclusively breast-fed, breast milk substitute-fed infants and where the mode of feeding is mixed or not known. Reference values for infants have been calculated from measurements of TEE plus allowances for energy deposited in new tissue. The latter were calculated by combining weight incremental data from the WHO Multicentre Growth Reference Study (2006) with estimates of energy deposited in new tissue.

30 Body Mass Index (BMI). An index of weight adequacy and obesity of older children and adults, calculated as weight in kilograms divided by the square of height in metres.

Table 14 Revised Estimated Average Requirements (EAR) for infants 1-12 months

Age (months)	EAR [a]					
	Breast-fed		Breast milk substitute-fed		Mixed feeding or unknown [b]	
	MJ/kg per day (kcal/kg per day)	MJ/day (kcal/day)	MJ/kg per day (kcal/kg per day)	MJ/day (kcal/day)	MJ/kg per day (kcal/kg per day)	MJ/day (kcal/day)
Boys						
1-2	0.4 (96)	2.2 (526)	0.5 (120)	2.5 (598)	0.5 (120)	2.4 (574)
3-4	0.4 (96)	2.4 (574)	0.4 (96)	2.6 (622)	0.4 (96)	2.5 (598)
5-6	0.3 (72)	2.5 (598)	0.4 (96)	2.7 (646)	0.3 (72)	2.6 (622)
7-12	0.3 (72)	2.9 (694)	0.3 (72)	3.1 (742)	0.3 (72)	3.0 (718)
Girls						
1-2	0.4 (96)	2.0 (478)	0.5 (120)	2.3 (550)	0.5 (120)	2.1 (502)
3-4	0.4 (96)	2.2 (526)	0.4 (96)	2.5 (598)	0.4 (96)	2.3 (550)
5-6	0.3 (72)	2.3 (550)	0.4 (96)	2.6 (622)	0.3 (72)	2.4 (574)
7-12	0.3 (72)	2.7 (646)	0.3 (72)	2.8 (670)	0.3 (72)	2.7 (646)

[a] Calculated as TEE + energy deposition (kJ/day) as in Table 5.
[b] These figures should be applied for infants when there is mixed feeding and the proportions of breast milk and breast milk substitute are not known.

195. The population PAL values estimated by COMA in 1991 (i.e. 1.56 for boys, 1.48 for girls, and 1.4 for adults) were underestimated. Even relatively sedentary populations exhibit higher rates of PAEE than previously thought. Median PAL values of 1.75 for adolescents and 1.63 for adults are thought to reflect current UK average activity levels better.

196. The revised population EARs for children aged 1-18 years old, based on the 50th centile of weights from the UK-WHO Growth Standards (ages 1-4 years) (RCPCH, 2011) and the UK 1990 reference for children and adolescents (ages 5-18 years) (Freeman *et al.*, 1990) are:

Table 15 Revised population Estimated Average Requirements (EAR) for children aged 1-18 years old[a]

	EAR MJ/d (kcal/d)		
Age (years)	PAL[b]	Boys	Girls
1	1.40	3.2 (765)	3.0 (717)
2	1.40	4.2 (1004)	3.9 (932)
3	1.40	4.9 (1171)	4.5 (1076)
4	1.58	5.8 (1386)	5.4 (1291)
5	1.58	6.2 (1482)	5.7 (1362)
6	1.58	6.6 (1577)	6.2 (1482)
7	1.58	6.9 (1649)	6.4 (1530)
8	1.58	7.3 (1745)	6.8 (1625)
9	1.58	7.7 (1840)	7.2 (1721)
10	1.75	8.5 (2032)	8.1 (1936)
11	1.75	8.9 (2127)	8.5 (2032)
12	1.75	9.4 (2247)	8.8 (2103)
13	1.75	10.1(2414)	9.3 (2223)
14	1.75	11.0 (2629)	9.8 (2342)
15	1.75	11.8 (2820)	10.0 (2390)
16	1.75	12.4 (2964)	10.1 (2414)
17	1.75	12.9 (3083)	10.3 (2462)
18	1.75	13.2 (3155)	10.3 (2462)

[a] Calculated from BMR x PAL. BMR values are calculated from the Henry equations, using weights and heights indicated by the 50th centiles of the UK-WHO Growth Standards (ages 1-4 years) and the UK 1990 reference for children and adolescents.
[b] Physical Activity Level

197. The revised population EAR values for adults, calculated using a PAL value of 1.63 and BMR values calculated at weights equivalent to a BMI of 22.5kg/m^2 at current mean heights for age are:

Table 16 Revised population Estimated Average Requirement (EAR) values for adults

Age range (years)	Men		Women	
	Height cm[a]	EAR MJ/d (kcal/d)[b]	Height cm[a]	EAR MJ/d (kcal/d)[b]
19-24	178	11.6 (2772)	163	9.1 (2175)
25-34	178	11.5 (2749)	163	9.1 (2175)
35-44	176	11.0 (2629)	163	8.8 (2103)
45-54	175	10.8 (2581)	162	8.8 (2103)
55-64	174	10.8 (2581)	161	8.7 (2079)
65-74	173	9.8 (2342)	159	8.0 (1912)
75+	170	9.6 (2294)	155	7.7 (1840)
All adults	175	10.9 (2605)	162	8.7 (2079)

[a] Values for illustration derived from mean heights in 2009 for England (Health Survey for England 2009) (NHS IC, 2010)
[b] Median PAL= 1.63

198. These revised values apply to all adults, unless energy expenditure is impaired due to immobility or chronic illness, e.g. in extreme old age. For this group a lower PAL value of 1.49 should be used.

199. For some population groups, the revised population EAR values are higher than previous estimates. This should not be interpreted to mean that these groups have increased their activity and thus need to eat more, but rather that the new values represent a closer approximation of energy needs at current activity levels, estimated using updated methodology.

200. For pregnancy and lactation, the COMA DRV (DH, 1991) and US DRIs (IoM, 2005) are advised, respectively. For pregnancy an increment of 0.8MJ/d (191kcal/d) above the pre-pregnancy EAR[31], during the last trimester only, is recommended. This assumes that the majority of women in the UK enter pregnancy adequately nourished and that there are decreases in physical activity during pregnancy that may partly compensate for the increased energy costs. It also assumes that the woman has completed her growth; this may not be the case for adolescents entering pregnancy. For lactation, an increment of 1.4MJ/d (335kcal/d) in the first six months is recommended.

201. There is consistent evidence that increasing physical activity is associated with reductions in the risk of chronic diseases such as coronary heart disease, stroke and type 2 diabetes, and the risk of preventable death (DH, 2004). It may also reduce the risk of becoming obese provided energy expenditure is greater than energy intake. If the UK population respond to recommendations to increase physical activity, PAL values would increase and would be closer to the current 75[th] centile identified for the 'more active' population. EAR values would also increase as a result.

31 It is important to note that energy requirements for pregnant women are based on actual pre-conceptional body weights rather than healthy body weights for non-pregnant women (see paragraph 129).

202. These recommendations offer the best estimates of EAR values for population subgroups. Values for individuals will vary considerably. Those population groups with BMI values greater than 25kg/m^2 are likely to benefit from reduced food energy intakes. Increased physical activity is also likely to benefit health, and may help reduce body weight particularly if combined with a reduction in energy intake.

Research Recommendations

203. Scientific evidence has expanded considerably since the previous COMA report (DH, 1991). However, gaps in the data to enable estimation of energy requirements remain and in particular, data are lacking for younger adults aged 18-30 years and for older adults aged >80 years, and are limited for infants and children.

204. The PAL values identified in this report derive from data sets in which TEE, and in most cases BMR, have been measured. This can be considered to be the best and most appropriate available evidence. Nevertheless, uncertainties exist about some of the data which has been used and specific UK population groups are also unrepresented within these data sets. Notably, the adult PAL values used in this report are derived from USA study populations and further investigation is needed both to establish PAL values for the UK population and the health outcomes associated with them.

205. In order to improve future recommendations for energy requirements research in the following areas is required.

Measurements of TEE

206. There is insufficient understanding of the variation in energy expenditure, and hence energy requirements, within individuals over time and between individuals because of behavioural differences (i.e. spontaneous physical activity) are not currently described in lifestyle assessments. It is also not clear why the average value and distribution of PAL appears to be the same in subjects who are obese compared with those with normal weight. Therefore:

- Intra-individual variation in DLW measures of TEE needs to be characterised.
- Improved understanding in all age groups of the relationship between patterns of physical activity, in terms of its intensity and amount, and TEE, body weight maintenance and long term health is needed. This will enable the identification of reference values for physical activity which in turn will allow EAR values to be defined in terms of prescriptive values in relation to physical activity.
- Identifying new and improving existing objective measures of energy expenditure, especially those which allow integrated measurements of TEE comparable to the DLW method, is required.

Specific population groups

- The database of DLW-derived TEE values for children, adolescents, adults aged 18-30 years and those aged >80 years should be expanded.

- Further investigation is required to improve understanding of the marked variation in physiological changes in energy requirements, expenditure and body composition in relation to pregnancy outcomes for mother and child especially in relation to maternal fat gain and its post-pregnancy retention.

- There is a lack of evidence to establish energy requirements for overweight and obese women during pregnancy. The effectiveness and safety of maternal weight management during pregnancy needs to be clarified to inform advice.

- Further data on the relationship between energy intake and the quality and quantity of growth in infants, regardless of the mode of feeding, should be collected.

- Understanding of the potential interaction of diet composition and physical activity in body weight regulation and the development and maintenance of obesity should be improved.

- Activity patterns and values for TEE in older age groups likely to be associated with the maintenance of mobility and reduced risk of morbidity and mortality need to be identified.

Prediction of the BMR

- The predictive accuracy of the Henry equations for BMR within the current UK population needs to be established.

Acknowledgements

The Committee would like to thank the principal investigators from the OPEN and Beltsville studies, Amy Subar (National Cancer Institute, USA) and Alanna Moshfegh (United States Department of Agriculture), for providing data for this report. Thanks to Tim Cole (University College London Institute of Child Health, London) and Kirsten Rennie (University of Ulster) for their contributions to Working Group meetings.

Membership of Energy Requirements Working Group

Chair

Professor Alan Jackson	Professor of Human Nutrition, University of Southampton

Members

Professor Marinos Elia (External Expert)	Professor of Clinical Nutrition and Metabolism at the University of Southampton and Honorary Consultant Physician at Southampton General Hospital
Professor Ian Macdonald	Professor of Metabolic Physiology at the University of Nottingham and Director of Research in the Faculty of Medicine and Health Sciences
Professor Joe Millward (External Expert and Consultant)	Emeritus Professor Human Nutrition, Faculty of Health and Medical Sciences, University of Surrey
Professor Andrew Prentice (External Expert)	Head of MRC International Nutrition Group and Professor of International Nutrition at the London School of Hygiene and Tropical Medicine
Professor Chris Riddoch (External Expert)	Professor of Sport and Exercise Science, University of Bath
Dr Anita Thomas	Consultant Physician in Acute Medicine, Plymouth Hospitals NHS Trust
Dr Anthony Williams	Reader in Child Nutrition and Consultant in Neonatal Paediatrics, St George's, University of London
Dr Stella Walsh (Consumer representative)	Postgraduate Programme Leader, Leeds Metropolitan University
Dr Robert Fraser (Co-opted from SACN's Sub-group on Maternal and Child Nutrition to advise on energy requirements during pregnancy and lactation)	Reader, Reproductive and Developmental Medicine, University of Sheffield

External Consultant

Dr Peter Sanderson

Secretariat

Dr Alison Tedstone (Scientific)

Dr Sheela Reddy (Scientific)

Ms Rachel Elsom (Scientific)

Ms Emma Peacock (Scientific)

Mr Andrew James (Statistics)

Ms Verity Kirkpatrick (Scientific)

Membership of Scientific Advisory Committee on Nutrition

Chair

Dr Ann Prentice (From June 2010)	Director, MRC Human Nutrition Research, Cambridge
Professor Alan Jackson (Until June 2010)	Professor of Human Nutrition, University of Southampton

Members

Professor Peter Aggett	Honorary Professor, School of Medicine and Health, Lancaster University, and Emeritus Professor and Past Head of School of Postgraduate Medicine and Health
Professor Annie Anderson (Until February 2011)	Professor of Food Choice, Centre for Public Health Nutrition Research, University of Dundee
Late Professor Sheila Bingham (Until March 2009)	Formerly Director, Medical Research Council's Dunn Human Nutrition Unit, Cambridge.
Mrs Christine Gratus (Lay representative)	Retired advertising and marketing research director.
Dr Paul Haggarty	Head of Lifelong Health, Rowett Institute of Nutrition and Health, University of Aberdeen
Professor Timothy Key	Professor in Epidemiology and Deputy Director of Cancer Epidemiology Unit, University of Oxford
Professor Peter Kopelman (Until November 2010)	Principal, St George's, University of London
Professor Susan Lanham-New (From November 2009)	Head, Nutritional Sciences Division and Reader in Nutrition, Faculty of Health and Medical Sciences at the University of Surrey
Professor Julie Lovegrove (From November 2009)	Reader in Nutritional Metabolism and Deputy Director of the Institute of Cardiovascular & Metabolic Research at the University of Reading
Professor Ian Macdonald	Professor of Metabolic Physiology at the University of Nottingham and Director of Research in the Faculty of Medicine and Health Sciences

Professor Harry McArdle (From November 2009)	Deputy Director of Science and the Director of Academic Affairs at the Rowett Institute of Nutrition and Health, University of Aberdeen
Dr David Mela (Industry representative)	Science Leader, Unilever R&D Vlaardingen, The Netherlands
Professor Hilary Powers (From November 2009)	Professor of Nutritional Biochemistry and Head of Human Nutrition Unit, University of Sheffield
Dr Anita Thomas (Until February 2011)	Consultant Physician in Acute Medicine, Plymouth Hospitals NHS Trust
Professor Angus Walls (From November 2009)	Professor of Restorative Dentistry and Director of The Centre for Oral Health Research, Newcastle Biomedicine, Newcastle University
Dr Stella Walsh (Consumer representative)	Postgraduate Programme Leader, Leeds Metropolitan University
Dr Anthony Williams	Reader in Child Nutrition and Consultant in Neonatal Paediatrics, St George's, University of London
Professor Ian Young (From November 2009)	Professor of Medicine and Director of the Centre for Public Health at Queen's University Belfast

Observers

Dr Alison Tedstone	Department of Health
Dr Sheela Reddy	Department of Health
Dr Fergus Millan	Scottish Government, Health Department
Mrs Maureen Howell	The Welsh Assembly, Health Promotion Division
Dr Naresh Chada	Department of Health, Social Services and Public Safety, Northern Ireland

Secretariat

Dr Elaine Stone (Scientific Secretary)

Mrs Vicki Pyne (Scientific)

Ms Rachel Elsom (Scientific)

Ms Rachel White (Scientific)

Mr Michael Griffin (Administrative)

Appendix 1 – SACN working procedures

Meetings

Energy Requirements Working Group meetings

207. The SACN Energy Requirements Working Group was established in 2005. The Working Group met three times in 2005, twice in 2006 and 2007, three times again in 2008, then twice in 2009 and one final time in 2010. At the final meeting, the Working Group considered the comments received in response to the consultation on the draft report (see below). The minutes of all the meetings are available on the SACN website (www.sacn.gov.uk).

Main SACN meetings

208. The draft report was considered by the full Committee in its February 2009 and June 2010 meetings and by correspondence.

Consultation

209. The draft report was published on the SACN website on 5 November 2009. Interested parties were invited to submit comments relating to the science of the report by 11 February 2010.

210. Submissions were received from the following organisations and individuals.

    1. British Dietetics Association (BDA)
    2. British Nutrition Foundation (BNF)
    3. Professor Elisabet Forsum, Linköping University, Sweden
    4. Professor Jeya Henry, Oxford Brookes University
    5. Ki Performance (Consultants) Limited
    6. Medical Research Council Human Nutrition Research (MRC-HNR), Cambridge
    7. Public Health Nutrition Research Group, University of Aberdeen
    8. *Safe*food
    9. School Food Trust
    10. Sugar Bureau
    11. Professor JT Winkler, London Metropolitan University

211. The Working Group's response to all the submissions is available on the SACN website (www.sacn.gov.uk).

Appendix 2 – Energy yields from substrates

212. This Appendix provides details on the energy provided by different macronutrients and alcohol

Fat

213. The amount of energy yielded from fat in food varies slightly with the type of food, mainly according to the chain length and degree of saturation of constituent fatty acids in dietary triglycerides. It is generally assumed that the digestibility of all dietary fat is the same (≈95%). In practice, because dietary fat incorporates a mixture of different fatty acids these differences are ignored and the metabolisable energy (ME) content of dietary fat (the general Atwater factor) is assumed to be 37kJ (9.0 kcal/g), i.e. equal to the digestible energy content at 95% of gross energy (GE).

Carbohydrate

214. The amount of energy yielded from different carbohydrates in food varies according to the molecular form i.e. glucose, disaccharides and starch, the actual available energy content per unit weight is 15kJ (3.6 kcal/g), 16kJ (3.8 kcal/g) and 17kJ (4.0 kcal/g) respectively. FAO/WHO/UNU (FAO, 2004), however, has recommended that when carbohydrate is expressed as monosaccharide equivalents, a conversion factor of 16 kJ/g (3.8 kcal/g) should be used, and when determined by direct analysis, this should be expressed as the weight of the carbohydrate with a conversion factor of 17 kJ/g (4.0 kcal/g); the latter value being an estimated average of the different forms of carbohydrate in food. It should be noted that this method is used in the UK but not in all other countries. In the USA, for example, carbohydrate is calculated by difference, i.e. from total dry weight of food minus protein, fat and ash. This method includes unavailable carbohydrate (i.e. dietary fibre) and other non-carbohydrate components (e.g. lignin, organic acids, tannins, waxes, Maillard products) in the calculated value. This means that for an average diet, total energy intakes can be 12% higher and carbohydrate intake 14% higher when carbohydrate by difference is used in dietary analysis compared with available carbohydrate analysed directly. As a result, estimates of the extent of under-reporting in dietary surveys in the USA (Subar *et al*, 2003) are often lower than those in the UK (Dr Alison Lennox[32], personal communication).

215. Other carbohydrates may also provide energy. Fermentation of non-starch polysaccharides (NSPs) in the colon results in the formation of short-chain fatty acids, some of which are absorbed into the blood stream and are used as energy. A conversion factor of 8 kJ/g (1.9 kcal/g) has been suggested (FAO, 2003). The

32 Dr Alison Lennox is Head of Population Nutrition Research at the Medical Research Council Human Nutrition Research, Cambridge.

UK food composition table energy values only include carbohydrate expressed as monosaccharide (FSA, 2002).

Protein

216. The amount of energy yielded from protein in food can vary according to both the quality and the digestibility of the protein. These differences can mean that the energy yield from some highly digestible animal proteins like egg may be 40% greater than for some less digestible plant proteins. Also, in relation to the net metabolisable energy (NME), the immediate metabolic fate of amino acids absorbed into the body includes deamination and other reactions which contribute to the TEF. However, this energy loss is usually ignored. The ME content of dietary protein (the general Atwater factor) is assumed to be 17kJ (4.0 kcal/g), a value which is slightly lower than the actual value for most animal proteins and higher than that for most plant proteins. The average energy yield will usually be close to the assumed ME content because dietary protein usually represents a mixture of several animal and plant protein sources.

Alcohol

217. Alcohol yields 29 kJ (6.9 kcal) per gram consumed. Alcohol oxidation starts rapidly after absorption, and alcohol is eventually completely eliminated by oxidation (Prentice, 1995; Schutz, 2000) so that it does not directly add to body energy stores. Consumption of alcohol can modestly activate the hepatic *de novo* lipogenesis pathway, but some of the acetate produced in the liver by alcohol dehydrogenase (the major quantitative fate of ingested ethanol (Siler *et al*., 1999)) is released into plasma and inhibits adipose tissue lipolysis. This influence on tissue fuel selection can reduce fat oxidation and contribute to an increase in adiposity. The results from studies on the magnitude of the TEF after alcohol consumption vary, with reported values ranging between 9% and 28% (Prentice, 1995; Raben *et al*., 2003).

Appendix 3 – Components of energy expenditure and factors affecting it

Components of energy expenditure

Basal and resting metabolism

218. The basal metabolic rate (BMR) is a standardised measure of an individual's metabolism in a basal, postabsorptive state: i.e. while awake and resting. This is a standardised metabolic state corresponding to the situation at thermoneutrality when food and physical activity have minimal influence on metabolism. BMR and resting metabolic rate (RMR) are often used interchangeably, but if RMR is not measured at the standardised metabolic state, it can include additional metabolic activity (e.g. thermic effect of food and excess post-exercise oxygen consumption, see paragraph 228), and would therefore be greater than the BMR. Because the BMR includes a small component associated with arousal, it is slightly higher than the sleeping metabolic rate.

Physical activity

219. Physical activity is a complex and multi-dimensional behaviour taking place in a variety of domains: in transportation, domestic life, occupation and recreation (Wareham & Rennie, 1998). The dimensions of a specific physical activity are defined as its volume, frequency, intensity, time and type.

220. 'Physical activity', 'exercise', and 'physical fitness' are terms that describe different concepts. Physical activity is defined as any bodily movement produced by the contraction of skeletal muscles resulting in energy expenditure (Caspersen *et al.*, 1985). Exercise is a subset of physical activity that is planned, structured, and repetitive; its final or intermediate objective is the improvement or maintenance of physical fitness and/or health. Physical fitness is a set of attributes that are health and/or performance-related. The degree to which people have these attributes can be measured with specific tests (Caspersen *et al.*, 1985).

221. Physical activity-related energy expenditure (PAEE) is quantitatively the most variable component of total energy expenditure (TEE), usually accounting for 25-50% of energy requirements and up to 75% of energy requirements (Westerterp, 1998) in extreme situations which are highly unusual and not sustainable. Due to differences in body size, skill and training there is a large inter-individual variation in the energy expended for any given activity.

222. The energy costs of different physical activities can be expressed as multiples of BMR to account for differences in body size, i.e. the physical activity ratio (PAR). Such energy costs can also be expressed as multiples of the metabolic energy equivalent (MET): a fixed rate of oxygen consumption assumed to represent that of an adult measured at supine rest (defined as 3.5 ml oxygen consumption/kg body

weight/minute). 1 MET is similar to the BMR, but its precise relationship will vary as a function of body weight, age and gender, which each influence BMR per kg body weight (IoM, 2005). The physical activity level (PAL), the ratio of daily energy expenditure to BMR, used in TEE calculations is discussed in Appendix 5.

223. The role of physical activity in raising TEE depends on the intensity and duration of the activity undertaken and whether this affects the degree to which other physical activities are performed, i.e. an increase in one component in PAEE may be balanced by a compensatory decrease in another. In addition, many studies of human subjects indicate a short-term elevation in RMR following single exercise events (generally termed the excess post-exercise oxygen consumption; EPOC). This EPOC appears to have two phases, one lasting less than two hours and a smaller, more prolonged effect lasting up to 48 hours (Speakman & Selman, 2003). The EPOC varies with exercise intensity and duration. The effects of long term exercise training on BMR, however, are less clear (Speakman & Selman, 2003).

Spontaneous physical activity and non-exercise activity thermogenesis

224. Spontaneous physical activity (SPA) is a term used to describe all body movements associated with activities of daily living, change of posture and 'fidgeting' (Ravussin *et al.*, 1986). SPA accounts for a between-individual variation in energy expenditure of ± 15% and has been shown to be highly reproducible within individuals, a familial trait, and to significantly correlate with free-living TEE measured by doubly labelled water (DLW) (Snitker *et al.*, 2001; Zurlo *et al.*, 1992). SPA displays an inverse relationship with future weight gain (Zurlo *et al.,* 1992) and it has therefore been described as a putative obesity subphenotype (Snitker *et al.,* 2001). It has been argued that because SPA has only been quantified within a calorimeter it cannot be regarded as a component of free-living TEE in its own right in comparison to BMR and the thermic effect of food (TEF, see paragraph 228). It is described as "*a useful paradigm to quantify an individual's propensity to locomotion under standardized conditions*" (Snitker *et al.,* 2001).

225. Non-exercise activity thermogenesis (NEAT) is another term used to describe the additional energy expenditure attributable to spontaneous physical activities other than volitional exercise, during everyday activities (Levine *et al.*, 1999). Like SPA, NEAT has been implicated in energy balance regulation following observations in overfeeding studies that subjects exhibiting high levels of NEAT (Levine *et al.*, 1999) displayed a resistance to fat gain. More recently, however, the term NEAT has been applied by the same author to all ambulatory activity, apart from specific sports, including relatively high level exertion activities such as dancing (Levine, 2004), and with walking identified as the predominant component. In this context, NEAT ceases to be an appropriate term since easily identifiable exercises have been included.

226. Although both SPA and NEAT may be used to describe "fidgeting" in popular parlance, they are each likely to involve a much wider range of behaviours influencing energy expenditure. Thus, SPA as an "*individual's propensity to locomotion*" (Snitker *et*

al., 2001) is likely to exhibit itself in terms of the non volitional choices between low and high expenditure activities which are a continuous feature of daily living (e.g. standing or walking up escalators, using lifts or stairs). In this report the term SPA, rather than NEAT, will be used in the wider sense to represent spontaneous physical activity that can occur within any domain. As such, SPA is potentially quantifiable as the technology for measuring movement and activity improves.

227. SPA, with its potential magnitude, and large between-individual but small within-individual variation, represents a behavioural phenotype which is likely to significantly influence individual energy expenditure independently of lifestyle categories.

Other components of energy expenditure

Thermic effect of food

228. The thermic effect of food (TEF) or heat increment of feeding can be attributed to the metabolic processes associated with ingestion, digestion and absorption of food, and the intermediary metabolism and deposition of nutrients. TEF varies mainly according to the amount and composition of dietary macronutrients and is usually assumed to account for 10% of energy intake (Kleiber, 1975). This means that for any individual the energy used in TEF varies in absolute terms according to overall rates of energy intake, and will usually be greater than 10% of BMR over a 24 hour period in individuals eating a mixed diet.

Factors affecting energy expenditure

Body size and composition

229. Energy expenditure is related to body size, mainly in terms of weight and to a lesser extent height. In general, larger people have more tissue mass than smaller people and therefore have a higher BMR.

230. The metabolically active tissue mass of the body is termed fat-free mass (FFM) and comprises muscle, bone, skin and organs. FFM is the principal determinant of inter-individual variation in BMR and RMR, after adjustment for body size (Byrne *et al.*, 2003; Heymsfield *et al.*, 2002; Illner *et al.*, 2000; Johnstone *et al.*, 2005; Muller *et al.*, 2004; Nelson *et al.*, 1992; Wang *et al.*, 2000; Weinsier *et al.*, 1992). Fat mass (FM) has been observed to account for a small amount of the inter-individual variation in BMR and RMR in most studies (Cunningham, 1991; Ferraro & Ravussin, 1992; Fukagawa *et al.*, 1990; Johnstone *et al.*, 2005; Karhunen *et al.*, 1997; Muller *et al.*, 2004; Nelson *et al.*, 1992; Svendsen *et al.*, 1993; Weinsier *et al.*, 1992), but not all (Bogardus *et al.*, 1986; Segal *et al.*, 1987).

231. In adults, variation in the composition of FFM, after adjustment for body size, may also account for a small amount of the inter-individual variation in BMR and RMR (Gallagher *et al.*, 1998; Garby & Lammert, 1994; Heymsfield *et al.*, 2002; Illner *et al.*, 2000; Sparti *et al.*, 1997).

232. In infants, children and adolescents, there is an increase in BMR with age, due to growth and increasing body size. Body composition changes during growth. At birth, the newborn is about 11% FM. Progressive fat deposition in the early months results in a peak in the percentage of FM (about 31%) at three to six months, which declines to about 27% by 12 months of age (Butte *et al.,* 2000b). During infancy and childhood, girls grow more slowly than boys and girls have slightly more body fat. During adolescence, the gender differences in body composition are accentuated (Tanner, 1990). In boys, there is a rapid increase in FFM, coinciding with the rapid growth spurt in height, and a modest increase in FM during early puberty, followed by a decline; in girls, adolescence is characterised by a modest increase in FFM and a continual accumulation of FM. The pubertal increase in FFM ceases after about 18 years of age (Tanner, 1990).

233. Body mass and body composition have been shown to impact on PAEE and TEE in children (Ekelund *et al.*, 2004a; Johnson *et al.*, 1998) and adults (Black *et al.*, 1996; Butte *et al.*, 2003b; Carpenter *et al.*, 1995; Goran *et al.*, 1993a; Mâsse *et al.*, 2004; Plasqui *et al.*, 2005; Roberts & Dallal, 1998; Rush *et al.*, 1999; Schulz & Schoeller, 1994).

234. In adults, however, on a population basis and up to a moderate level of fatness (BMI <30 kg/m^2), the relative proportions of FFM and FM are probably unlikely to influence TEE in ways other than through their impact on body weight (Durnin, 1996). In adults with higher percentages of body fat composition, an effect on the mechanical efficiency of movement can increase the energy expenditure associated with weight-bearing activities (Prentice *et al.*, 1996c).

235. Overweight and obese individuals have been shown to have higher absolute TEE than normal weight individuals, because of the effect of a higher BMR associated with increased body size (FM and FFM (Das *et al.*, 2004; Prentice *et al.*, 1996a)). Because weight gain in men contains a higher proportion of FFM than in women, the increase in BMR with BMI is greater in men than women. For example, within the Beltsville DLW cohort examined in this report (Moshfegh *et al.,* 2008) (Appendix 8), the slope of the regression of measured BMR on BMI in men was twice that in women. The observed increase in TEE is not in direct proportion to body weight since, when expressed per kg, both TEE and PAEE decline with increasing BMI (Prentice *et al.*, 1996a). Nevertheless, the decline in PAEE/kg with increasing BMI is relatively shallow, decreasing in the combined OPEN (Subar *et al.*, 2003; Tooze *et al.*, 2007) and Beltsville (Moshfegh *et al.,* 2008) adult DLW data sets used in this report by only 1% for each one unit increase in BMI. Because of this, even though this cohort included a wide range of BMI values (14.4 to 51 kg/m^2: mean=27 kg/m^2), BMI explained less than 5% of the variance in PAEE and was not significantly correlated with either PAL values or the residuals from a multiple regression of age, weight, height and gender on TEE. This lack of any obvious difference in behaviour in terms of PAEE with increasing obesity is probably explained by any reduction in weight-bearing activity with increasing obesity cancelling out the increasing energy cost of such activities.

Gender

236. Gender differences in TEE largely reflect differences in body size and composition, with less FFM and more FM/kg in women than men. Thus, absolute and per kg TEE is lower in women. In most studies, BMR and TEE are observed to be lower in girls (Goran *et al.*, 1994a; Goran *et al.*, 1995; Kirkby *et al.*, 2004) and women (Arciero *et al.*, 1993; Carpenter *et al.*, 1998; Dionne *et al.*, 1999; Ferraro *et al.*, 1992; Poehlman *et al.*, 1997) even after adjustment for body size and composition; however, some studies have observed no differences in TEE between sexes after adjustment for FFM in children (Ekelund *et al.,* 2004a) and adults (Blanc *et al.*, 2004; Klausen *et al.*, 1997).

237. In pre-menopausal women, a small increase in BMR, RMR, sleeping metabolic rate and TEE during the luteal phase of the menstrual cycle, has been observed in several studies (Bisdee *et al.*, 1989; Day *et al.*, 2005; Ferraro *et al.*, 1992; Hessemer & Bruck, 1985; Howe *et al.*, 1993; Lariviere *et al.*, 1994; Meijer *et al.*, 1992; Melanson *et al.*, 1996; Pelkman *et al.*, 2001; Reimer *et al.*, 2005; Solomon *et al.*, 1982; Webb, 1986), although not all (Diffey *et al.*, 1997; Kimm *et al.*, 2001; Li *et al.*, 1999; Piers *et al.*, 1995). This suggests an effect on energy expenditure by sex hormones; in premenopausal women, pharmacological suppression of oestrogen and progesterone release has been observed to reduce RMR (Day *et al.,* 2005). There have been no longitudinal studies of energy expenditure in women across the menopausal transition to determine whether the natural withdrawal of sex hormones influences energy expenditure. It has been speculated that suppression of ovulation with contraceptives could prevent the increase in energy expenditure observed in the luteal phase (Bisdee *et al.*, 1989), but results from studies investigating the effect of contraceptive drug use on energy expenditure have been equivocal (Bisdee *et al.*, 1989; Day *et al.*, 2005; Diffey *et al.*, 1997; Eck *et al.*, 1997; Ferraro *et al.*, 1992; Hessemer & Bruck, 1985; Howe *et al.*, 1993; Kimm *et al.*, 2001; Lariviere *et al.*, 1994; Li *et al.*, 1999; Meijer *et al.*, 1992; Melanson *et al.*, 1996; Pelkman *et al.*, 2001; Piers *et al.*, 1995; Reimer *et al.*, 2005; Solomon *et al.*, 1982; Webb, 1986).

Age

238. There is a decline in BMR with older age and this is mainly attributable to the progressive loss of FFM observed with aging (Kyle *et al.*, 2001); however, a small decline in BMR with age, independent of any age-related changes in FFM, has been observed in most cross-sectional studies (Bosy-Westphal *et al.*, 2003; Johnstone *et al.*, 2005; Kim *et al.*, 2002; Klausen *et al.*, 1997; Pannemans & Westerterp, 1995; Piers *et al.*, 1998; Poehlman *et al.*, 1991; Roberts *et al.*, 1995b; Vaughan *et al.*, 1991; Visser *et al.*, 1995), but not all (Cunningham, 1980; Das *et al.*, 2001). The age-related decline in BMR was fully accounted for by a reduction in FFM and proportional changes in its metabolically active components in one study in healthy subjects (Bosy-Westphal *et al.*, 2003).

239. It is unclear whether changes in the BMR with age are entirely a result of changes in body composition or whether this is related to other factors, e.g. a decline in sodium-potassium ATPase activity (see paragraph 16), decreased muscle protein turnover, and changes in mitochondrial membrane protein permeability (Wilson

& Morley, 2003). A difficulty encountered in studies of the effects of aging on the decline in BMR is the differentiation of the aging process itself from common age-associated diseases and the subsequent effects on organ metabolic rates e.g. left ventricular hypertrophy (Bosy-Westphal *et al.*, 2003).

240. PAEE has been observed to decline with aging (Roberts & Rosenberg, 2006; Wilson & Morley, 2003), but the results from studies investigating an effect of aging on diet-induced thermogenesis have been inconsistent (Kunz *et al.*, 2000; Melanson *et al.*, 1998).

Genetics

241. Genetic inheritance potentially influences all factors affecting inter-individual variation in energy expenditure, e.g. body size and composition, and a familial influence on RMR, independent of FFM, age, and sex, has been reported (Bogardus *et al.*, 1986; Bouchard *et al.*, 1989). Genotype association studies have largely been restricted to candidate genes whose dysfunction might reasonably be expected to have an effect on energy expenditure.

242. Overall, the results do not show a consistent effect on energy expenditure of any of the different genotypes studied thus far: the adrenoceptors (*ADRB1* (Dionne et al., 2002; Nagai et al., 2003); *ADRB2* (Oomen *et al.*, 2005); *ADRB3* (Dionne et al., 2001; Gagnon et al., 1996; Hojlund et al., 2006; Rawson et al., 2002; Rissanen et al., 1997; Shiwaku et al., 2003; Tchernof et al., 1999) the leptin receptor gene (Loos *et al.*, 2006; Stefan *et al.*, 2002; Wauters *et al.*, 2002), the mitochondrial uncoupling protein genes (*UCP1* (Oppert et al., 1994; Ukkola et al., 2001; Valve et al., 1998); *UCP2* (Astrup et al., 1999; Kimm et al., 2001; Klannemark et al., 1998; Maestrini et al., 2003; Ukkola et al., 2001; Walder et al., 1998; Yanovski et al., 2000); *UCP3* (Kimm et al., 2001; Ukkola et al., 2001) sodium-potassium ATPase genes (*ATP1A1*, *ATP1BL1*) (Deriaz *et al.,* 1994) or the intestinal fatty acid binding protein 2 gene (Kim *et al.,* 2001; Sipilainen *et al.,* 1997).

243. Several other individual studies have found associations between energy expenditure and genotypes for the glucocorticoid receptor gene (Di Blasio *et al.,* 2003), interleukin-6 gene (Kubaszek *et al.,* 2003), melanocortin-4 receptor gene (Rutanen *et al.,* 2004; Krakoff *et al.,* 2008) and the dopamine D2 receptor gene (Tataranni *et al.,* 2001). Genome wide association studies, in several populations, have detected significant linkage on several chromosomes with measures of energy expenditure (Cai *et al.,* 2008; Wu *et al.,* 2004; Norman *et al.,* 1998).

244. There have, as yet, been no clearly established relationships between specific genotypes and energy expenditure.

Genetics of obesity

245. Many studies have examined possible hereditary factors predisposing to human obesity that influence energy expenditure. It is notable however that, thus far, all monogenic defects identified as causing human obesity disrupt hypothalamic pathways and have an effect on satiety and food intake (O'Rahilly & Farooqi, 2006). Genome-wide association studies have shown genetic variation in the *FTO*

gene (a 2-oxoglutarate-dependent nucleic acid demethylase gene (Gerken *et al.,* 2007) to be associated with FM and obesity across multiple populations (Dina *et al.*, 2007; Frayling *et al.*, 2007; Hinney *et al.*, 2007; Hunt *et al.*, 2008; Kring *et al.*, 2008; Scuteri *et al.*, 2007), although only accounting for a small amount of the variation. In 13 cohorts, with a total of 38,759 participants from the UK and Finland, the 16% of adults who had the AA genotype for the *FTO* gene weighed about 3 kg more and had a 1.67 fold increased odds of obesity compared with the non-carriers (TT genotype) (Frayling *et al.,* 2007). The genetic variation in the *FTO* gene has also been implicated in appetite control (Cecil *et al.*, 2008; Timpson *et al.*, 2008; Wardle *et al.*, 2008; Wardle *et al.*, 2009), but not energy expenditure, after adjustment for body size (Berentzen *et al.*, 2008; Cecil *et al.*, 2008; Do *et al.*, 2008). The association between *FTO* variants and obesity has been observed to be reduced in those who are more physically active (Rampersaud *et al.,* 2008) and accentuated in those who are less physically active (Andreasen *et al.,* 2008). Inter-individual differences in susceptibility to obesity, therefore, may be determined, in part, by genetic variants impacting on appetite control in the presence of environmental exposures.

Ethnicity

246. Differences in body composition and FFM composition exist between different ethnic groups, e.g. between white and black (Jones, Jr. *et al.*, 2004) and white and Asian populations (Soares *et al.*, 1998; Wouters-Adriaens & Westerterp, 2008).

247. Most studies, although not all (Blanc *et al.*, 2004; Kushner *et al.*, 1995; Lawrence *et al.*, 1988; Nicklas *et al.*, 1997; Sun *et al.*, 1998), suggest that BMR, adjusted for differences in FFM and FM, is lower in black subjects than white subjects (Albu *et al.*, 1997; Blanc *et al.*, 2004; Carpenter *et al.*, 1998; Chitwood *et al.*, 1996; Forman *et al.*, 1998; Foster *et al.*, 1997; Foster *et al.*, 1999; Gannon *et al.*, 2000; Jakicic & Wing, 1998; Kaplan *et al.*, 1996; Kimm *et al.*, 2001; Lovejoy *et al.*, 2001; Morrison *et al.*, 1996; Sharp *et al.*, 2002; Sun *et al.*, 2001; Treuth *et al.*, 2000a; Weinsier *et al.*, 2000; Weyer *et al.*, 1999; Wong *et al.*, 1999; Yanovski *et al.*, 1997). This difference, however, may be due to racial differences in the composition of FFM i.e. metabolically active organ mass, rather than ethnic differences in metabolism (Byrne, 2003; Gallagher, 2006; Hunter, 2000; Jones *et al.*, 2004; Tershakovec, 2002).

248. Racial differences in BMR, adjusted for body weight, between Asian and white subjects/populations appear to be accounted for by differences in FFM and FM (Soares *et al.*, 1998; Wouters-Adriaens & Westerterp, 2008). Differences in body composition, therefore, appear to be mainly responsible for the reported differences in energy expenditure between ethnic groups.

Endocrine state

249. As discussed above (paragraph 237), sex hormones may affect energy expenditure. Other hormones have also been implicated in the regulation of energy expenditure.

250. Thyroid status is a major determinant of metabolic rate. Hyperthyroidism increases while hypothyroidism decreases RMR (Danforth & Burger, 1984). It is unclear, however, whether variation within the normal physiological range of plasma thyroid hormone, tri-iodothyronine (T_3), concentration is associated with variation in BMR, independently of FFM (Al Adsani *et al.*, 1997; Astrup *et al.*, 1992; Bernstein *et al.*, 1983; Johnstone *et al.*, 2005; Muller *et al.*, 1989; Obarzanek *et al.*, 1994; Onur *et al.*, 2005; Rosenbaum *et al.*, 2000; Svendsen *et al.*, 1993; Toubro *et al.*, 1996; Van Wymelbeke *et al.*, 2004; Welle *et al.*, 1990).

251. Plasma noradrenaline concentration has been observed to be associated with RMR, adjusted for FFM (Rosenbaum *et al.*, 2000; Toubro *et al.*, 1996), but not all studies have found this association (Obarzanek *et al.*, 1994).

252. The hormone leptin is involved in energy balance and is produced primarily in white adipose tissue; it is subject to acute regulation, particularly by the sympathetic nervous system (Trayhurn, 2001). It is unclear whether variation in plasma leptin concentration is associated with variation in RMR, adjusted for FFM and FM (Bobbioni-Harsch *et al.*, 1999; Filozof *et al.*, 2000; Haas *et al.*, 2005; Johnstone *et al.*, 2005; Jorgensen *et al.*, 1998; Kennedy *et al.*, 1997; Nagy *et al.*, 1997; Neuhauser-Berthold *et al.*, 2000; Nicklas *et al.*, 1997; Pauly *et al.*, 2000; Roberts *et al.*, 1997; Salbe *et al.*, 1997; Satoh *et al.*, 2003; Toth *et al.*, 1997; Wauters *et al.*, 2002). Some of the discrepancies observed may reflect problems in accounting for the confounding effects of FM on plasma leptin concentration (Neuhauser-Berthold *et al.*, 2000). Administration of leptin has not been shown to affect RMR (Hukshorn *et al.*, 2000; Hukshorn *et al.*, 2003a; Hukshorn *et al.*, 2003b; Mackintosh & Hirsch, 2001; Rosenbaum *et al.*, 2002), but during weight loss (negative energy imbalance) leptin may reduce the increased work efficiency of skeletal muscle, seen in response to energy restriction, and thereby reduce the observed decline in PAEE (Rosenbaum *et al.*, 2003; Rosenbaum *et al.*, 2005). The influence of leptin on energy expenditure is, therefore, unclear.

253. Metabolic stress and fever have also been observed to increase BMR; this is discussed in Appendix 9.

Pharmacological agents

254. Smoking has been shown to increase energy expenditure to a small extent probably through the sympathoadrenal activation by nicotine (Collins *et al.*, 1994; Kimm *et al.*, 2001; Perkins, 1992). Caffeine also increases energy expenditure to a small extent (Arciero *et al.*, 1995; Astrup *et al.*, 1990) with an additive thermogenic effect to nicotine (Arciero *et al.*, 1995; Collins *et al.*, 1994; Jessen *et al.*, 2003; Perkins *et al.*, 1994). For alcohol, while no acute effect has been observed (Perkins *et al.*, 1996), alcoholics have a higher RMR, adjusted for FFM, than healthy, social drinking controls (Addolorato *et al.*, 1998) which falls with abstinence from alcohol (Addolorato *et al.*, 1998; Levine *et al.*, 2000).

255. Administration of glucocorticoids (Chong *et al.*, 1994; Tataranni *et al.*, 1996), adrenaline (Diepvens *et al.*, 2007; Fellows *et al.*, 1985), amphetamines and some anti-obesity drugs (Heal *et al.*, 1998) have all been shown to increase energy

expenditure; whereas, the administration of opiates (Swinamer *et al.*, 1988) and barbiturates (Dempsey *et al.*, 1985) reduce energy expenditure. Growth hormone administration may increase energy expenditure (Wallace *et al.*, 2002), but this may be partly explained by increased FFM (Hansen *et al.*, 2005). β-blockers (β-adrenergic antagonists) have also been shown to reduce RMR (Jung *et al.*, 1980).

Environment

256. With cold exposure, increasing energy expenditure can occur by shivering thermogenesis and increasing muscular activity (Haman, 2006), although this is unlikely to be a significant contributor to energy expenditure for those living at relatively constant ambient temperatures.

257. Studies of adult subjects executing a standard daily activity protocol at different ambient temperatures (16 to 28°C) varied over the short-term, identified an inverse association of ambient temperatures with sedentary TEE (Blaza & Garrow, 1983; Buemann *et al.*, 1992; Dauncey, 1981; Valencia *et al.*, 1992; van Marken Lichtenbelt *et al.*, 2001; van Marken Lichtenbelt *et al.*, 2002; Warwick & Busby, 1990; Westerterp-Plantenga *et al.*, 2002). Whether ambient temperature influences RMR, however, is less clear (Blaza & Garrow, 1983; Buemann *et al.*, 1992; Dauncey, 1981; Lean *et al.*, 1988; Valencia *et al.*, 1992; van Marken Lichtenbelt *et al.*, 2001; van Marken Lichtenbelt *et al.*, 2002; Warwick & Busby, 1990; Westerterp-Plantenga *et al.*, 2002).

258. Seasonal variation in measures of energy expenditure, adjusted for FFM have also been reported, with increased energy expenditure during colder months (Bitar *et al.*, 1999; Goran *et al.*, 1998a; Plasqui *et al.*, 2003; Plasqui & Westerterp, 2004). Seasonal differences in physical activity levels may also occur (Haggarty *et al.*, 1994).

259. Overall, these studies suggest that the variation in energy expenditure due to differences in environmental temperature can account for about 2-5% of TEE. The maintenance of indoor temperatures to within 20-25°C and the use of clothes to control body heat loss, however, mean that changes in ambient temperature are unlikely to have much impact on energy requirements in the UK.

Appendix 4 – Basal metabolic rate prediction equations

260. The factorial calculation of energy reference values as a basal metabolic rate (BMR) multiple for any population group identified in relation to age, gender and size, requires appropriate BMR prediction equations. The FAO/WHO/UNU report (FAO, 2004) calculated BMR with Schofield equations (Schofield *et al*., 1985) (see Table 17). It has been shown that Schofield prediction equations may overestimate BMR in many communities; therefore alternative prediction equations, the Henry equations (see Tables 18 and 19), based on either weight or both weight and height have been developed (Henry, 2005).

261. The validity of all published prediction equations for resting energy expenditure was tested in US and Dutch cohorts (separately) of overweight and obese adults (body mass index (BMI) range 25-40) aged 18–65 years (Weijs, 2008). The US cohort was a subset of those in the Dietary Reference Intakes (DRI) doubly labelled water (DLW) database (IoM, 2005). Against the US cohort, the accuracy of the Henry equations (% of subjects predicted within ±10% of the measured resting metabolic rate, (RMR)) was 79%, which was as good as, or better than, all other equations tested, including the Schofield equations (69% accurate). Against the Dutch cohort however, all prediction equations performed less well.

262. The two sets of prediction equations (Schofield *et al*., 1985 and Henry 2005) were tested against a larger US cohort DRI DLW database (IoM, 2005) in which BMR values were measured for most subjects (n=767, age 20-96, BMI 18.5-62 kg/m^2, men n=334; women n=433). The calculated statistics included accuracy, the root mean squared error (RMSE), and the mean, minimum and maximum % difference (bias) between estimated and measured RMR, as well as mean values for BMR and physical activity level (PAL).

263. The comparison is shown in Table 20. The differences between these prediction equations were small, but both Henry prediction equations, especially those based on weight and height, performed slightly better than the Schofield equations. The inclusion of height, as well as weight, is particularly appropriate as the range of healthy adult body weights used in this report was generated from BMI and height categories. Height was included in the original Schofield equations (Schofield *et al*., 1985), however, height is not generally included when these equations are used. Although there does not appear to have been any recent validation within the current UK population, the Henry equations based on weight and height were chosen to estimate BMR in this report.

Table 17 Prediction equations for BMR[a]: Schofield[b]

Gender	Age (years)	BMR (MJ/day)		BMR (kcal/day)	
		Weight coefficient	constant	Weight coefficient	constant
Males	<3	0.249	-0.127	59.51	-30.4
	3-10	0.095	2.110	22.706	504.3
	10-18	0.074	2.754	17.686	658.2
	18-30	0.063	2.896	15.057	692.2
	30-60	0.048	3.653	11.472	873.1
	>60	0.049	2.459	11.711	587.7
Females	<3	0.244	-0.130	58.317	-31.1
	3-10	0.085	2.033	20.317	485.9
	10-18	0.056	2.898	13.38	692.6
	18-30	0.062	2.036	14.818	486.6
	30-60	0.034	3.538	8.126	845.6
	>60	0.038	2.755	9.082	658.5

[a] Coefficients and constants shown for equations of the form BMR = weight coefficient x weight (kg) + constant

[b] Equations as quoted by FAO/WHO/UNU (FAO, 2004) which derive from Schofield *et al.*, 1985. These differ slightly from those quoted by Henry (2005) which derive from the modified Schofield equations (DH, 1991).

Table 18 Prediction equations for BMR[a]: Henry[b] weight and height

Gender	Age (years)	BMR (MJ/day)			BMR (kcal/day)		
		Weight coefficient	Height coefficient	constant	Weight coefficient	Height coefficient	constant
Males	<3	0.118	3.59	-1.55	28.2	859	-371
	3-10	0.0632	1.31	1.28	15.1	313	306
	10-18	0.0651	1.11	1.25	15.6	266	299
	18-30	0.0600	1.31	0.473	14.4	313	113
	30-60	0.0476	2.26	-0.574	11.4	541	-137
	>60	0.0478	2.26	-1.070	11.4	541	-256
Females	<3	0.127	2.94	-1.2	30.4	703	-287
	3-10	0.0666	0.878	1.46	15.9	210	349
	10-18	0.0393	1.04	1.93	9.40	249	462
	18-30	0.0433	2.57	-1.180	10.4	615	-282
	30-60	0.0342	2.1	-0.0486	8.18	502	-11.6
	>60	0.0356	1.76	0.0448	8.52	421	10.7

[a] Coefficients and constants shown for equations of the form BMR = weight coefficient x weight (kg) + height coefficient x height (m) + constant

[b] Henry, 2005

Table 19 Prediction equations for BMR[a]: Henry weight[b]

Gender	Age (years)	BMR (MJ/day)		BMR (kcal/day)	
		Weight coefficient	constant	Weight coefficient	constant
Males	<3	0.255	-0.141	61.0	-337
	3-10	0.0937	2.15	23.3	514
	10-18	0.0769	2.43	18.4	581
	18-30	0.0669	2.28	16.0	545
	30-60	0.0592	2.48	14.2	593
	>60	0.0563	2.15	13.5	514
	60-70	0.0543	2.37	13.0	567
	>70	0.0573	2.01	13.7	481
Females	<3	0.246	-0.0965	58.9	-23.1
	3-10	0.0842	2.12	20.1	507
	10-18	0.0465	3.18	11.1	761
	18-30	0.0546	2.33	13.1	558
	30-60	0.0407	2.90	9.7	694
	>60	0.0424	2.38	10.1	569
	60-70	0.0429	2.39	10.2	572
	>70	0.0417	2.41	10	577

[a] Coefficients and constants shown for equations of the form BMR = weight coefficient x weight (kg) + constant
[b] Henry, 2005

Table 20 Comparison of BMR prediction equations against the US DRI data set[a]

	BMR		PAL		Accuracy[b]	RMSE	Bias%		
	Mean	sd	Mean	sd	%	Mean	Mean	Min	Max
	kcal/d					kcal/d			
Reported	1524	300	1.71	0.29					
Predicted values									
Henry weight and height[c]	1514	273	1.71	0.29	73	114	-0.32	-33	35
Henry weight[c]	1509	292	1.72	0.30	70	121	-0.32	-33	35
Schofield/FAO[d]	1531	278	1.69	0.28	69	123	1.33	-36	43

[a] IoM, 2005
[b] percentage of subjects predicted within ±10% of the RMR measured
[c] Henry, 2005
[d] FAO, 2004

264. The Henry BMR prediction equations are based on the age bands 18-30 years, 30-60 years and >60 years. The age bands used in this report do not exactly match those presented for the Henry BMR prediction equations and there is no Henry prediction equation for BMR for all ages. The following Henry BMR prediction equation age bands were therefore used to calculate predicted BMR for the age bands used in this report.

Table 21 Clarification of the Henry BMR prediction equation age bands applied in this report

SACN EAR age bands (years)	Henry[a] BMR prediction equation age bands
19-24	18-30
25-34	18-30
35-44	30-60
45-54	30-60
55-64	30-60
65-74	>60
75+	>60
All Adults	30-60

[a]Henry, 2005

265. When creating BMR prediction equations, there is insufficient data to allow equations to be generated for narrow age bands. Consequently, age bands tend to be wide and it is difficult to avoid the overlap between the age bands used for the Henry equations and those used in this SACN report, as seen in Table 21. The approach outlined in this table was considered reasonable because the BMR values estimated using the Henry age bands generally describe average BMR for each age band in this report.

266. In this report, one of the two large data sets of DLW measures of total energy expenditure (TEE) (Subar, 2003) did not measure BMR values. As a result, it was necessary to estimate BMR values using the Henry equations and these BMR values were then used to extract PAL values as measured TEE/estimated BMR = PAL.

Appendix 5 – The physical activity level (PAL) and its use in the prediction of energy requirements

Theoretical aspects of calculation of PAL and its factorial prediction

PAL as an index of TEE adjusted for BMR

267. The daily rate of total energy expenditure (TEE) for individuals or groups can be expressed as a multiple of basal metabolic rate (BMR), which has been defined as the physical activity level (PAL). This allows prediction of TEE and consequent energy reference values as PAL x BMR, each of which represents a physiologically generalisable and predictable term. The introduction of the concept by FAO/WHO/UNU in 1985 (WHO, 1985) was considered a simplifying approach to the determination of energy reference values. Thus BMR, a relatively fixed function of body composition, which is predictable as a function of weight, age and gender, is separated from all other components of TEE which are assumed to be variable. These other components reflect dietary intake, through the heat increment of feeding, miscellaneous thermogenic influences and lifestyle in terms of physical activity. Thus, in principle PAL is an index of TEE adjusted for BMR, which should mean it is independent of weight, age and gender. While most have embraced the concept, important reservations have been expressed (Carpenter *et al.*, 1995; Goran, 2005) with preference given to an alternative approach based on multiple regression techniques to develop prediction models of TEE as a function of measured predictor variables such as body weight or age. These reservations need to be addressed.

268. Firstly, it has been implied (Goran, 2005) that this factorial approach is not sufficiently evidence based. This presumably refers to the approach as introduced by FAO/WHO/UNU (WHO, 1985) when in the absence of an extensive database of measures of TEE, not only BMR but also PAL was predicted from time allocated calculations of physical activity ratio (PAR) values (activity cost/BMR). As discussed below (paragraphs 278-282), there are limitations with PAL values determined in this way but direct assessment of PAL from measured TEE and BMR is evidence based.

269. Secondly, it is argued that body weight predicts more of the variance in TEE than BMR and consequently "the regression based approach provides a more physiologically appropriate model for TEE, as compared to the PAL approach which assumes TEE is composed of multiples of BMR" (Goran, 2005). Body weight could explain more of the variance in TEE than BMR when a) variability of physical activity is relatively small, and b) any influence of weight on physical activity is very marked. In fact, within the data sets examined here, there is little evidence

that these conditions apply. For the adult data set (Appendix 8), the regression of TEE on weight, height, age and gender has the same R^2 value (0.62) as that for TEE on BMR (0.63), with BMR capturing all the variance in TEE associated with weight, height, age and gender in a factorial regression model. For adults in the DLW data sets published in the US DRI report (IoM, 2005), in a multiple regression with weight and BMR, the BMR coefficient (regression coefficient 0.71, p value = <0.0001) was more influential than weight (regression coefficient: 0.05, p = 0.18), which may indicate that BMR explained a larger proportion of the variance of TEE than weight. For children (Carpenter *et al.*, 1995), although variance in physical activity energy expenditure (PAEE) is less marked than in adults, nevertheless within the US DRI data set for children, BMR explained slightly more of the variance in TEE than weight (regression coefficient = 0.59 for BMR and 0.37 for weight). Thus, it is not the case that body weight predicts more variance in TEE than BMR.

270. Since the difference between TEE and BMR is mainly PAEE, which will account for the variance not explained by the BMR, TEE can be assumed to be composed of multiples of BMR, especially in adults but also in children. On this basis, regression models of TEE with body weight are arguably physiologically limited since they fail to account for an important between-individual source of variation in energy expenditure, which is physical activity energy expenditure (PAEE). Regression residuals indicate the range of between-individual variation in TEE and are highly correlated with PAEE and PAL but also include any variation in the BMR component of TEE as a function of the anthropometric variables. Within the adult DLW-determined TEE data set (see Appendix 8), the residuals of a regression of TEE on gender, weight, height and age account for much less of the variance in PAEE (TEE – BMR) in women (68%) compared with men (91%), when examined by linear regression. Given that there are no gender differences in PAL values, the most likely explanation of this relates to the gender differences in BMR as a function of weight, and the adequacy of partitioning differences in the BMR component of the TEE between men and women with a single term in the regression equation. This shows that whereas PAEE is a physiologically transparent quantity, the regression residuals are not. Furthermore, the absolute magnitude of the residual range is less than that of PAEE because residuals comprise only the difference between average PAEE predicted in the regression and individual values. Their maximum range as a fraction of maximum PAEE is 77%, which is a similar value to the slope of their regression on PAEE. This means that the residuals cannot strictly be used as an unambiguous measure of variance in PAEE in terms of their magnitude and distribution. As a result, the use of residuals in terms of predictors of the likely range of variation in PAEE within population groups is limited and they have not been used as such in any previous report.

271. Thirdly, a theoretical argument against the BMR multiple approach has been presented (Goran, 2005) that: "the PAL model assumes a linear relationship between TEE and BMR that has a slope equivalent to PAL and a zero intercept" and that "the presence of significant and variable intercepts in the regression equations relating total energy expenditure to either BMR or weight invalidates the use of the traditionally used ratios (i.e. TEE/BMR or TEE/body mass) for expressing total energy expenditure data".

272. The BMR multiple approach as used in this report makes no assumptions about the regression relationships between TEE and BMR and such relationships are not strictly relevant to the discussion of the use of PAL within the factorial model of describing TEE. This is because it has never been suggested that PAL should be calculated as the slope of a linear regression of TEE on BMR for a population group. With information on TEE and BMR, individual subject PAL values can be calculated. From this, the distribution of PAL values for the population group can be calculated and the way in which PAL varies with age, body weight and any other demographic variable can be usefully examined. Indeed in the case of gender, which could influence TEE as some have suggested through gender differences in PAEE as well as through its known influence on BMR, a multiple regression approach to the role of gender on TEE would be unable to distinguish between these possibilities. Only the factorial approach based on measured BMR, measured TEE and calculated PAL allows any gender influences on PAL to be examined in a straightforward way. This has been the approach adopted for this report.

273. Much of the criticisms and confusion over the BMR multiple approach arise from attempting to fit linear regressions to DLW data derived from studies of children. In this case, there does appear to be somewhat less variance in physical activity at any particular age or weight, and in addition, there is on average an increase in physical activity and consequent PAL values with age and weight. This means that the relationship between TEE and BMR is not linear. With small data sets analysed by linear regression the between-individual variation in PAEE will give varying slopes and intercepts which will be inversely correlated. It is therefore not surprising that this has been observed by Carpenter *et al* (1995). In the FAO/WHO/UNU report (FAO, 2004), while a simple linear regression of TEE on weight was satisfactorily applied to infants in the first year of life, for children aged 1-18 years a quadratic polynomial regression equation of TEE on weight was derived from the DLW TEE data. PAL values were then extracted from this analysis by comparing TEE with BMR for each age group. These PAL values increased with age and the variance in PAEE at any age was expressed by calculating PAL values ±15% for children after the age of five years. This change with age in PAL is apparent in the data set of mean TEE, BMR and PAL values assembled in this report for children (see Appendix 12). There is an obvious and marked increase in PAL after the age of three years and then to a lesser extent in older children (Figure 12 Appendix 12) i.e. the plot of TEE on BMR is clearly curvilinear as PAL increases with age. Only when weight becomes a minor predictor of PAEE but explains most of the variance in BMR will the slope of TEE on BMR tend towards the mean PAL and the intercept tend towards zero. This is observed within the combined OPEN-Beltsville adult data set (Moshfegh *et al.*, 2008; Tooze *et al.*, 2007) assembled for this report. As shown in Figure 8 (Appendix 8), the intercept of the line of best fit of TEE on BMR is not significantly different from zero and the slope is similar to the median PAL.

274. The question of variation of PAL with body weight i.e. the influence of body weight on the energy cost of specific activities is nevertheless an important issue that needs to be examined separately. When the use of PAL was introduced by FAO/WHO/UNU (WHO, 1985) PAL values were estimated with factorial calculations

of time allocated energy costs of individual activities, expressed as PAR values. These were used to identify categories of activities based on lifestyles, so that those engaged in identifying energy requirements could make such calculations for specific population groups. To this end, lists of PAR values for activities are reproduced in the recent FAO/WHO/UNU report (FAO, 2004) (on the basis of a comprehensive review (Vaz *et al.*, 2005). MET values for various activities are listed in the US DRI report (IoM, 2005). Thus, each of these reports assumes that having derived a PAL value for a particular lifestyle, it can apply equally to individuals regardless of body weight. Careful calorimetric studies have shown, however, that this is not strictly the case: i.e. PAR values increased with weight (Haggarty *et al.*, 1997). The directly measured energy costs of a fixed programme of work expressed as a BMR multiple increased with body weight between 48 and 80kg by about 13% of the mean cost of the activity. This was consistent with theoretical calculations reported by the authors. The implications of this are that the energy requirements of adults engaged in similar tasks will be higher in larger compared with smaller adults. The authors showed that for a 40kg adult, the energy requirement calculated as PAL x BMR with PAL derived from measurements of PAR values for the specific activities in a 70kg adult will be an overestimation of about 10%.

275. The effect of body weight on PAR values for specific activities is only of practical importance, however, when it is assumed that PAL can be calculated with some confidence from a factorial, time-allocated list of the PAR values. As discussed below (paragraphs 278-282), such predictions of PAL values are unlikely to be accurate and are not recommended in this report.

276. The final issue of general importance for this report is whether there is an influence of gender on PAL. Gender differences in PAL and hence the predicted energy requirement was a feature of the 1985 FAO/WHO/UNU energy report (WHO, 1985) and the 1991 COMA DRV report (DH, 1991). Gender differences in behaviour are said to influence overall TEE, and therefore PAL values, for similar lifestyles or activities (Erlichman *et al.*, 2002). However, most studies have failed to identify differences between men and women in PAEE or PAL, and both recent reports argue that average energy costs of activities expressed as a multiple of BMR, or PAR, should be similar for men and women (FAO, 2004; IoM, 2005). Within one meta-analysis of DLW-derived TEE values, PAL values were observed to be 11% lower in women than men, but it was not possible to identify whether this reflected an absence of subjects recruited from more active groups or a general tendency of women to be less involved in strenuous activity (Black *et al.*, 1996). In the DLW data sets examined in this report, no differences in PAL with gender were identified for children or adults.

277. In summary, therefore, none of the concerns expressed above regarding the use of the BMR-multiple approach are likely to be of importance within the framework of this SACN report. As developed below, estimates of PAL values for population groups are best derived from individual measurements of TEE and BMR and not by regression approaches. Within the adult data set assembled here, there is no evidence of any significant variation of PAL with either weight or gender (Appendix 8). For children, individual PAL values do show some increase with age and weight but

these are almost certainly behavioural changes with development that in no way detract from the validity of the use of the BMR-multiple approach.

Factorial prediction of PAL

278. When the concept of PAL was introduced by FAO/WHO/UNU in 1985 (WHO, 1985) it was implicit that PAL values could and would be estimated from the time allocated summation of individual activity energy costs expressed as PAR values. This formed the basis of the 1991 COMA report on DRVs (DH, 1991), and the principle is embodied within the FAO/WHO/UNU report (FAO, 2004) and the US DRI report (IoM, 2005). This approach is not adopted in this report for the following reasons.

279. Firstly, there is disagreement about how to use published PAR values for the calculation of PAL. The FAO/WHO/UNU (FAO, 2004) used PAR values from a list compiled specifically for the report (Vaz *et al.*, 2005). The authors of the US DRI report (IoM, 2005) express concern that factorial calculations from published PAR values may underestimate daily TEE because of a lack of specific inclusion of energy expenditure associated with feeding, the themic effect of feeding (TEF) and/or excessive post exercise oxygen consumption (EPOC). In the US DRI report (IoM, 2005), PAR values were derived from a list of MET values for specific activities (1MET = a fixed rate of oxygen consumption/kg). The MET values were converted to the BMR multiple PAR values (a ≈7% reduction) and then increased by 26.5% to account for TEF (10%) and EPOC (15%: i.e. increase by 1.15*1.10, see legend to Table 24). Thus, the factorial calculations in the US DRI report were made from values that were about 26.5% greater than those in the FAO/WHO/UNU report (FAO, 2004). Since there is no evidence that this is justifiable in all cases, some PAR values may be overestimated by up to 26.5%.

280. Secondly, even if PAR or ΔPAL values are known with certainty, variation in spontaneous physical activity (SPA) (Snitker *et al.,* 2001) (see paragraphs 224-227 Appendix 3), both within and between specific designated activities can introduce considerable error in factorial predictions of PAL. The concept of SPA or non exercise activity thermogenesis (NEAT) (Levine, 2007) is that energy expenditure is variable between individuals because of the variable expression of a behavioural phenotype associated with high levels of physical activity: i.e. SPA is an "inherent propensity to locomotion" (Snitker *et al.,* 2001). This concept derived from observations of greater than expected variation in TEE during calorimeter studies where "workout" type activity is restricted. In these circumstances, activity is limited to daily living and postural changes yet PAL values ranged from 1.2-1.724, (Zurlo *et al.,* 1992; Snitker *et al.,* 2001) (see paragraph 283). In free-living circumstances the potential for such behaviour is more marked (Levine, 2007) and calorimeter- measured PAL values predict the range of higher free-living PAL values (Snitker *et al.,* 2001). It is highly unlikely that subjects with a high SPA phenotype would ever exhibit the range of PAL values associated with the sedentary category of 1.40-1.69 identified by FAO/WHO/UNU (FAO, 2004) even if they had the seated occupations assumed for this category.

281. Thirdly, some individuals may have what is to some extent the opposite of the SPA phenotype, exhibiting marked compensatory *reduced* activity after periods of intense activity. Such a phenotype has not been widely investigated but has been reported in at least one study in which quite variable PAL values were observed in subjects engaged in highly controlled work activities (Haggarty *et al.*, 1997). PAL varied markedly between 1.53 and 2.08 mainly because of marked differences in discretionary activities. Thus, subjects with the lowest DLW-determined PAL values (1.58, n=5) reverted to the basal state between-work periods exhibiting mean PAR values for discretionary energy expenditure of 1.02. The rest of the group exhibited discretionary PAR values averaging 1.89 (n=8) with higher average PAL values (1.95).

282. The existence of such behavioural phenotypes is increasingly being recognised within the context of energy balance regulation (Levine *et al.*, 1999; Zurlo *et al.*, 1992), although their frequency within populations is currently unknown. Nevertheless, for such phenotypes, TEE is unpredictable within factorial models that have been applied to date. In other words, there may be a continuous spectrum of behavioural activities ranging from, on the one hand fidgeting while being otherwise stationary and choosing to walk up escalators, to on the other hand, adopting a resting posture whenever the possibility presents. The extent of this is indicated in studies reviewed below showing the degree of variation in individual PAL values within specific activity categories.

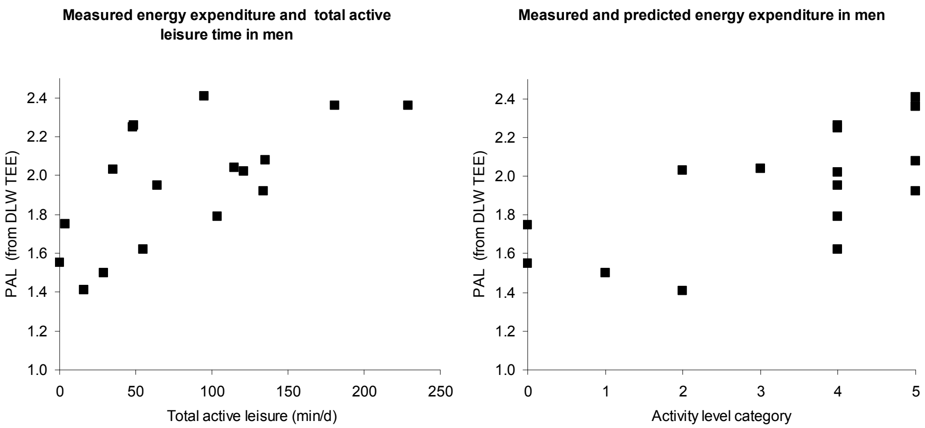
Observed variation in PAL within specific population groups

283. Many published studies of PAL values of population subgroups only include mean values, but where individual values are reported a much greater range of PAL values is often observed than would be anticipated. As already indicated (paragraph 280), 24 hour restricted activity studies within a calorimeter have repeatedly shown wide variation in PAL values: from 1. 15-1.7 in one report of 177 subjects (Ravussin *et al.*, 1986) and from 1.20-1.65 in a second report (Snitker *et al.,* 2001), in the latter case with individual values correlating with free-living DLW-measured PAL (1.35-2.15). PAL values of urban Chinese adults with manual or sedentary occupations classified into activity categories varied within these categories by up to 0.7 (Yao *et al*., 2002). Within a group of inactive men and women studied prior to a training programme the range of PAL values was 1.5-2.2 (Westerterp *et al.*, 1992). In a study of older people investigating energy expenditure, self reported physical activity, and mortality, there was no relationship between self reported activity and PAL even though PAL predicted mortality (Manini *et al.*, 2006).

284. The difficulty of predicting PAL values in terms of reported or measured physical activity is exemplified by a study of men with sedentary occupations but varying levels of physical activity (Haggarty *et al.*, 1994). Figure 5 shows a comparison of PAL with either measured activity time or activity categories predicted by the authors from the activity diaries. The relationship between measured PAL and recorded physical activity is only moderate. Thus, total active leisure time and activity level category explained only 41 and 48% of the variation in PAL. Subjects with a PAL ≥2, recorded leisure activities ranging from 35 to 229 minutes per day and were categorised into categories two to five. Within three of the five

categories individual PAL values varied by 0.6 and the difference in PAL between the lowest subjects in category five and the highest in category one was only 0.17 PAL units. The authors of this study emphasised the large differences that can occur between highly active individuals with a large exercise capacity compared with untrained individuals in the potential energy expended in tasks that are only loosely defined within activity diaries. This explains some of the poor correlation shown in Figure 5. Whatever the explanation for the discrepancy, it is clear that if the energy requirements of these subjects were individually predicted from their activity categories, there would be substantial errors.

Figure 5 – Relationship between PAL and measured leisure time activity and categorised activity level[a]



[a]*Haggarty et al., 1994*

285. Within a data set of DLW studies in healthy adults assembled for this report, all of the studies which reported PAL values (n=63) were examined for descriptions of the activities/lifestyles of the subjects. Just over half the studies (n=33) provided no information, but for those that did (n=30), the mean PAL values were assigned to categories of light, moderate or heavy activity, with those studies with a mixture of activities assigned to moderate. The values in Table 22 show that, notwithstanding the somewhat arbitrary assignment to activity categories, the range of mean study PAL values is considerable, especially in the "light" category (1.23-1.98).

Table 22 Mean PAL values from DLW studies assigned (where possible) to activity groups

		PAL value	
Number of studies	Activity category[a]	mean	range
33	none	1.73	1.42-1.97
15	light	1.67	1.23-1.98
12	moderate	1.84	1.63-2.01
3	heavy	2.09	1.91-2.48
63	all	1.75	1.23-2.48

[a] Activity categories assigned based on written comments by study authors

286. Although an exhaustive review has not been conducted for this report, it is clear that considerable variation in overall rates of TEE and PAL occur within subjects who would be classified as exhibiting similar lifestyles. As the values shown in Table 22 are mean study values, the range of individual values will, in fact, be much wider. The extent of inter-individual variability in PAL is hard to judge, however, because so few studies report individual PAL values within occupational or lifestyle categories. Thus, with the exception of groups at the extreme of the range (either very restricted or with high levels of physical activity), the relationship between PAL and lifestyle, or even self-reported physical activity, appears to be weak. Consequently, predictions of PAL values are unlikely to be accurate and are not recommended in this report.

Magnitude and variation in PAL within the general population

Observed lower and upper limits of PAL

287. The FAO/WHO/UNU 1985 report (WHO, 1985) identified the lower limit of PAL, a 'survival' value, to be 1.27, which is consistent with studies assembled elsewhere in non-ambulatory chair-bound and non-exercising subjects confined to a calorimeter (where PAL values of 1.17-1.27 were observed) (Black, 1996).

288. The lower limit of energy expenditure in subjects who are ambulatory but only exhibiting the minimal activities associated with daily living (e.g. grooming, showering and dressing), equates to a PAL value between 1.35 and 1.4 (Alfonzo-Gonzalez *et al.*, 2004; Goran *et al.*, 1994b; Warwick, 2006). In grossly obese subjects confined to a whole-body calorimeter following a standardised sedentary protocol apart from two 30 minute exercise periods (30 minutes of cycling at 25W and 30 minutes of stepping on and off a 20-cm block at a rate 40/minute), mean PAL values were 1.35 (1.27-1.42) (Gibney *et al.,* 2003). In the healthy, most elderly, the mean PAL value (after trimming for PAL values <1.1) was 1.38 (Rothenberg *et al.*, 2000).

289. The US DRI report compiled a list of activities of daily living, which accounts for about four hours in total and amounts to a ΔPAL of 0.29 (IoM, 2005). This is reported to equate to a sedentary PAL of 1.39, i.e. 1+0.1(TEF) +0.29. Such calculations, however, can only be relevant for subjects in the basal state for 20 hours per day. Further, the listed activities may overestimate actual costs since they include additions of energy expenditure (≈28%) to allow for corrections in TEF and EPOC.

290. Overall, a PAL value of 1.38 seems the likely lower limit for free-living individuals. This indicates that the *upper limit* of the sedentary PAL range (1.4) suggested in the US DRI report is too low.

291. The upper limit to human physical activity is that exhibited for limited periods of time by elite endurance athletes and soldiers on field exercises, for whom PAL values between 3 and 4.7 have been reported (Black *et al.*, 1996; Hoyt & Friedl, 2006). The maximum PAL value associated with a sustainable lifestyle within the general population, however, appears to be about 2.5 (Black *et al.*, 1996; Westerterp

& Plasqui, 2004). This value has been supported by subsequent studies in long-term exercising women (Withers *et al.*, 1998) and physically active men (Black *et al.*, 1996; Davidson *et al.*, 1997; Haggarty *et al.*, 1994; Westerterp & Plasqui, 2004).

Observed magnitude and variation in PAL

292. Several large data sets of TEE measures containing individual PAL values have been made available for this SACN report. These include the US DRI data set (IoM, 2005), the Beltsville study (Moshfegh *et al.*, 2008) and the OPEN study (Subar *et al.*, 2003; Tooze *et al.*, 2007). Although the OPEN study did not measure BMR, this has been estimated using the Henry BMR prediction equations based on weight and height (Henry 2005). The distribution statistics for these three large data sets are shown below in Table 23. Details of the OPEN and Beltsville cohorts are given in Appendix 8.

293. The US DRI data set (IoM, 2005) of adults aged 18 or more years (n=767, 360 overweight or obese and 407 normal weight) includes the data in the Black *et al*. (1996) analysis. There is a fall in PAL with age, most notably in terms of lower values in the small sample at ages greater than 80 years for both the normal and overweight/obese populations. Gender differences in mean PAL values are only apparent within the overweight/obese group, but the difference is small. The characteristics of this data set shown in Table 23 are those after trimming to exclude the very old (aged >80 years). The median PAL (1.72) is higher than the median PAL values for the OPEN and Beltsville studies. The US DRI data set is, however, a collection of many individual studies with much smaller sample sizes, and cannot be considered to be representative of a normal adult population. There may be an over representation of subjects with PAL values >2, i.e. very physically active subjects, as indicated by the 90th centile value of 2.10 compared with 1.96 and 1.92 for the Beltsville and OPEN studies, respectively.

Table 23 Distribution of PAL values of large data sets[a,b,c]

Distribution boundaries	US DRI[a] (n=724; age 18-80y)	OPEN[b] (n=451; age 40-69y)	Beltsville[c] (n=478; age 30-69y)
10th centile	1.38	1.40	1.32
lower quartile	1.55	1.49	1.46
median	1.72	1.61	1.62
upper quartile	1.92	1.77	1.78
90th centile	2.10	1.92	1.96

[a] IoM, 2005
[b] OPEN data set (Subar *et al*., 2003; Tooze *et al*., 2007)
[c] Beltsville data set (Moshfegh *et al*, 2008)

294. In the OPEN study (Subar *et al.,* 2003), mean PAL values fall slightly with age (from 1.70 at 40 years to 1.57 at 70 years), are slightly higher for women than men (by 0.04 units), but are independent of weight or BMI. The distribution pattern of PAL values is clearly shifted downwards compared with the US DRI data set values.

295. In the Beltsville study (Moshfegh *et al.,* 2008), there is little change in PAL with age or with BMI (by regression or with category). Thus, the possibility that the energy cost of physical activity may be affected by adiposity (Byrne *et al.*, 2005; Forsum *et al.*, 2006; Leenders *et al.*, 2001) does not seem to influence the range of PAL values within normal and obese subjects. The distribution is similar to the OPEN study for the lower and upper quartiles and median, which, to some extent, lends confidence to the calculation of BMR in the OPEN data. For the Beltsville study, however, the 10th centile PAL value is slightly lower (1.32) and the 90th centile PAL value is slightly higher (1.96), than in the OPEN study (1.40 and 1.92).

296. The characteristics of PAL values within the combined OPEN-Beltsville cohort are shown in Appendix 8.

Observed effect of additional physical activity on PAL

297. The energy cost of additional amounts of sport or strenuous leisure activity has been considered in terms of ΔPAL values. One widely quoted meta-analysis of DLW studies (Black *et al.*, 1996) reported that mean PAL values increase from 1.63 to 1.99 with imposed activity (physical training) on a low activity background.

298. Adults with normally sedentary occupations who did not exercise or play sport on a regular basis increased their PAL value by 0.41 (from 1.59 with a range 1.43-1.68, to 1.99 with a range 1.66-2.42) with a nine week incremental programme of jogging up to one hour/day, five days/week (Bingham *et al.*, 1989). Inactive men and women following a 40 week incremental running programme sufficient to enable running a half marathon, increased their mean PAL values by 0.44 PAL units above their initial range of 1.5-2.2 (Arthur *et al.*, 1987): obese boys achieved a mean increase of 0.27 with a four week training program of cycling for 45 minutes five times/week at 50-60% of VO_2max (i.e. from 1.77 (±0.15) to 2.04 (±0.15) (Blaak *et al.*, 1992). The implications of these studies in the light of theoretical calculations of the likely response of PAL to exercise are considered at the end of this section.

299. Individuals involved in competitive sport or who have habitual high levels of physical activity exhibit PAL values up to 0.6 units higher than those who do not take regular exercise. In a small number of healthy men, there was a mean difference in PAL of 0.64 between competitive runners (PAL = 2.26) and those reporting no leisure activity (Davidson *et al.*, 1997). In women aged 50-70 years there was a mean difference in PAL of 0.61 between long-term exercisers (PAL= 2.48: range 1.60–3.43) or long-term non-exercisers, (PAL= 1.87: range 1.63–2.25) (Withers *et al.*, 1998).

Predicting the effect of additional physical activity on PAL

300. The US DRI report (IoM, 2005) lists the energy cost of various activities in terms of METs and ΔPAL values. Selected examples, including those for walking, are given in Table 24, together with actual additional rates of energy expenditure associated with the activities calculated for a standard woman (57kg age 30-60 years) and man (70kg age 30-60 years) (Henry, 2005). As indicated above, such ΔPAL values are calculated from METs after adjustment of the equivalent PAR value to include an additional 26.5% to account for TEF and EPOC. The calculations are similar but

not exactly the same (see footnote b of table 24) and could be overestimates if the MET values were not obtained in subjects in the basal state. They will be higher than equivalent values quoted by FAO/WHO/UNU (FAO, 2004) and others (Vaz *et al.*, 2005). The ΔPAL values are the increases in the daily PAL expected when the one hour of the activity (mean PAR-1/24) replaces the BMR. The ΔPAL values shown in Table 24 are slightly lower than those in the US DRI report (IoM, 2005) because the reference BMR values are slightly different and the additions for EPOC and TEF are made a little differently (x 1.15 x 1.10 =+26.5% in this report and x 1.15 x 1/0.9=+27.8% in the US DRI report). The PAR values can be compared with directly measured values in men with light occupations and variable leisure activities while walking at moderate pace (2.54), walking briskly or carrying a load (4.09) and jogging or running (13.1) (Haggarty *et al.*, 1994). By and large the values compare well.

Table 24 Influence of activities on PAL

Activity	METs[a]	PAR[b]	ΔPAL/10 min	kJ(kcal)/10 min		ΔPAL /h	kJ(kcal)/h	
				woman	man		woman	man
Walking (2mph)	2.5	2.9	0.013	71(17)	91(22)	0.08	436(104)	559(134)
Walking (3 mph)	3.3	3.9	0.02	109(26)	140(33)	0.12	654(157)	839(201)
Walking (4mph)	4.5	5.3	0.03	164(39)	210(50)	0.18	981(235)	1258(301)
Tennis (doubles)	5	5.9	0.034	185(44)	238(57)	0.2	1091(261)	1398(335)
Dancing	6	7.1	0.042	229(55)	294(70)	0.25	1363(326)	1748(418)
Roller Skating	6.5	7.7	0.046	251(60)	322(77)	0.28	1527(365)	1957(469)
Swimming	7	8.2	0.05	273(65)	350(84)	0.3	1636(392)	2097(502)
Walking (5 mph)	8	9.4	0.058	316(76)	405(97)	0.35	1908(457)	2447(586)
Jogging (6 mph)	10	12	0.08	436(104)	559(134)	0.46	2508(600)	3216(770)
Rope skipping	12	14.1	0.091	496(119)	636(152)	0.55	2999(718)	3845(920)
Squash	12	14.1	0.091	496(119)	636(152)	0.55	2999(708)	3845(920)

[a] 1 MET =0.0175 kcal/minute/kg

[b] Energy expenditure as a multiple of the BMR, calculated from METs as follows.

1. Conversion to PAR BMR/multiple values: BMR is calculated as mean BMR value for reference women (57kg age 30-60 years)=0.0159kcal/min/kg), and reference man (70kg age 30-60 years) =0.0166kcal/min/kg: Henry BMR prediction equations (Henry, 2005). The resulting PAR value is 7% lower than the MET value.
2. Addition of 26.5% for EPOC and the thermic effect of feeding. Note that ΔPAL values are slightly lower than those in the DRI report because the reference BMR values are slightly different and because the additions for EPOC and TEF are made slightly differently (x 1.15 x 1.10 =+26.5% here and x 1.15 x 1/0.9=+27.8% DRI report (IoM, 2005)).

Overall these changes mean that the PAR value is 17.7% greater than the MET value.

301. It is important to recognise that the overall effect of an additional activity at some fixed rate will have a variable effect on the PAL value according to the magnitude of energy expenditure which it replaces, even if no other changes in energy expenditure occur.

302. In the US DRI report (IoM, 2005) the factorial calculations of PAL values from MET and ΔPAL values for various activities, such as those shown in Table 24 (e.g. walking (4mph) ≡ 0.18 ΔPAL) involve an assumption that each activity replaces the BMR. This implies in turn that its effect on PAL will be the same for all individuals. In practice, much of the non-sleeping time energy expenditure is greater than BMR and may even be much higher for those exhibiting a high SPA phenotype. The effect of an additional activity, such as one hour/day walking at 4mph, will be to replace activity likely to be at a higher rate than the BMR and will, therefore, result in a lower increase in PAL. Thus, individuals with a low rate of background discretionary activity will experience a higher overall increase in PAL for the extra activity, than those with a higher background rate.

303. This is shown in Table 25, where one hours walking at 4 and 5 mph or jogging 10 minute miles (ΔPAL values of 0.18, 0.35 and 0.46, PAR values of 5.3, 9.4 or 12.0), replace one hours activity in subjects exhibiting PAL values of 1.5, 1.6 or 1.7. Such PAL values in subjects sleeping for eight hours a day imply average discretionary PAR values of 1.75, 1.9 and 2.05. Substitution of an average hour of discretionary activity with one hour of the increased physical activity will result in actual new PAL and ΔPAL values which are 10-25% less than the listed ΔPAL value for the activity. For high SPA phenotypes additional planned activity may have little effect on overall energy expenditure and PAL. Only if the new activity replaced a period of complete inactivity (PAR=1) would the expected increase be observed. If the additional activity resulted in a period of compensatory reduced activity as discussed above, the overall effect would be even less than indicated. These calculations show the difficulty of calculating a planned change in energy expenditure. In practice, however, because the variation in the magnitude of ΔPAL with mean discretionary PAR is small, and given the insecurities involved in such calculations, this uncertainty is not considered in this report.

Table 25 Influence of specific additional activities on PAL in subjects with varying initial PAL values

		1 hours additional planned activity		
		4mph walking	5mph walking	10min mile jogging
	PAR[a]	5.3	9.4	12.0
	ΔPAL[b]	0.18	0.35	0.46
Initial PAL	Mean discretionary PAR[c]	new PAL[d] (actual ΔPAL[e])		
1.5	1.75	1.65 (0.148)	1.82 (0.320)	1.93 (0.427)
1.6	1.9	1.74 (0.142)	1.91 (0.313)	2.02 (0.421)
1.7	2.05	1.84 (0.135)	2.01 (0.307)	2.11 (0.415)

[a] Values from Table 24
[b] = (PAR-1)/24: assuming the activity replaces a period at the BMR
[c] Assuming 8 hrs at PAR =1(sleeping)
[d] Calculated as the1hr activity replacing 1 hr at the mean discretionary rate
[e] New PAL-initial PAL

304. Overall, these theoretical calculations indicate that PAL can be increased by about 0.2 for one hours brisk walking (with brisk defined as slightly faster than 4mph), about 0.4 by one hours jogging at 6 mph, and up to about 0.6 by an intense aerobic exercise programme associated with training for competitive sport. This allows a re-examination of a widely quoted statement derived from a DLW meta-analysis (Black, 1996; Black *et al.*, 1996) that 30-60 minutes of active sport 4-5 times per week can raise PAL by about 0.3 units. On the basis of the ΔPAL values in Table 25 and if 'active sport' is equivalent to jogging at ΔPAL per hour of about 0.43, then 30 minutes a day for four days a week and 60 minutes a day for five days a week would raise PAL on average by about 0.12 and 0.31, respectively. Thus, a more accurate statement would be that 60 minutes of active sport five times per week can raise PAL by about 0.3 units and this is generally consistent with the observed effects discussed in paragraphs 297-299 above.

PAL values in relation to health outcomes

305. The US DRI report discusses a 'Physical Activity Level consistent with a normal Body Mass Index' in terms of one hour of moderately intensive physical activity (walking at 4 miles per hour) resulting in an increase of 0.2 PAL units (IoM, 2005). The FAO/WHO/UNU report identified a desirable PAL value of 1.75 or more (FAO, 2004).

306. Information on the relationship between PAL and mortality has been published in a prospective study of healthy older adults (n=302; aged 70 to 79 years) (Manini *et al.*, 2006). TEE was determined using the DLW technique and BMR by indirect calorimetry. Over an average of 6.15 years of follow-up, participants in the upper tertile of PAEE (PAL greater than 1.78) had a reduced risk of all-cause mortality (HR 0.43, 95% CI 0.21-0.88; P_{trend} = 0.02) compared to those in the lowest tertile (PAL less than 1.57). Thus, this objectively measured free-living PAEE was strongly associated with lower risk of all-cause mortality in these healthy older adults. The published Kaplan-Meier Survival Plots indicate that the separation of survival statistics between the tertiles did not occur until after two years following the initial measures. This suggests that the increased mortality in the lowest PAL tertile was not due to reverse causality (i.e. subjects exhibiting low PAL values because they were ill and at increased risk of mortality), but this cannot be ruled out. Although the intensity and type of physical activity was not objectively measured, physical activity questionnaires suggested that the proportion of individuals who reported high-intensity exercise and walking for exercise, in terms of both duration and intensity, was similar across tertiles of free-living activity energy expenditure. This implies that simply expending energy through any activity may influence survival in older adults and that specific, high intensity exercise *per se* may not be required to produce health benefits.

Utilising PAL values to determine reference energy intakes

307. Notwithstanding the uncertainties of predicting PAL for population groups, it remains the case that all previous dietary energy recommendations have recognised the need to include a variable physical activity factor in the derivation

of energy requirements for school-aged children and adults. Although criticisms have been levelled at the BMR x PAL approach, no satisfactory alternative has been identified. There are two major considerations in the practical application of this approach: identifying suitable PAL values appropriate for groups and populations; and utilising this information to derive energy reference values.

308. In the past, starting with the FAO/WHO/UNU report (WHO, 1985), a range of PAL values has been determined by factorial calculations which equate to occupation and lifestyle. This has been with the intention of providing guidance to the health-care professional using the reports in making a judgement about the appropriate PAL value for individuals and population groups. The 1991 COMA DRV report (DH, 1991) required a choice from a 3 x 3 matrix of PAL values (three occupations, three leisure activities), for each gender over a range of 1.4-1.9 for men and 1.4-1.7 for women. In addition, a table of EAR values calculated for nine PAL values from 1.4 to 2.2 is was given. The FAO/WHO/UNU report (FAO, 2004) classified the intensity of a population's habitual physical activity into three categories identified by a range of PAL values for each category: sedentary or light activity 1.40-1.69, active or moderately active 1.70-1.99, vigorous 2.00-2.40, with worked examples using the midpoint of these three ranges. The report then listed tables of daily average energy requirements calculated for six PAL values (1.45, 1.60, 1.75, 1.90, 2.05 and 2.20) with further worked examples for individuals or groups with PAL values not included in the list (e.g. PAL =1.8). The implication of both the COMA (DH, 1991) and FAO/WHO/UNU (FAO, 2004) reports is that it is possible to utilise the information on PAR values for activities to predict PAL values for specific population groups to within 0.05-0.1 of a PAL value.

309. The US DRI report (IoM, 2005) included physical activity factors in the prediction equations for TEE for men and women based on weight, height, age and the physical activity coefficient (PA)[33].

310. The physical activity (PA) coefficient, which scales the weight and height factors, results in the equation containing a form of PAL x BMR, since the height and weight factors should capture the BMR data (in fact, calculations with the published DLW data set within the report, show that the prediction equations calculate the same values for TEE as PAL x estimated BMR). The PA coefficient is not PAL *per se*, but constants assigned to each of the four PA categories (sedentary, low active, active, or very active). For men these were: 1.0, 1.11, 1.25 or 1.48; and for women: 1.0, 1.12, 1.27, or 1.45. In effect, this results in four parallel prediction equations for each gender, one for each PAL category. The user of the report must choose the appropriate PA coefficient by locating the individual or population group under consideration within one of four PAL ranges. It is not clear how these PAL ranges have been derived, but factorial calculations of PAL are derived in the report from lists of ΔPAL values for various activities. The examples shown are for PAL values of 1.39 (sedentary), 1.49 (low active), 1.75 and 1.77 (active) and 2.06 (very active). Assuming

33 PA is the physical activity coefficient, which depends on whether the individual is estimated to be in the sedentary (PAL ≥ 1.0-1.4), low-active (PAL ≥ 1.4<1.6), active (PAL ≥ 1.6<1.9) or very active (PAL ≥ 1.9 < 2.5) PAL categories.

that the sedentary PA factor of 1.0 is equivalent to a PAL of 1.39, then the other PA factors in the equation are equivalent to PAL values of 1.54, 1.74 and 2.06 (men) and 1.56, 1.77 and 2.02 (women), which generally correspond to the worked examples.

311. The user of the US DRI report (IoM, 2005) is required only to locate an individual within a PAL range rather than a precise PAL value, and consequently this makes it, to some extent, simpler than FAO/WHO/UNU (FAO, 2004) procedure. Indeed, with a lower limit of PAL for free-living individuals of about 1.38 (see paragraph 290), few subjects in any population would be assigned to the sedentary category leaving the choice to be made between one of the other three categories. In this SACN report, the difficulty in choosing between adjacent PAL categories has been highlighted. Miscategorisation between adjacent categories would result in predicted TEE, and hence energy reference values, being 13-18% too high or low for subjects in the middle of each category, or up to 40% for individuals at the top or bottom of ranges.

312. It is clear from the above discussion that the inter-individual variability of PAL is such that it is an unrealistic expectation that PAL values can be predicted for specific lifestyle-dependent population groups with the precision implicit in any of these previous reports (FAO, 2004; IoM, 2005). Furthermore, little can be said about the health implications of specific PAL values apart from the general desirability for values to be within the higher rather than lower range of values. Nevertheless, the definition of TEE and the energy requirement for a population group on the basis of PAL x BMR remains a sound principle and an alternative approach to its use is therefore required.

313. The simplest approach is to decide that TEE for a population group can only be predicted on the basis of direct measurements of TEE for reference populations from which average PAL values and their distribution can be identified. Thus, medians and ranges of PAL values observed in the reference populations can be applied to similar population groups in terms of age, BMI and gender, assuming only that reference population groups are appropriate. TEE is then predicted as a function of the BMR. Dietary reference values can then be framed against such distributions. Such framing could involve an average (median) reference intake for the population together with lower (e.g. 25th centile) and higher (e.g. 75th centile) values identified for those representing the less active or more active sections of the population and with additional amounts of energy likely to be needed for lifestyle changes associated with additional activities. Identifying objective measures of activity allowing assignment of populations to the less or more active sections is an important research task.

314. Such advice is clearly a major departure from previous approaches for adults although it is in principle similar to the method adopted by FAO/WHO/UNU (FAO, 2004) in their recommendations for children. However, it is an approach which recognises the reality that prediction of rates of TEE for individuals or population groups is inherently uncertain.

Appendix 6 – Doubly labelled water (DLW) method

315. In 2009, the International Atomic Energy Agency (IAEA) published information on the theoretical background, as well as the practical application of state of the art methodologies, to monitor total energy expenditure (TEE) using stable isotopes (and changes in body composition). IAEA reviewed recent advances in analytical techniques developed by an international group of experts which readers are referred for more detailed information (IAEA, 2009). The doubly labelled water (DLW) method is a minimally invasive stable isotopic technique of measuring carbon dioxide (CO_2) production in free-living subjects over a period of several weeks. The subject drinks a weighed amount of the DLW containing known amounts of the stable isotopes of hydrogen (^2H) and oxygen ($^{18}O_2$), based on their body weight. The isotopically labelled water equilibrates with normal body water and a sample is taken typically after about five hours to measure the initial isotope enrichment, which also indicates the size of the total body water pool from isotope dilution. As water is lost from the body in urine, sweat and evaporation from the lungs during normal water turnover, the labelled water containing ^2H and $^{18}O_2$ is lost. However, $^{18}O_2$ in water exchanges with the oxygen in CO_2 because of the carbonic anhydrase reaction. This means that CO_2 excretion will also result in an additional loss of $^{18}O_2$ as $C^{18}O_2$. This means that $^{18}O_2$ leaves the body faster than ^2H, the difference being proportional to CO_2 production. Loss of the two isotopes from body water is assessed by measurement of the rate of decline in concentration of the isotope in a sample of the subject's urine or saliva, collected during the study period, and measured by isotope ratio mass spectrometry. The difference between the elimination rates of the two isotopes reflects the rate at which CO_2 is produced from metabolism. TEE can then be estimated from the CO_2 production rate after assigning an energy value to CO_2 calculated from the assumed average respiratory quotient (RQ) value (ratio of CO_2 produced to the O_2 consumed), which is determined by the balance of macronutrients oxidised during the period. This in turn is assumed to reflect the composition of the dietary intake.

316. The accuracy and precision of the DLW method for measuring TEE is influenced by isotope fractionation during evaporative water loss and CO_2 excretion. Corrections for isotopic fractionation of water lost in breath and (non-sweat) transcutaneous loss need to be made when using labelled water to measure water turnover or CO_2 production (Schoeller *et al*., 1986). The technique, therefore, is based on assumptions about the amount of water lost from the body by evaporation and the extent of incorporation of ^2H and $^{18}O_2$ into body tissues, especially during growth. This technique, however, provides an indirect measure of TEE and is the most accurate available measure in free-living subjects. The estimated TEE is the energy expended during a time period, including the energy required for tissue synthesis, but does not include the energy content of tissue laid down (growth,

pregnancy, weight gain) or milk produced during lactation; these are estimated from analysis of tissue deposition and milk secretion.

Methodology critique

317. Although the DLW method has become established as the method of choice for the estimation of free-living TEE, uncertainties exist about its application and, consequently, in the interpretation of published DLW studies. As this SACN report relies almost entirely on information on TEE determined by the DLW method, some of the important issues in relation to the reliability of DLW studies are briefly discussed.

318. The application of the DLW technique has not been standardised, with different approaches taken in both laboratory isotopic analysis and in the experimental design of the measurement of $^{18}O_2$ and $^{2}H_2$ turnover in body water. The three most widely used approaches are the 'slope-intercept', '2-point', and 'modified' methods of calculation. This lack of standardisation means its potential high precision (the within-individual coefficient of variation (CV) for the validation of TEE from DLW against respiratory gas exchange) is not always realised. A double-blind between-laboratory variability study identified substantial between-laboratory variability in results, in some cases with precision as low as 35% and with some reporting physiologically impossible results (Roberts *et al*., 1995a). This seems to reflect analytical error rather than methods of calculation or the approach used to assess isotope decay rates. The different approaches are in principle equally valid although some reviewers suggest that collecting samples repeatedly over the measurement period rather than by collecting them only before and after the measurement period may decrease the error to a small degree. Of the data sets assembled for this report, the adult values from the Beltsville study (Moshfegh *et al*., 2008) involved the multipoint slope-intercept approach while the OPEN study (Trabulsi *et al*., 2003) involved the 2-point approach. However, the data set assembled for children and adolescents involves reports from many different laboratories so that between-investigator errors could represent cause for concern in our identification of energy reference values for these population groups.

319. Estimates of measurement precision vary between investigators. Not all investigators document error terms for the dose, background determinations, or uncertainties in fractionated evaporative water loss and RQ. A change in background $^{18}O_2$ enrichment in water during the study resulting from travel or a change in the dietary water source could affect the final CO_2 production rate, especially in subjects where very low isotopic dosing is used. Observed variation in repeated measurement studies will reflect both methodological error as well as actual variation in TEE due to a change in behaviour, making interpretation difficult. In one study, careful duplicate measurement of TEE in six adult women at a six month interval indicated that the within-subject CV for TEE was 7.8%, most of which was estimated by the investigators to be physiological variation (6.4%) due to variation in activity (Schoeller & Hnilicka, 1996). They also reviewed 16 studies with at least two DLW measurements, which indicated the reliability of the method to be 7.8%, except under conditions of high water flux.

320. Within the OPEN study, repeated isotopic analysis and repeated DLW measurement in a subset of 25 subjects identified an overall CV of the TEE measurement of 5.1%, of which 2.9% was due to analytical variation and 4.2% was due to within-individual physiological variation (Trabulsi *et al*., 2003). Others have identified a value for analytical variation of 4% (Elia *et al*., 2000). Within the Beltsville study, repeat DLW measurements were conducted in a subset of 32 subjects and an overall CV of 12.6% was identified. As the repeat measures were more than one year after the initial measurements (unlike the OPEN subset study which compared repeat measures after two weeks) it might be expected that variation would be larger. Some of the individuals in the Beltsville subset study (Moshfegh *et al*., 2008) exhibited TEE in the second study of only 50% of the first. This highlights the difficulty of identifying rates of TEE within free-living populations of relatively small sample sizes.

321. The application of the DLW approach usually involves 7-14 day measurements; however it is unknown if this is representative of TEE in the longer term. The Beltsville subset study points to potential errors in this assumption e.g. the R^2 of the second measure compared with the first was only 0.35. Individuals are likely to change their physical activity with time, due to differences in season for example, resulting in potentially considerable within-person variability. Concerns have been expressed about this issue (Willett, 2003) and about the design of the OPEN Study due to the within-person CV of TEE (5.1%). This value appears too low when compared with values obtained from a quantitative review of the reproducibility of TEE measured by DLW in 25 studies with repeated measurements (Black & Cole, 2000). In this latter review, estimates of 8% for within-subject variation in DLW measurements were reported. This estimate included analytical errors plus inherent within-subject biological variation in TEE due to changes in weight, season and physical activity. This biological variation increased as might be expected with increased time between measurements to about 15% at a time span of 12 months. The authors of the OPEN study (Kipnis *et al*., 2003) subsequently reanalysed the data from studies examined in the review and found that for studies of only free-living subjects (as in the OPEN study) within-person variation did not increase with time. The Beltsville study, however, shows a large increase in the CV when replicate studies are conducted over a longer time period.

322. Another potential concern is the influence of growth on measurements due to sequestration of isotope within body tissues during the study. Deuterium can be incorporated into tissues during reductive biosynthesis especially of fat and cholesterol. Such sequestration would decrease the difference in $^{2}H_2$ and $^{18}O_2$ decay rates and lead to an under-estimation of CO_2 production and TEE. The extent to which this is a problem is a difficult question to resolve. Technical problems in assessing the relative influences of isotope sequestration and isotope fractionated water loss, which have opposite influences on the estimation of TEE, make the effect on TEE measures in rapidly growing infants difficult to assess. A consensus review (NAHRES-4, 1990) concluded that under extreme anabolic conditions and using pessimistic assumptions regarding *de novo* fat synthesis, the maximum error in estimation of TEE due to ^{2}H sequestration could be 5% but that "it seems unlikely that the error would be as high as this under many circumstances".

323. The extent to which increasing body fat can influence the method is also an issue. One study in obese and lean subjects identified an underestimation of TEE by DLW of 0.285 MJ/day for each additional 10 kg of fat (Ravussin *et al*., 1991). Others, however, have failed to observe such an effect (Gibney *et al*., 2003).

324. Another potential problem is recruitment bias. Subject selection in any study requires subjects to either volunteer or to agree to participate when randomly approached and this raises the possibility of the healthy volunteer effect with study subjects atypical of the general population, in this case more physically active with higher than average rates of TEE. In a validation study for NDNS (Ruston *et al*., 2004), DLW studies were conducted in a small adult population (n=66) indicating mean PAL values of 1.74 (range 1.36-2.2) for women and 1.88 (range 1.37-2.50) for men. These values are on average higher than those of the two large population studies (i.e. OPEN and Beltsville) used in this report to identify adult population PAL values. This may be because the NDNS DLW subsample exhibited the healthy volunteer effect, or that the men in the NDNS DLW sub-sample were heavier than those in the OPEN-Beltsville data set and in the NDNS main study, or it may simply reflect the smaller cohort size. For the OPEN study, volunteers were derived from a random sample of 5000 households so the healthy volunteer effect is likely to be less than in the Beltsville study in which subjects were recruited through advertisements and letters of invitation. The fact that the distribution of TEE was so similar in these two studies would tend to suggest that recruitment bias was not a significant factor.

Summary

325. It is likely that recent DLW studies have benefited from the experience gained with the method over 25 years of its use. The data set of DLW studies used to derive the EAR for children and adolescents in this SACN report, however, includes a wide range of studies assembled over many years and by many investigators. Although technical problems can be minimised with careful investigators, some caution must be used in examining this data set.

326. More important, however, is the difficulty posed by true, within-individual variation in physical activity and consequent TEE. To illustrate this, the repeat DLW assessments of TEE conducted more than one year after the first measurement in the Beltsville study are given below (see Table 26) (Moshfegh *et al*., 2008). The within-subject average CV for TEE of 12.6% was just over half the between-subject average value of 22.8% for the larger whole cohort. Some of this latter figure reflects between-subject variation in TEE with size due to variation in basal metabolic rate (BMR). After adjusting for BMR and calculating PAL there is a lower CV of 15.4%. During the within-subject repeat measurements of TEE, the subjects were generally weight stable and the BMR would not be expected to have changed. This means that most of the variance must reflect change in physical activity. The within-subjects CV and the CV of the between-subject PAL values are similar: 12.6 % compared with 15.4%. This indicates the difficulty of identifying energy requirements with any certainty for individuals and small groups of subjects, which are likely to reflect average values for extended periods of time.

Table 26 Beltsville[a] Doubly Labelled Water (DLW) data set: within and between subject variability

	within subject CV[a] %		between subject CV[b] %			
	TEE	n	TEE	n	PAL	n
Normal	12.1	19	20.2	210	16.2	203
Overweight	12.4	14	22.4	186	14.4	179
Obese	15.5	9	20.8	101	15.4	96
All subjects	12.6	42	22.8	497	15.4	478

[a] As reported by Moshfegh *et al* (2008)
[b] Calculated directly from the data set

Appendix 7 – Physical activity and energy balance

Background

327. Physical activity energy expenditure (PAEE) is the most variable component of total energy expenditure (TEE) and is amenable to modification. Decreases in PAEE may affect an individual's ability to maintain energy balance and consequently maintain a stable body weight. The rising prevalence of obesity has been attributed in part to population-level changes in PAEE (WHO, 1998; Royal College of Physicians, 2004; Foresight, 2007).

328. The aim of this Appendix is to consider the energy expended during physical activity in relation to risk of weight gain and obesity, and to determine whether a specific level of physical activity can be defined that is protective against a positive energy imbalance, and hence weight gain. Data on the current physical activity levels of the UK population are also considered.

Measuring physical activity

329. Studies investigating physical activity have employed either subjective measures based on self-report and questionnaires, objective measures based on measures of physiological response (e.g. heart rate) or bodily movement (e.g. accelerometers), or TEE measured by the doubly labelled water (DLW) method over longer periods.

Subjective measures of physical activity

330. Many types of physical activity questionnaires have been used in surveys and epidemiological studies. Information obtained is often converted into a summary measure that is then used to categorise or rank the physical activity level of subjects. Questionnaires can detail physical activities performed during a specified period, but their accuracy is limited, especially in assessing non-exercise physical activity (Sesso, 2007).

331. Children are less likely than adults to make an accurate self-reported physical activity assessment (Welk *et al*., 2000) and in children of younger age groups it is virtually impossible to obtain valid self-reported physical activity data (Rennie *et al*., 2006).

332. Even when physical activity questionnaires are logically constructed with attention to the different domains of activity, they are still relatively imprecise as a measure of PAEE (Wareham *et al*., 2002). In adults, subjective measures of physical activity have proved sufficient to demonstrate associations with many disease outcomes, to monitor compliance with physical activity guidelines, and have allowed the estimation of dose-response effects. Objective methods are more accurate, however, and allow more precise estimations of dose-response

effects. It has been suggested that many physical activity questionnaires have an arbitrary grading in their classification of relative activities, which can result in an overestimation (Erlichman *et al*., 2002).

Objective measures of physical activity

333. Techniques such as heart rate monitoring (HRM) and accelerometry provide minute-by-minute data and give information on the total levels of physical activity, as well as its intensity, duration and frequency. Accelerometry measures body movement, usually in one (vertical) or three (vertical, lateral and anterior-posterior) planes, but is limited in its ability to measure activities such as swimming and cycling. By applying movement count cut-off points, minute-by-minute data from accelerometers can be summated into time spent in low- moderate- and vigorous-intensity activity. Accelerometers overcome some of the problems of measuring children's physical activity through subjective measures. There is uncertainty, however, in defining cut-offs for different intensity levels, which causes problems when comparing studies (Freedson *et al*., 2005; Guinhouya *et al*., 2006). The HRM method is limited in its ability to differentiate between modest increases in heart rate (HR) above resting levels and increases in HR associated with stress or other causes. The combining of HRM with movement sensors addresses these issues and improves accuracy (Rennie *et al*., 2006). PAEE can be estimated in groups using HRM and accelerometry (Corder *et al*., 2007), but the DLW method provides a more accurate assessment.

334. The DLW method measures TEE over several days and in conjunction with measures, or estimates, of basal metabolic rate (BMR) or resting metabolic rate (RMR) can be used to measure PAEE indirectly. The DLW method is considered the most suitable method for measuring TEE under free-living conditions. It does not, however, give day-to-day information nor does it give information on the forms, frequency and intensity of physical activity undertaken (Rennie *et al*., 2006). The DLW method is discussed further in Appendix 6.

Assessment of physical activity levels in the UK population

335. Data on physical activity levels of the UK population are available from the national Health Surveys for England, Scotland, Wales and Northern Ireland (Aresu *et al*., 2009; Corbett *et al*., 2010; Statistics for Wales/Ystadegau ar gyfer Cymru, 2010; Northern Ireland Statistics and Research Agency, 2006; NHS IC, 2010), the National Diet and Nutrition Survey (NDNS) of adults aged 19-64 years (Ruston *et al*., 2004), the Low Income Diet and Nutrition Survey (LIDNS) (Nelson *et al*., 2007) which included adults and children, and the unpublished NDNS comparison study. The Health Surveys use a seven-day recall method to assess physical activity. The adults NDNS used a seven-day diary method and DLW data was also collected in a small subset of survey participants (see paragraph 113), while LIDNS used a four day recall method.

336. Both the NDNS and the Health Surveys provided estimates of physical activity as the proportion of the survey population who reported achieving the physical activity recommendations for England published in 2004 (DH, 2004)[34]. The surveys consistently show that the majority of people do not meet these physical activity recommendations. These estimates are based on self-reported physical activity questionnaires which are likely to over-report physical activity in the population and have limited accuracy in representing habitual levels.

337. In children, additional studies assessing physical activity levels via accelerometry and the proportions meeting physical activity recommendations have been published (Basterfield *et al*., 2008; Mattocks *et al*., 2007; Riddoch *et al*., 2007; van Sluijs *et al*., 2008; Trayers *et al*., 2006). However, the results are mixed as they are dependent on the threshold used to define moderate-to-vigorous activity.

UK population PAL values

338. It is not possible to derive PAL values from the Health Surveys of England, Scotland, Wales and Northern Ireland because they do not record the amount of reported time spent on all types of activities. In NDNS, PAL values for adults were derived in a validation study conducted prior to the main adult survey (Ruston *et al*., 2004). Physical activity was assessed in a sample of 66 adults using both seven day physical activity questionnaires and DLW assessment of TEE. The mean PAL values from the questionnaires were 1.76 and 1.66 for men and women respectively, while the PAL values derived from the DLW data were 1.88 and 1.74. However, correlation between the two sets of values was weak, with the activity values explaining only 14% of the variation in the DLW data, indicating that estimates of PAL derived from self-reported physical activity questionnaires resulted in considerable misclassification of activity levels (see paragraph 323). Also, the PAL values are higher than you would expect for the general population, suggesting that this sub-sample is not representative.

Physical activity and body fatness

339. Physical activity has long been considered an integral component in the treatment of those who are obese and in the prevention of weight regain in those who have lost weight (Astrup, 2006; Miller *et al*., 1997; Shaw *et al*., 2006). Physical activity alone appears a relatively inefficient means for losing weight, but appears to be an important factor in the successful maintenance of weight loss and in improving insulin sensitivity and cardiovascular health (Astrup, 2006; Atlantis *et al*., 2006; Bensimhon *et al*., 2006; CMOs, 2011; Wing & Hill, 2001).

340. This section focuses on the role of physical activity in the primary prevention of weight gain and obesity.

34 In the UK, adults are recommended to have at least 150 minutes of moderate intensity physical activity a week. Previous recommendations were based on a pattern of activity encompassing 30 minutes of moderate activity on five or more days of the week (DH, 2004); however, the new recommendations (Chief Medical Officers of England, Scotland, Wales and Northern Ireland, 2011) recognise that the 150 minutes can be achieved in a variety of ways. For children and young people, a total of at least 60 minutes each day of at least moderate to vigorous intensity physical activity is recommended.

Prospective studies of self-reported physical activity and weight gain

341. Prospective studies relating to physical activity and weight change in both adults and children have been systematically reviewed (Fogelholm & Kukkonen-Harjula 2000; Molnar & Livingstone 2000 and Wareham *et al*., 2005).

Adults

342. The Fogelholm and Kukkonen-Harjula systematic review (2000) included 16 prospective studies investigating the relationship between self-reported physical activity and weight change in adults. It concluded that there was inconsistent evidence of a predictive effect of higher levels of physical activity at baseline being associated with less weight gain over time. The association between weight gain and change in activity was observed to be stronger, although still modest.

343. A follow-up systematic review by Wareham *et al* (2005) included 12 prospective studies investigating the relationship between self-reported physical activity and weight change. Nine studies reported a negative association between baseline physical activity and subsequent weight gain (Bell *et al*., 2001; Drøyvold *et al*., 2004; Hu *et al*., 2003; Koh-Banerjee *et al*., 2003; Macdonald *et al*., 2003; Schmitz *et al*., 2000; Sherwood *et al*., 2000; Wagner *et al*., 2001; Wenche *et al*., 2004) and two found no association (Ball *et al*., 2002; Rainwater *et al*., 2000). One study reported an inverse association suggesting higher baseline levels of BMI predicted physical inactivity (Petersen *et al*., 2004). The majority of studies suggested that low levels of physical activity were associated with future weight gain, but the effect size was small. The more recent studies included in this review (Wareham *et al*., 2005) had at least 500 participants, whereas the previous review (Fogelholm & Kukkonen-Harjula, 2000) included five studies with less than 500 participants and, therefore, less power to detect small differences. Improvements in study design could be a factor, as could publication bias, in determining why the follow-up systematic review reported more consistent results.

344. Wareham *et al* (2005) concluded that in longitudinal cohort studies, individuals who reported higher levels of leisure-time physical activity tended to be less likely to gain weight, but studies varied in their conclusions due to issues of confounding, measurement error and reverse causality, i.e. obesity may lead to physical inactivity (Petersen *et al*., 2004).

345. Studies published after these systematic reviews have observed leisure-time physical activity to be inversely associated with BMI (Wilsgaard *et al*., 2005), waist circumference (Waller *et al*., 2008) and weight gain, particularly in those with a larger baseline weight (Gordon-Larsen *et al*., 2009), suggesting a favourable effect of physical activity on weight maintenance.

Children and adolescents

346. The Molnar and Livingstone systematic review (2000) identified two prospective studies that investigated the influence of self-reported physical activity on the change in relative BMI. One study found increases in children's leisure activity at

follow-up to be associated with decreases in subsequent weight gain (Klesges *et al*., 1995), while the other found no association (Maffeis *et al*., 1998).

347. The Wareham *et al* systematic review (2005) identified a further 11 studies. All studies except one (Tammelin *et al*., 2004), used reported change in BMI or sum of skinfolds as the outcome. Five of the studies did not observe an association between physical activity or sedentary behaviour and weight gain (Bogaert *et al*., 2003; Davison & Birch, 2001; Francis *et al*., 2003; Kimm *et al*., 2001; Mamalakis *et al*., 2000). The other six studies found an inverse association between higher levels of physical activity and weight gain or a positive association between weight gain and sedentary activities (Berkey *et al*., 2000; Berkey *et al*., 2003; Hancox *et al*., 2004; Horn *et al*., 2001; O'Loughlin *et al*., 2000; Proctor *et al*., 2003; Tammelin *et al*., 2004).

348. Overall, the results were mixed and it was concluded that, as in the adult studies, the measures of association tended to be small (Wareham *et al*., 2005). Another review of prospective studies (Must and Tybor, 2005) also concluded that the results were mixed and that the associations identified were generally of a small magnitude.

349. Of the studies that have reported subsequent to the Wareham *et al* review (2005), two studies (Kimm *et al*., 2005; Mundt *et al*., 2006) have observed physical activity to attenuate increases in fat mass development in boys, but not in girls. One observed no difference in BMI changes or the percentage of students classified as obese between schools with higher and lower frequency of physical education (Wardle *et al*., 2007). Another study observed reported physical activity and inactivity to be related to accrual of body fat, particularly among children with at least one overweight parent (Must *et al*., 2007).

350. Most obese children remain obese as adults (Magarey *et al*., 2003), a progression that is referred to as 'tracking' of overweight. Several studies have examined whether adolescent physical activity affects subsequent weight gain through to adulthood. Some prospective studies do provide evidence that a decline in reported physical activity between adolescence and adulthood may increase risk of weight gain and obesity, but these associations are generally weak and inconsistent (Boreham *et al*., 2004; Kvaavik *et al*., 2003; Parsons *et al*., 2006; Pietilainen *et al*., 2008; Tammelin *et al*., 2004; Twisk *et al*., 2000; Yang *et al*., 2006; Yang *et al*., 2007).

351. On balance, the available evidence from prospective cohort studies suggests that increased physical activity and decreased sedentary behaviour may be protective against relative weight and fatness gains; however, the results are mixed and the associations that are identified are generally of a small magnitude. It is likely that imprecise measurement of activity exposures weakens the observed relationships (Must & Tybor, 2005). Measurement error is probably an important factor as most studies rely on subjective measures of reported physical activity and assess fatness using BMI, which is limited in its ability to determine fat and lean tissue mass across the normal range in adults (Wells *et al*., 2007a) and in children (Wells *et al*., 2002).

Prospective studies of objectively measured physical activity and weight gain

Adults

352. The Wareham *et al* systematic review (2005) identified two studies investigating objectively measured physical activity in relation to weight gain. This has been updated by the Wilks *et al* systematic review (2011) of studies including objectively measured physical activity, which identified 16 prospective studies, six in adults and ten in children.

353. In adults, the length of follow up varied between 1.5 and 5.6 years and three of the six studies were carried out in the USA. Three adult studies reported no association between baseline PAEE and subsequent change in body weight. The other three observed favourable associations with increased PAEE inversely correlated with weight outcomes; however, due to methodological issues two of the latter studies were not truly prospective (Bailey *et al*., 2007; Weinsier *et al*., 2002).

Children and adolescents

354. The Molnar and Livingstone systematic review (2000) identified five prospective studies that investigated the association between objectively measured physical activity or PAEE and change in indices of body fatness in children and adolescents.

355. One study observed children with low levels of physical activity (assessed using accelerometry) to gain substantially more subcutaneous fat than more active children (Moore *et al*., 1995). One small study (n=18) observed reduced TEE, and particularly PAEE, to be associated with weight gain (Roberts *et al*., 1988), while other, larger studies have found no association (Davies *et al*., 1991a; Goran *et al*., 1998b).

356. The Wareham *et al* (2005) systematic review identified five subsequent studies that had investigated the relationship between physical activity and body weight. The children included in these studies were mostly younger than 10 years and the duration of follow-up ranged from 2 to 8 years.

357. One study found increased physical activity, assessed using accelerometry, to be associated with smaller gains in BMI and subcutaneous fat (Moore *et al*., 2003). Overall, however, the results from the other studies using DLW methods to assess energy expenditure were inconsistent (Figueroa-Colon *et al*., 2000; Johnson *et al*., 2000; Treuth *et al*., 2003; Wells & Ritz, 2001).

358. The Wilks *et al*., review (2011) identified 10 studies in children. All studies were carried out in the USA and the majority of children were aged between 2-12 years at baseline. The duration of follow-up ranged between 1 and 8 years. Six studies found no association between physical activity and adiposity, three found a negative association, and one found a positive association.

359. Overall, the results from prospective studies using objective measures of physical activity in children, adolescents and adults were inconsistent and the associations identified were generally of small magnitude (Must & Tybor, 2005; Wareham *et al*., 2005; Wilks *et al*., 2011). For example, the degree of variance in BMI attributed to physical activity in several studies was less than one percent (Ekelund *et al*., 2004b; Styne, 2005).

360. The lack of consistent associations between DLW-derived measures of PAEE and measures of body fatness could be interpreted as evidence that energy intake is a more important determinant of excess fat mass gain. There are difficulties in the interpretation of these data, however, because of the controversy regarding the means of comparing TEE and PAEE among individuals of different sizes (Dietz, 1998). It has been suggested that when studies evaluate associations between PAEE or PAL and percentage body fat, the differences between energy expended in physical activity are likely to be overestimated between leaner and fatter children and the differences in body fatness to be underestimated, resulting in associations being biased towards null (Rennie *et al*., 2006). The use of DLW measures to identify how much PAEE is necessary to prevent obesity is complex; even if appropriate adjustment for body composition is made, comparisons between populations are difficult. It is also important to note that the energy expended in activity may not be the same as the amount of physical activity required to prevent excess FM gain; thus, assessment of physical activity by methods such as HRM and accelerometry is also required (Rennie *et al*., 2006).

361. The potential impact of exercise intensity on change in body weight and FM remains unclear (Grediagin *et al*., 1995; Lemura & Maziekas, 2002; Tremblay *et al*., 1994; Yoshioka *et al*., 2001) and it is not known which, if any, of the subcomponents of free-living physical activity contributes more to change in body weight and FM.

Trials using physical activity as an intervention to prevent weight gain

362. Interventions aimed at weight reduction or at preventing weight regain are not included in this consideration. A systematic review by Hardeman *et al* (2000) identified nine interventions (eleven publications) using physical activity as the primary prevention against weight gain. Interventions lasted from 6 weeks to 36 months. Four interventions took place in the community (Fitzgibbon *et al*., 1995; Forster *et al*., 1988; Jeffery & French, 1997; Sherwood *et al*., 1998; Stolley & Fitzgibbon, 1997) and five were school based (Caballero *et al*., 1998; Cairella *et al*., 1998; Davis *et al*., 1993; Donnelly *et al*., 1996; Gittelsohn *et al*., 1998; Simonetti D'Arca *et al*., 1986).

363. It was concluded that overall the results suggested mixed effects and, for various methodological reasons, they were uncertain in their conclusions about whether increasing physical activity was effective in preventing weight gain. Effectiveness appeared to be greater among older, male and high-income participants, and lower among low-income participants, school students and smokers. Where diet

and physical activity were described, positive effects were usually obtained, but the validity of this was limited as they were measured by self-report.

364. This systematic review (Hardeman *et al*., 2000) was subsequently updated with a further seventeen trials (Wareham *et al*., 2005). A total of six trials aimed at increasing physical activity and preventing weight gain in adults were identified. The interventions took place in populations at risk of weight gain or in whom a public health intervention might be targeted. Interventions lasted from 12 weeks to 5 years. In the four trials where differences in body composition between intervention and control group were observed, two found an increase in body weight in the control group and weight stability in the intervention group (Littrell *et al*., 2003; Simkin-Silverman *et al*., 2003), one found a weight reduction in the intervention group (Muto *et al*., 2001) and the other observed decreases in both groups (Proper *et al*., 2003). Two trials observed no effect on weight gain (Burke *et al*., 2003; Polley *et al*., 2002).

365. A total of eleven trials were identified in children aimed at preventing unhealthy weight gain by increasing physical activity or reducing sedentary behaviour (Wareham *et al*., 2005). Nine trials were school-based and the others home or family-based. Interventions lasted from 12 weeks to 3 years. Three of the trials reported a small intervention effect at follow-up (Kain *et al*., 2004; McMurray *et al*., 2002; Sallis *et al*., 2003), with two of them reporting effects in boys only (Kain *et al*., 2004; Sallis *et al*., 2003). The other eight trials reported no significant effects on body weight or composition at follow-up (Baranowski *et al*., 2003; Caballero *et al*., 2003; Dennison *et al*., 2004; Neumark-Sztainer *et al*., 2003; Pangrazi *et al*., 2003; Robinson *et al*., 2003; Sahota *et al*., 2001; Warren *et al*., 2003).

366. Wareham *et al* (2005) concluded that there were still relatively few trials aimed at the primary prevention of weight gain using physical activity as an intervention and that there remained insufficient evidence on which to base conclusions regarding which approaches were effective.

367. A subsequent review by Wilks *et al* (2011) identified five intervention trials using objective measures of physical activity, one in adults and four in children. The adult study investigated the effect of a home based exercise programme on adoposity, with compliance measured by accelerometry. However, no change in body weight was observed in either the control or intervention group (Cooper *et al*., 2000). Of the studies in children (aged between 4-12 years), two were community based, one was nursery based and one was school based. Only one physical activity intervention found a siginificant effect on adiposity (Verstraete *et al*., 2007).

368. Additionally, another school-based intervention (where physical activity was assessed by questionnaires) observed a slower gain in BMI, especially in non-overweight adolescents, with increasing physical activity (Simon *et al*., 2008).

Sedentary behaviour and weight gain

369. Sedentary behaviour is a different concept to physical activity with a different physiology and different determinants. Many behaviours are largely sedentary,

including those where sitting or lying are the predominant activity (e.g. listening to the radio or music, watching television, using computers and reading).

370. It has been suggested that the increased use of information and communication technology is a sedentary factor affecting obesity prevalence (Fox & Hillsdon, 2007; Kautiainen *et al*., 2005). The use of computers, both at home and at work, has been one of the most rapidly expanding activities in the past 20 years and could potentially impact on overall physical activity levels.

371. A meta-analysis has been conducted of prospective studies and trials investigating the relation between television viewing and video/computer game use and body fatness and physical activity in children and adolescents (Marshall *et al*., 2004). The only significant relationship observed was between television viewing and body fatness, but it was concluded that this was likely to be too small to be of substantial clinical relevance and that media-based inactivity may be unfairly implicated in recent epidemiologic trends of overweight and obesity among children and adolescents. It was also noted that relationships between sedentary behaviour and health were unlikely to be explained using single markers of inactivity, such as television viewing or video/computer game use. Physical activity and sedentary behaviours are regulated through a complex series of decision-making mechanisms and restricting television viewing alone may not be effective in increasing physical activity (Nelson *et al*., 2005).

372. Several prospective studies conducted since this meta-analysis have also observed positive associations between television viewing in children and subsequent weight gain (Davison *et al*., 2006; Hancox *et al*., 2004; Hancox & Poulton, 2006; Jago *et al*., 2005; Parsons *et al*., 2008; Reilly *et al*., 2005; Viner & Cole, 2005). It has been suggested that although the effect size appears small for time spent watching television as a predictor of weight gain in childhood, it is larger than the effect sizes commonly reported for dietary intake and physical activity; thus, television viewing could be an important contributing factor to childhood obesity (Hancox & Poulton, 2006).

373. The issue of measurement error in these studies and the need to select measures of television viewing that are valid and reliable to examine with greater accuracy the influence of television viewing on childhood overweight, has been highlighted (Bryant *et al*., 2007).

374. Most studies examining the prospective and longitudinal associations between sedentary behaviour and BMI have relied on self-reported data. One prospective population-based cohort study measured sedentary behaviour by individually calibrated HRM in 393 healthy adults (Ekelund *et al*., 2008). At 5.6 years follow-up, sedentary time did not predict any of the obesity indicators (body weight, BMI, fat mass and waist circumference); however, the obesity indicators predicted sedentary time at follow-up after adjustment.

375. A systematic review has been conducted of trials to reduce sedentary behaviour among children, either alone or in combination with other health messages (Demattia *et al*., 2007). The interventions ranged from four weeks to four years,

with six of the studies targeting clinic-based populations that were overweight or at risk of overweight. A further six were population-based prevention studies. The magnitude of change in weight parameters was modest and was difficult to interpret, as normal BMI ranges vary with age and development in children. The z-BMI score (BMI normalised to age and sex) was only reported in a few of the studies. Virtually all of the interventions, however, consistently resulted in slowing of the increase in the subjects' BMI relative to similar aged controls. As the sedentary behaviour messages in these interventions are often combined with other health information (e.g. healthy eating and exercise), it was not possible to estimate the magnitude of the weight influences due to sedentary behaviour messages alone.

Summary

376. The assessment of UK population physical activity levels, i.e. the Health Surveys and the NDNS and LIDNS, is currently dependent upon subjective measures of physical activity. It is likely that these give an overestimation of physical activity in the population. To enable the accurate determination of habitual physical activity and PAEE in the UK population, surveys need to employ objective measures.

377. Methodological constraints are a severe limitation in defining the role of physical activity in the regulation of body weight. Most studies rely on subjective measures of reported physical activity and assess fatness using BMI, which is of limited value in determining fat and lean tissue mass across the normal range in adults (Wells *et al*., 2007a) and children (Wells *et al*., 2002). Error is introduced on both sides of the relationship thereby reducing the ability to detect any change. The application of more precise methods for the measurement of physical activity and body fatness is required to define their interrelationship.

378. On balance, the available evidence from prospective cohort studies suggests that increased leisure time physical activity may be protective against relative weight and fatness gains; however, the findings are mixed and the associations that are identified are generally of a small magnitude. Prospective studies also suggest lengthy television watching may be a predictor for weight gain, but again the associations are weak and inconsistent. Evidence from studies employing objective measures of PAEE and trials of the primary prevention of weight gain through increased physical activity is also inconsistent.

379. The issue of whether there is a specific level of physical activity required to prevent unhealthy weight gain is complex, and available data is insufficient to reach a definitive conclusion (Blair *et al*., 2004; Wareham *et al*., 2005).

Appendix 8 – Characteristics of the data set of DLW measures of energy expenditure for adults: combined OPEN/Beltsville DLW data set.

380. The two data sets of total energy expenditure (TEE) measures used were the OPEN study (Subar *et al*., 2003; Tooze *et al*., 2007) (individual data obtained from Amy Subar, National Cancer Institute USA) and the Beltsville underreporting study (Moshfegh *et al*., 2008) (individual data obtained from Alanna Moshfegh, US Department of Agriculture (USDA), Agricultural Research Service).

381. The two USA populations studied were similar in terms of anthropometry, ethnicity and social class. However, there is no indication in either study of the range of lifestyles within the cohorts in terms of physical activity levels. Because of this and in the absence of any other cross-sectional population study of TEE, it is not possible to determine how representative the distribution of TEE and physical activity level (PAL) values is of the US or, most importantly, the UK population. However, on the basis of the doubly labelled water (DLW) data examined in the production of this report, it is likely that the median PAL value identified here, 1.63, represents a light activity population. As identified in Appendix 5, this PAL value is lower than either 1.72, the median value of the data base assembled for the US DRI report on energy requirements (IoM, 2005), or 1.75 the mean value of published studies tabulated in Table 22 (Appendix 5). It is also lower than the median value for the NDNS data set i.e. 1.75 for 156 subjects aged 19-75years, with a mean body mass index (BMI) of 27.2kg/m^2. In Table 22, study populations categorised as exhibiting light activity exhibited a mean PAL of 1.67; FAO/WHO/UNU (FAO, 2004) identified a PAL range associated with sedentary or light activity lifestyle to be 1.40-1.69.

382. The OPEN study (n=451) involved healthy volunteers aged 40–69 years recruited from a random sample of 5000 households in the metropolitan area of Washington, DC (Montgomery County, MD). The cohort comprised 245 men and 206 women, of which 85% were white with the rest mainly black or Asian. Most (87%) had some college schooling with 63% college graduates or post graduates. The distribution by BMI groups was 31% normal (18.5 to less than 25 kg/m^2), 41% overweight (25 to less than 30 kg/m^2), and 29% obese (30 kg/m^2 or more).

383. BMR was not measured in the OPEN study, but BMR was estimated using the Mifflin predictions based on weight, height and age (Tooze *et al*., 2007). For reasons discussed in Appendix 4, the data presented in this SACN report are calculated using the Henry prediction equations based on weight and height (Henry, 2005). The validity of these Mifflin BMR prediction equations is indicated by the fact that the regression of BMR on BMI within the OPEN cohort is almost identical to that for the Beltsville study (in which BMR was measured) for men and quite similar for

women. Consequently, BMR values estimated by BMI within these two cohorts differed by less than 0.5% in men and by less than 2% in women. Also, as shown in Table 27 below, the distribution of PAL values within the OPEN cohort is the same.

384. The Beltsville study (n=525) involved a study cohort of volunteers aged 30–69 years residing in the greater Washington, DC metropolitan area. These volunteers were recruited through advertisements in local newspapers and on websites; announcements sent to employees of USDA (Beltsville, MD), local industries, and offices; and the use of a Beltsville Human Nutrition Research Center database of persons known to be interested in participating in human studies.

385. The subjects were predominately non-Hispanic white and were distributed evenly by sex and approximately by age. Only 8% of subjects had not attended college. Approximately 21% of the subjects (both sexes) were obese (BMI greater than 30 kg/m^2). More females (48%) than males (36%) were considered normal weight. Only 5% of the men and 6% of the women were current smokers. BMR was measured.

386. The characteristics of the distribution of the PAL values for the two data sets are shown in Table 27. They were very similar. The combined OPEN and Beltsville data set was trimmed for PAL values less than 1.27 or greater than 2.5, on the grounds that these are the limits of sustainable PAL values within a healthy population and that values outside this range are unphysiological. This removed one subject with a PAL value greater than 2.5 and 38 subjects with PAL values less than 1.27 (mean 1.17, range 1.01- 1.269). The effect of trimming was to increase the median PAL value from 1.62 to 1.63.

Table 27 Physical activity level (PAL) value statistics for individual and combined Beltsville[a] and OPEN[b] data sets

PAL	n	Mean	SD	Min	10th centile	25th centile	Median centile	75th centile	90th centile	Max
Beltsville[a]	478	1.63	0.25	1.01	1.32	1.46	1.62	1.78	1.96	2.34
OPEN[b]	451	1.64	0.21	1.01	1.40	1.49	1.61	1.77	1.92	2.61
All	929	1.64	0.23	1.01	1.36	1.48	1.62	1.78	1.95	2.61
Trimmed	890	1.66	0.21	1.27	1.40	1.49	1.63	1.78	1.96	2.50

[a] Beltsville data set (Moshfegh et al, 2008)
[b] OPEN data set (Subar et al., 2003; Tooze et al., 2007)

387. The distribution of PAL by gender and age, and in terms of frequency, is shown in Figure 6. The distribution of subjects by BMI and age is shown in Figure 7. The regression of TEE on BMR is shown in Figure 8 indicating the very small intercept (33.5 kcal/kg (95% CI:-104 & 171; p=0.6), which is discussed further in Appendix 5.

Figure 6 – Distribution of physical activity level (PAL) by gender and age and frequency in the OPEN and Beltsville data sets

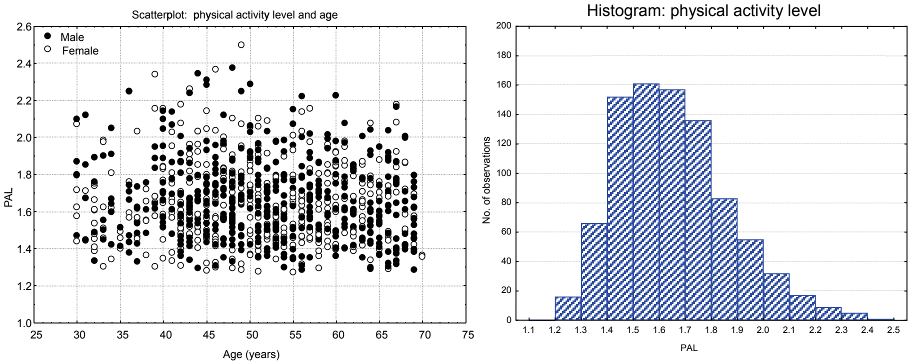


Figure 7 – Distribution of subjects by body mass index (BMI) (kg/m^2) and age of OPEN and Beltsville participants.

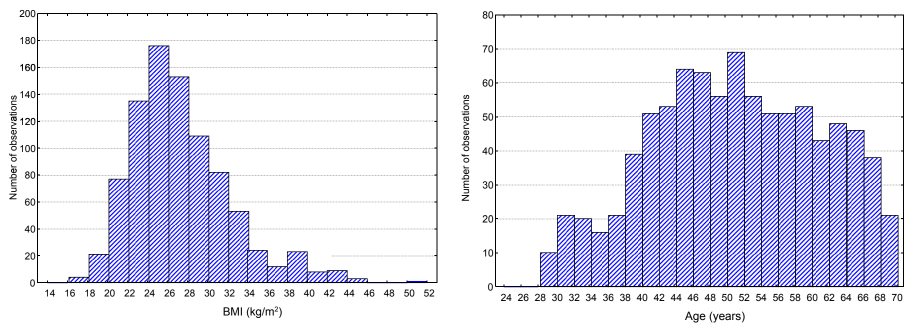
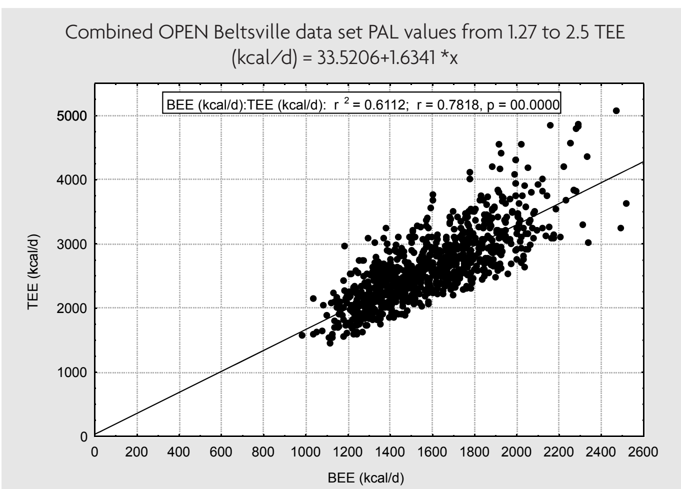


Figure 8 – Regression of total energy expenditure TEE on basal energy expenditure (BEE): combined OPEN and Beltsville data set[a]

[a] PAL values from 1.27 to 2.5. TEE (kcal/d) = 33.5206 + 1.6341*X



388. The population demographics for the combined data sets are shown in Table 28, and the distribution of BMI and its relationship to PAL is shown in Table 29 and Table 30.

Table 28 Population demographics for combined Beltsville[a] and OPEN[b] data sets

		n	Mean	SD	Min	Max
Age	F	424	51	10	30	70
years	M	466	52	10	30	69
	All Groups	890	51	10	30	70
Height[c]	F	206	1.63	0.06	1.47	1.82
meters	M	239	1.77	0.07	1.58	1.94
	All Groups	445	1.70	0.10	1.47	1.94
Weight[c]	F	206	73	17	45	126
kg	M	239	88	16	54	138
	All Groups	445	81	18	45	138
BMI	F	424	27	6	17	51
kg/m^2	M	466	28	4	18	43
	All Groups	890	27	5	17	51
BMR	F	424	1393	166	982	1960
kcal/d	M	466	1757	217	1279	2522
	All Groups	890	1584	266	982	2522
TEE	F	424	2290	386	1442	3731
kcal/d	M	466	2923	516	1889	5061
	All Groups	890	2621	557	1442	5061

[a] Beltsville data set (Moshfegh et al, 2008)
[b] OPEN data set (Subar et al., 2003; Tooze et al., 2007)
[c] Beltsville data set contains only BMI, not height or weight
F=female, M=male

Table 29 Distribution of body mass index (BMI) and its relationship to physcial activity level (PAL) for the combined Beltsville[a] and OPEN[b] data sets

			BMI kg/m^2				PAL		
		N	mean	sd	mean	median	sd	Min	Max
Normal	All	322	22.7	1.69	1.65	0.21	1.61	1.27	2.37
	Female	181	22.4	1.76	1.64	0.22	1.60	1.27	2.37
	Male	141	23.1	1.51	1.67	0.21	1.64	1.29	2.34
Overweight	All	348	27.2	1.45	1.66	0.20	1.65	1.29	2.50
	Female	136	27.1	1.41	1.66	0.21	1.63	1.29	2.50
	Male	212	27.3	1.47	1.66	0.20	1.66	1.29	2.31
Obese	All	220	34.1	3.94	1.66	0.23	1.63	1.28	2.37
	Female	107	34.7	4.37	1.65	0.21	1.63	1.28	2.15
	Male	113	33.5	3.41	1.67	0.24	1.63	1.29	2.37
All		890	27.3	4.97	1.66	0.21	1.63	1.27	2.50

[a] Beltsville data set (Moshfegh et al, 2008)
[b] OPEN data set (Subar et al., 2003; Tooze et al., 2007)

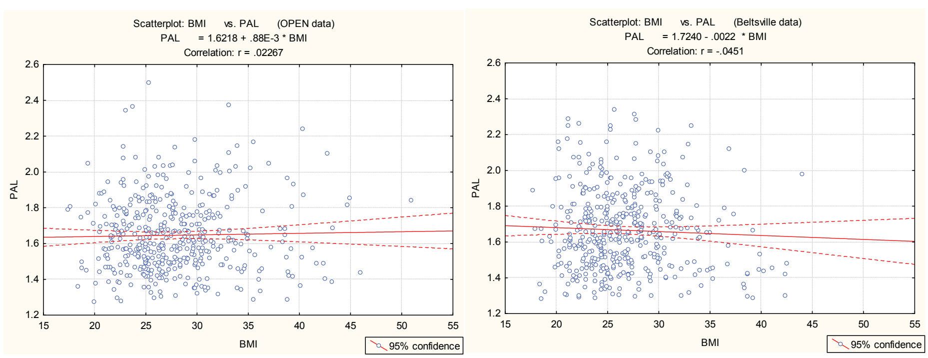
Table 30 Distribution of physcial activity level (PAL) according to body mass index (BMI) categories for the combined Beltsville[a] and OPEN[b] data sets

Status	N	Mean	Std.Dev.	Min	10th	Q25	Median	Q75	90th	Max
Normal[c]	322	1.65	0.21	1.27	1.40	1.49	1.61	1.78	1.95	2.37
Overweight[c]	348	1.66	0.20	1.29	1.41	1.50	1.65	1.79	1.92	2.50
Obese[c]	220	1.66	0.23	1.28	1.40	1.47	1.63	1.80	1.99	2.37
All	890	1.66	0.21	1.27	1.40	1.49	1.63	1.78	1.96	2.50

[a] Beltsville data set (Moshfegh et al, 2008)
[b] OPEN data set (Subar et al., 2003; Tooze et al., 2007)
[c] Using the following BMI definitions: Underweight: less than 18.5 kg/m^2; normal: 18.5 to less than 25kg/m^2; overweight: 25 to less than 30 kg/m^2; obese 30 kg/m^2 or more; overweight including obese 25 kg/m^2 or more; morbidly obese: 40 kg/m^2 or more

389. Within the cohort, there were similar numbers of overweight and obese subjects (36% normal, 39% overweight and 25% obese). Mean PAL values for BMI categories did not differ significantly (p=0.91) and neither did the distribution in terms of quartile boundaries and 10th and 90th centile values. The regression of PAL on BMI was also non-significant (p=0.64 for slope: R^2 less than 0.1%). Finally, as shown below in Figure 9, the regressions of PAL on BMI for the two cohorts were very similar even though PAL was calculated from a measured BMR for the Beltsville data set compared with an estimated BMR for the OPEN data. Thus, estimating BMR for an overweight/obese population did not appear to introduce bias. This lack of influence of BMI on PAL was also observed in the NDNS sample.

Figure 9 – Relationship of physical activity level (PAL) values with body mass index (BMI) for the combined Beltsville[a] and OPEN[b] data sets



[a] Beltsville data set (Moshfegh et al, 2008)
[b] OPEN data set (Subar et al., 2003; Tooze et al., 2007)

390. PAL fell with age (PAL=1.74-0.0016 x age, R^2=0.004, p=<0.03); however, the shallow slope means that the fall with age is small and age explains only 0.4% of the variance, i.e. PAL = 1.69 at 30 and 1.63 at 70. Also, as shown in Table 31, a comparison of PAL values between the younger subjects (aged ≤35 years) with those aged >35 years shows the absolute values of mean and median PAL values to be slightly lower in the younger subjects, although not significantly different (p=0.51 for mean values). This suggests that the lack of adult subjects aged <30 years is unlikely to represent a significant source of bias when the data set is used to represent PAL

values for all adults. This is supported by the NDNS DLW data set (see paragraph 113) for which there was no significant change in PAL with age, between the ages of 19 and 75 years (R^2 = 0.015, Regression Coefficient for age on PAL: — 0.0018). This would be a fall in PAL of around 0.1 units.

Table 31 Comparison of physical activity level (PAL) values between the youngest group (aged≤35 years) with those >35 years for the combined Beltsville[a] and OPEN[b] data sets

Age	N	Mean	Std. Dev.	Min	10th centile	Q25	Median	Q75	90th centile	Max
≤35	56	1.64	0.21	1.30	1.41	1.46	1.61	1.78	1.97	2.12
>35	834	1.66	0.21	1.27	1.40	1.49	1.63	1.78	1.96	2.50

[a] Beltsville data set (Moshfegh et al, 2008)
[b] OPEN data set (Subar et al., 2003; Tooze et al., 2007)

Appendix 9 – Energy requirements for disease

Introduction

391. There is a continuum in physical function between health and disease, and the dividing line between them can be difficult to define. The proportion of older individuals in the UK population is increasing (Walker *et al*., 2001) and the incidence of many diseases and disabilities also increases with age (Elia *et al*., 2000). In the 2001 UK General Household Survey, one in ten respondents under the age of 45 years reported a longstanding illness that limited physical activity, compared with a third of older respondents (Walker *et al*., 2001). Less severe disabilities may produce effects on physical activity that are difficult to distinguish from normal activity. The high prevalence of disease in older people makes it difficult to define 'normal', especially in extreme old age. A consideration of physical activity level (PAL) values in older adults is given in Appendix 5.

392. Determining the energy requirements of patients with acute and chronic diseases is more complex than for those in good health. Energy requirements in illness are greatly influenced by the type (acute or chronic), severity, and phase of the disease (acute or recovery phase). They are also affected by the presence of other physical and psychological disabilities, which may vary over time, the treatment of the disease, and the nutritional state of the patient (e.g. the presence of prior malnutrition). In some circumstances, it may not be appropriate to treat the disease or the associated malnutrition, as when a patient is approaching death and feeding is a burden.

393. The energy requirements of a number of severe acute diseases were thought to be increased (Elia, 1995), as were the requirements of individuals with spastic disorders, such as spastic cerebral palsy (Eddy *et al*., 1965). It is now understood that this is not usually the case.

Energy intake

394. Individuals with disease may be in substantial energy imbalance for days, weeks, and sometimes months. This imbalance is often caused by anorexia which is a common consequence of traumatic, infective, malignant, or inflammatory diseases. Clearly, accurate measurements of energy intake in disease do not necessarily reflect the energy required to maintain energy balance or the energy required to achieve optimal health. Furthermore, although in under-nourished subjects, additional energy is required for repletion and improvement in tissue function, health and well-being, the reverse applies to obese individuals. If absorption of nutrients is reduced due to illness or if urinary losses occur (e.g. in diabetes), then the nutritional value of ingested foods will be less than in an unaffected individual.

395. The timing of nutritional support is also important. Aggressive overfeeding in the acute phase of injury, for example, can cause metabolic disturbances, such as hyperglycaemia (diabetes of injury) and increased CO_2 production, which can be detrimental to patients with respiratory failure. In contrast, very slow repletion during recovery can prolong the period of ill health, and will also have a negative impact on work performance and quality of life. Finally, nutritional intake in disease may differ from that in health because it can be provided artificially using an enteral tube or venous catheter (sometimes for life), independent of appetite.

Energy expenditure

396. Basal metabolic rate (BMR) is more variable in disease than in health. Apart from being influenced by the type, severity and phase of the illness, BMR is also influenced by the nutritional status of the patient and a wide range of treatments, which may vary from surgical interventions, immobilisation and artificial ventilation, to blood transfusions and drug therapy. Standard reference tables or equations for estimating BMR (from weight, height and age) were established for use in healthy subjects without malnutrition and disease, and without dehydration and oedema, all of which can have substantial effects on body weight. The estimation of BMR, therefore, is more likely to be in error in disease than in health.

397. Whether measurements of total energy expenditure (TEE) (or measurements of BMR) are regarded as normal or abnormal relative to a healthy group may depend on the way in which the results are expressed. This can be problematic for patients with diseases associated with abnormal body composition and body proportions who preferentially lose or preserve particular tissues or organs (e.g. muscle wasting in certain neurological conditions). Different investigators have used over 20 indices to express energy expenditure, including absolute energy expenditure, energy expenditure per kilogram body weight, energy expenditure per kilogram fat free mass (FFM), and energy expenditure per m^2 (Elia, 1997). In children, energy expenditure has also been expressed as a percentage of the BMR values obtained in healthy children of the same age, the same surface area, or the same height. In infants with growth failure, there may be a decrease in both the whole body BMR and the tissue specific BMR, while BMR per kilogram body weight may be increased due to preferential preservation of the brain, which has a high metabolic rate (Elia, 1997). Similarly, TEE may be low when expressed in relation to values obtained in children of the same age, and normal when it is expressed in relation to healthy children of the same weight. The hypermetabolic effect of disease and the hypometabolic effect of weight loss alter BMR to a greater extent than in health, even when age, weight and height are taken into account. This affects the accuracy of expressing TEE as a ratio to BMR (i.e. PAL) (Elia, 1992). Consequently, PAL values based on measured BMR and estimated BMR (based on values established for healthy subjects) can vary widely, especially in acute disease which typically increases BMR.

398. As with energy intake, TEE does not necessarily indicate the requirements of the under-nourished patient who is in need of repletion, or the requirements of the over-nourished patient, who is in need of depletion of excess fat.

399. Measurements of TEE have helped establish important concepts about energy requirements in disease. Acute and chronic diseases are associated with simultaneous changes in BMR, physical activity and TEE. These factors, and the methodological limitations associated with their estimation, represent important practical and theoretical issues that should be considered when determining energy requirements in pathological states.

Diseases in adults

400. For many diseases, both TEE and energy requirements are decreased mainly because of a reduction in physical activity energy expenditure (PAEE). This reduction in PAEE can occur because disease produces lethargy and restricts physical activity (e.g. pain due to claudication). It also occurs because treatment, especially in hospital, usually requires restricted physical activity. Loss of weight will contribute to the reduction in TEE, partly because it reduces the energy expended in physical activity and partly because it reduces BMR.

401. TEE is normal or decreased in chronic diseases (Elia, 2005). In those with advanced disease or with disease-related weight loss, both PAEE and TEE are usually decreased, despite a possible increase in BMR. The weight loss that may occur in such diseases is more likely to be due to a reduction in energy intake than an increase in energy expenditure. A possible exception to this general rule concerns subgroups of patients with anorexia nervosa, who have been reported to have increased PAEE and TEE, when adjusted for weight (Casper *et al*., 1991). However, some studies of women with anorexia nervosa show no increase in TEE, after adjustment for weight (Marken Lichtenbelt *et al*., 1997). When no adjustment for weight is made, TEE is likely to be lower than in healthy women of the same age.

402. Acute diseases increase resting energy expenditure (REE) above that predicted for healthy individuals of the same age, weight and height by up to 100% (Bessey & Wilmore, 1988), although usually by 0-40%. Both the magnitude and duration of the increase in energy expenditure are dependent on the severity of disease. The effect of 'injury' on BMR is also influenced by age. For example, the increase in BMR may last a few days following elective surgery in adults and only a few hours in infants (Elia, 2000). Acute or sub-acute diseases usually cause a decrease in lean body mass. After the early phase of an illness, therefore, BMR may decline below pre-illness BMR before beginning to return to normal in the recovery phase.

403. In studies where the TEE of bed-bound artificially ventilated patients has been measured (e.g. those with head injuries or other critical illness), BMR is frequently increased. TEE is usually not elevated however, primarily because of the concomitant reduction in physical activity (Elia, 2005). The overall result is that TEE is normal or even decreased compared to values obtained in healthy subjects in free-living circumstances, with the exception of the most severe acute diseases, such as burns, when TEE may be transiently elevated above normal (Bessey & Wilmore, 1988).

404. In summary, in most of the chronic conditions examined BMR adjusted for weight is usually normal or slightly increased (about 10% in HIV/AIDS for example), while in most acute conditions it is usually increased (0-40% and occasionally more). This increase in BMR is counteracted by the decrease in PAEE, with the overall result that TEE is usually normal or decreased. More severe acute disease produces greater increments in BMR and greater reductions in PAEE.

Diseases in children

405. Assessment of the energy requirements of children is more difficult than in adults because of the need to consider growth and the most appropriate way to express energy expenditure.

406. In children, most chronic disease conditions do not produce an increase in TEE adjusted for body weight, despite a possible increase in BMR (e.g. cystic fibrosis (Magoffin *et al*., 2008). There are some exceptions, such as subgroups of patients with cystic fibrosis (cystic fibrosis genotype) and some children with congenital heart disease (Kuip *et al*., 2003). The general situation is analogous to that observed in adults, as is that for acute disease conditions, where TEE is not increased (actually decreased) because of the reduction in PAEE.

Summary

407. The lack of accurate information on TEE in a large number of diseases, does not allow a comprehensive assessment of the field. In those conditions which have been investigated, TEE is usually normal or reduced, partly because of a reduction in body weight and FFM (due to disease-related malnutrition or neurological causes of wasting), and partly because of reduced physical activity. The reduced physical activity compensates for any increase in BMR, which is common in acute diseases. There are some exceptions, such as subgroups of patients with cystic fibrosis, anorexia nervosa, and congenital heart disease, where TEE has been reported to be increased. Such patients are often underweight, and therefore weight adjustments are necessary to demonstrate differences compared to control groups. Without such adjustments, TEE is again typically not increased.

408. In determining energy requirements in disease there is a need to consider not only the energy required to maintain energy balance, but also the energy required to change body composition at different rates in both under-nourished and over-nourished patients.

Appendix 10 – Obesity prevalence in the UK

409. Body Mass Index (BMI) (kg/m^2) is often used as a convenient measure of adiposity, with recognised limitations. It has been used since the 1960s to assess obesity in adults (Keys *et al.*, 1972) and more recently in children (Cole *et al.*, 2005; Dietz & Robinson, 1998). While BMI is a good measure of weight independent of height, it fails to distinguish between adipose and non-adipose body mass. When used as a proxy for fat mass (FM) or adiposity, assumptions are made about absolute and relative body composition, which may not be valid. BMI fails to reflect body shape, and hence fat distribution, and is not strongly related to central adiposity, which is considered to be most harmful to health (Wells *et al.*, 2007a).

410. In adults, cut-off points for underweight, overweight and obesity are defined as BMI values of $\leq$18.5 kg/m^2, $\geq$25 kg/m^2 and $\geq$30 kg/m^2, respectively (NICE, 2006). Insufficient energy intakes are uncommon in healthy free-living individuals in the UK and do not generally arise from insufficient food supplies, but from accompanying physical or psychological conditions. In the 2009 Health Survey for England (HSE) (NHS IC, 2010), 2.3% of adults were classed as underweight. The Low Income Diet and Nutrition Survey (Nelson *et al.*, 2007) also found the prevalence of underweight to be low in adults living in low income households (2% of both men and women). In contrast, there is a high prevalence of overweight and obesity in the UK population resulting from a chronic excess of dietary energy intake over energy expenditure.

411. In children, BMI measures require cautious interpretation when comparing across groups that differ in age or when predicting a specific individual's total or percent body fat (Pietrobelli *et al.*, 1998). Children of the same age and gender have been shown to have a two-fold range of FM for a given BMI value, which is also observed in those who are obese (Wells *et al.*, 2007b). BMI normally changes during growth and this requires careful interpretation through the use of appropriate growth references or standards for age and sex. In the UK, the UK 1990 growth references (Cole, 1995) were used until 2009 for all children up to 18 years of age. From May 2009, for children aged between 0-4 years, revised standards have been adopted based on the WHO Child Growth Standards (SACN/RCPCH, 2007; RCPCH, 2011). For the purpose of population monitoring, children with a BMI over the 85th centile for age of the reference population are categorised as overweight and those with a BMI over the 95th centile for age as obese (NHS IC, 2010). In UK clinical practice, however, children over the 91st centile for the reference population are categorised as overweight and those over the 98th as obese (Hall & Elliman, 2003). Further thresholds have been proposed for the purpose of international comparison (Cole *et al.*, 2000); these allow estimation of the proportion of children at each age expected to exceed a BMI of 25 or 30 at the age of 18 years. The use of different

definitions that are not directly comparable can lead to difficulty in interpreting different surveys or studies of childhood obesity.

412. Temporal changes in the estimated prevalence of overweight and obesity in England are tabulated below for children aged 2-15 years based on the UK 1990 BMI growth reference (Cole, 1995) (Table 32), and for adults (NHS IC, 2010) (Table 33).

413. In 2009, 31% boys and 28% girls in England were overweight or obese (NHS IC, 2010). Obesity (as defined by a BMI over the 95th centile for age) increased from 11% and 12% in 1995 to 16% and 15% in 2009, in boy and girls respectively. Despite the overall increase since 1995, the proportion of girls aged 2 to 15 years who were obese decreased from 18% to 15% between 2005 and 2006 and remained 15% in 2009. There was no significant decrease among boys over the same period, with 17% classed as obese in 2006 and 2008.

414. For adults in England, the prevalence of obesity has increased from 15% in 1993 to 23% in 2009 (NHS IC, 2010), with 66% men and 57% women overweight and obese. Fewer people are in the normal weight range and the proportion of morbidly obese individuals has more than doubled. Importantly for women of childbearing age, for whom overweight and obesity results in increased clinical risk (McDonald *et al.*, 2010; Stothard *et al.*, 2009), the prevalence of overweight and obesity is substantial and increases with age. Thus, for the age groups 16 – 24 years, 25 – 34 years and 35 – 44 years, mean BMI values were 24.3, 25.8 and 27.2 and the prevalence of overweight and obesity was 37%, 48% and 62%, respectively. In a comparison of the HSE between 1993 and 2003, both BMI and central adiposity (waist circumference) increased more in the upper part of the distribution, with intermediate increases in the middle and little change at the lower end of the distribution (Wardle & Boniface, 2008). The observed temporal gains in central adiposity were not equivalent across the BMI distribution. Thinner people were almost as thin as they were 10 years earlier, but fatter people were considerably fatter.

415. The 2009 Scottish Health Survey (Corbett et al., 2010) shows there has also been a steady upward trend in the prevalence of overweight and obesity among both sexes since 1995 for adults and children. Most adults aged 16 years or over are either overweight or obese i.e. 66% of men and 58% of women. Overall obesity prevalence was 27% for men and 28% for women, and of these 1.0% and 3.5% were morbidly obese respectively, a prevalence which has stabilised since 2003. For children aged 2 to 15 years, boys were more likely than girls to be overweight or obese (29% versus 27%).

416. The Health Surveys for Scotland and England involve objective measures of weight and height, but the respective Health Surveys for Wales and Northern Ireland use self-reported measures and are therefore less accurate.

417. In the 2009 Welsh Health Survey (Statistics for Wales/Ystadegau ar gyfer Cymru, 2010), 62% of men were classified as overweight or obese compared with 52% of women. In men, 41% were overweight and 21% obese, while in women 31% were

overweight and 21% obese. In children, 34% were estimated to be overweight or obese, including 19% obese.

418. The 2005/6 Health and Social Wellbeing Survey in Northern Ireland (Northern Ireland Statistics and Research Agency, 2006) observed 64% men and 59% women to be either overweight or obese. In men, 39% were overweight and 25% obese, while in women 35% were overweight and 24% obese. In boys, 18% were overweight and 20% obese and in girls, 16% were overweight and 15% obese.

Table 32 Overweight and obesity prevalence among children, by year, 1995 to 2009, in the Health Survey for England[a]

All Children (aged 2-15 years)	Percentages														
	Unweighted[b]								Weighted[b]						
	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009
Overweight[c]	13.3	13.4	13.2	14.3	14.1	12.7	15.5	14	15	15.4	14.8	14	14.3	14.3	14.2
Obese[c]	11.7	12.2	12.8	13.7	15.5	14.5	15.2	17.4	16.9	18.9	18.6	16.3	16.8	16	15.7
Overweight including obese[c]	25	25.6	25.9	28	29.7	27.2	30.7	31.4	31.9	34.3	33.4	30.3	31.1	30.3	29.8

[a] NHS IC, 2010

[b] All years were weighted to adjust for the probability of selection, and from 2003 non-response weighting was also applied.

[c] Categories are independent, i.e. overweight does not include those who are obese. Overweight was defined as ≥ 85th < 95th UK BMI percentile; obese was defined as ≥ 95th UK BMI percentile

Table 33 Body Mass Index (BMI)[a] among adults[b], 1993 to 2009 in the Health Survey for England[a]

All adults[b]	Percentages																
	Unweighted[c]										Weighted[c]						
	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009
Underweight[d]	1.6	1.7	1.8	1.7	1.5	1.7	1.7	1.5	1.4	1.7	1.8	1.6	1.6	1.6	1.6	1.8	2.3
Normal[d]	45.5	45.2	43.7	42.2	41.6	40.6	40.4	38.6	37.0	37.7	37.8	36.7	37.9	36.8	37.7	36.8	36.4
Overweight[d]	38.0	37.4	38.1	38.7	38.5	38.3	38.0	38.8	39.2	38.1	37.9	38.8	37.3	37.6	36.7	36.9	38.3
Obese[d]	14.9	15.7	16.4	17.5	18.4	19.4	20.0	21.2	22.4	22.5	22.6	22.9	23.2	23.9	24.0	24.5	23.0
Overweight including obese[d]	*52.9*	*53.1*	*54.5*	*56.2*	*56.9*	*57.7*	*57.9*	*60.0*	*61.6*	*60.6*	*60.5*	*61.8*	*60.5*	*61.6*	*60.8*	*61.4*	*61.3*
Morbidly obese[d]	0.8	1.0	0.9	0.9	1.6	1.3	1.4	1.5	1.7	1.8	1.9	1.7	1.8	2.1	1.8	2.0	2.4

[a] NHS IC, 2010

[b] Adults aged 16 and over with a valid height and weight measurement

[c] Data from 2003 onwards have been weighted for non-response.

[d] Using the following BMI definitions: Underweight: less than 18.5 kg/m^2; normal: 18.5 to less than 25kg/m^2; overweight: 25 to less than 30 kg/m^2; obese 30 kg/m^2 or more; overweight including obese 25 kg/m^2 or more; morbidly obese: 40 kg/m^2 or more

Appendix 11 – A consideration of energy intake and physical activity in relation to weight gain

Background

419. Weight gain is dependent on the relationship between energy intake and energy expenditure, and it is therefore necessary to consider energy balance and energy flux, rather than energy intake and energy expenditure in isolation. This is the focus of this appendix.

420. It is not currently possible to accurately define the frequency, intensity and duration of physical activities that reduce the risk of weight gain. Available evidence suggests the influence of physical activity on body weight is weak, although methodological constraints are clearly an issue in interpreting the data (see Appendix 7). Furthermore, in experimental studies it is difficult to control for changes in energy intake which may occur during periods of altered physical activity.

421. The evidence investigating whether diet composition affects risk of weight gain is also weak (Jebb, 2007), but clearly a mismatch between energy intakes and energy expenditure is fundamental to weight gain. Methodological constraints have hampered the elucidation of the role of physical activity and diet composition in the development of overweight and obesity. Weight gain and obesity must result from a chronic positive energy imbalance, which implies a failure of auto-regulatory homeostatic responses to maintain energy balance. The asymmetry between the hunger and satiety arms of human appetite control has been implicated in this process (Prentice & Jebb, 2004).

422. The average daily weight gain in modern populations is relatively small and even in morbidly obese people the lifetime error in daily energy balance regulation is surprisingly small (Prentice & Jebb, 2004). Adult weight gain indicated in national surveys in the US is up to 8g/d (90th centile) (Hill *et al*., 2003; Hill, 2009) and up to twice this rate for excess weight gain in children (Butte & Ellis, 2003). Estimates of the energy cost of excess weight gain (mean growth costs in overweight less mean growth costs in normal weight children) are about 550kJ/d (130kcal/d) but this is equivalent to only 3-4% of estimated energy intakes (Butte & Ellis, 2003).

Energy balance and energy flux

423. It is a widely discussed hypothesis that the mechanisms controlling energy balance may be more accurate at higher levels of physical activity, with some suggesting a threshold of physical activity or energy flux below which these mechanisms become imprecise and dysregulated leading to a positive energy imbalance (Blair

et al., 2004) and obesity (Mayer & Thomas, 1967). Some evidence that the coupling between energy expenditure and energy intake may be less efficient at low levels of physical activity (Prentice & Jebb, 2004; Schoeller, 1998) is reviewed here.

424. Metabolic studies lasting between one and two weeks have covertly manipulated the energy density of foods supplied to volunteers fed *ad libitum* (Prentice, 1998; Prentice & Jebb, 2004; Stubbs et al., 1995a; Stubbs *et al*., 1995b) to investigate the impact on energy balance in subjects with different activity levels. For those fed a 40% fat energy diet, less physically active lean subjects (confined to whole-body calorimeters) had a positive energy imbalance of +850 kJ per day compared with a negative energy imbalance of -1,800kJ per day observed in those under free-living conditions.

425. Another two day whole-body calorimeter study showed that for lean subjects fed *ad libitum* the imposition of sedentary behaviour resulted in a positive energy imbalance especially on a high fat (covertly manipulated) diet whereas planned exercise enabled energy balance stability (Murgatroyd *et al*., 1999).

426. A further seven day whole-body calorimeter study showed that lean subjects fed *ad libitum* consumed the same energy intake when sedentary (1.4 x resting metabolic rate – RMR) compared with moderately active (1.8 x RMR) exhibiting a positive energy imbalance of 15.2 MJ over the seven days of the sedentary regimen (Stubbs *et al*., 2004).

427. Some investigators have proposed a threshold of physical activity below which appetite control is ineffective (Mayer, 1966). Based on a doubly labelled water (DLW) study of previously obese women (Schoeller, 1998), a physical activity level (PAL) value of 1.75 has been suggested as the threshold, although the evidence supporting this is not strong (Prentice & Jebb, 2004).

428. While these studies do provide some support for increased physical activity enabling better energy balance, the evidence is only short term (one to two weeks), and the concept of a threshold of energy flux above which body weight regulation is more sensitive has not been demonstrated convincingly.

429. Energy flux can be increased through weight gain, which increases basal metabolic rate (BMR) independently from any change in physical activity energy expenditure (PAEE). Indeed, the increasing cost of exercise with weight gain means that energy flux could increase in the obese even when the range and extent of actual activities falls. This is suggested by the lack of any obvious relationship between PAL and BMI (see Appendix 8). It is not clear whether the apparent improved energy balance regulation with increased physical activity, described above for lean adults, occurs in the obese (Hill, 2006). It may be, however, that the increasing energy costs of PAEE with weight gain represent a barrier to achieving sufficient increases in PAEE to improve energy balance regulation.

430. While physical activity alone seems to be a relatively inefficient means for losing weight in overweight individuals, it appears it can be a factor in the successful maintenance of weight loss (Astrup, 2006) (see Appendix 7). The efficacy of

increased physical activity in maintaining weight loss could be partly due to the increased energy flux; the psychological effects of exercise may also be important by enhancing well-being and status of control, and hence compliance with a restrictive dietary regimen (Prentice & Jebb, 2004).

431. Even when increasing PAEE and energy flux does not lead to a loss of body weight but a compensatory increase in energy intake, if energy balance is maintained this may also result in beneficial nutritional effects through the increased intake of other food constituents, such as micronutrients, which reduce the risk of nutritional deficiencies and poor nutritional status (Melzer *et al.*, 2005).

Exercise and appetite control

432. There appears to be a spontaneous reduction in hunger associated with participation in exercise programmes (Elder & Roberts, 2007). Short-term (1-2 day) and medium-term (7-16 day) physical activity intervention studies show substantial initial negative energy balances but energy intake subsequently increases to provide partial compensation for about 30% of the energy expended in activity (Blundell *et al.*, 2003), although the extent of compensation varies between individuals (King *et al.*, 2007). However, a study investigating the effect of an imposed sedentary routine on appetite, energy intake, energy balance and nutrient balance in lean men over seven days found no equivalent compensatory reduction in energy intake, leading to a significantly positive energy imbalance (Stubbs *et al.*, 2004).

433. It seems, therefore, that it might take considerable time for energy intake to fully adjust to changes in PAEE. However, there is also evidence that eventual increases in food intake do not follow the same pattern in obese as in lean individuals (Melzer *et al.*, 2005).

434. There is some evidence to suggest that exercise may modulate appetite control by improving the sensitivity of the physiological satiety signalling system (Blundell *et al.*, 2003; Martins *et al.*, 2008a; Martins *et al.*, 2008b). Compensation for a high-carbohydrate preload was observed to be more accurate in habitual exercisers than non-exercisers (Long *et al.*, 2002), as well as in those who had completed a six week moderate-intensity exercise intervention (Martins *et al.*, 2007). Following a bout of exercise, subjects were observed to discriminate more accurately between the energy content of different beverages (King *et al.*, 1999).

Summary

435. While it may not be possible to accurately define the nature of the relationship between physical activity, diet and weight gain, evidence reviewed here suggests that the matching of energy intake and expenditure may be improved at higher expenditure levels.

436. Most previous research has focused exclusively on the effects of one type of diet or physical activity, or has examined diet composition and physical inactivity independently, but few studies have compared the effects of a combination of diet composition and physical activity. Any effect of diet composition may be

dependent on the pattern of physical activity. Conversely, any effect of physical activity may depend, in part, on diet composition. A better understanding of these two factors and how they interact may help explain why obesity is so prevalent in the UK, while allowing for the fact that not everyone within that environment is obese.

Appendix 12 – Characteristics of the data set of doubly labelled water (DLW) measures of energy expenditure in children and adolescents

437. The available doubly labelled water (DLW) data from studies of children aged more than one year were examined. A data set of 845 individual values was assembled for the US DRI report (IoM, 2005), probably limited to studies published before 2002. The distribution by age was uneven and included large numbers of infants aged under two years, 4-5 and 8-10 year olds, with fewer 2-3 and 10-18 year olds.

438. The FAO/WHO/UNU report (FAO, 2004) was based on studies assembled by Torun (2005) which were listed in terms of mean study values for children aged over one year. This involved many more individual children with a more even age spread than that examined in the US DRI report (IoM, 2005).

439. For this SACN report, a data set of all published DLW studies of children aged over one year was compiled (the SACN children's dataset). This included all those studies assembled by Torun (2005) and other studies published up to 2006 (Anderson *et al.*, 2004; Arvidsson *et al.*, 2005; Bandini et al., 2002; Bratteby *et al.*, 2005; Craig *et al.*, 1996; DeLany *et al.*, 2006; Goran *et al.*, 1993b; Hernandez-Triana *et al.*, 2002; Hoos *et al.*, 2003; Lindquist *et al.*, 2000; Lopez-Alarcon *et al.*, 2004; McGloin *et al.*, 2002; Montgomery *et al.*, 2005; Perks *et al.*, 2000; Roemmich *et al.*, 2000; Rush *et al.*, 2003; Sjoberg *et al.*, 2003; Sun *et al.*, 1999; Wong *et al.*, 1999). All studies were tabulated according to study mean values for boys and girls for specific age groups (see below Table 37). This resulted in 170 data points (study means) representing a total of 3502 individual measurements (females=2082, males=1420). They included four studies from Central and South America (Brazil, Chile, Guatemala and Mexico) but all children were considered well-nourished. The remaining studies were mainly from the UK or the USA with single studies from Canada, Denmark, the Netherlands and Sweden. Those from the USA involved Caucasian-American, African-American and native-American children. UK studies (500 individual measurements) included only 15% of the total children studied and these were mainly infants and younger children. Only 6% of the total adolescents studied were from the UK.

440. Physical activity level (PAL) values and basal metabolic rate (BMR) values were not included in many of the studies. For all studies which did not report BMR, BMR values were estimated using the Henry equations (Henry, 2005) for weight and height or just weight if no height data were reported. PAL values were then derived from total energy expenditure (TEE) and BMR (see paragraph 79).

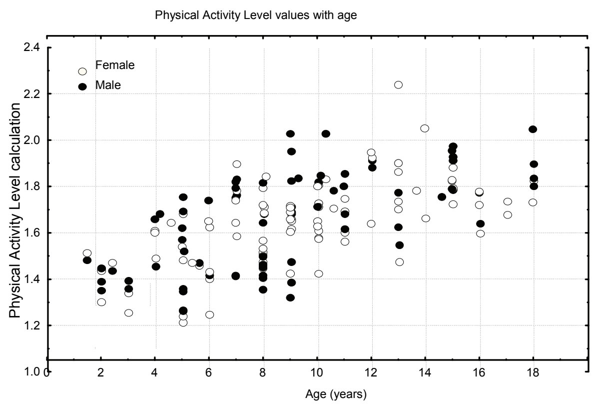
441. In addition, a data set of individual values obtained from the UK NDNS unpublished comparison study was examined. This included 65 subjects between the ages of

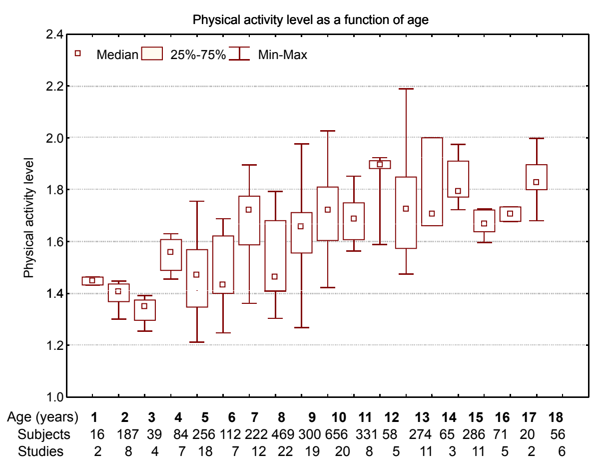
4 – 18 years. Whilst this sample size was too small to represent a reference data set, because it was a uniform sample in terms of the measurement methodology and is exclusively UK data it was able to provide useful comparative information (see Figure 12 and paragraph 444).

Number of studies and individuals within the studies and general characteristics

442. Mean PAL study values for the SACN children's data sat are shown in Figure 10. The tabulated data can be found in Table 37 at the end of this Appendix. The data set includes a reasonable overall age spread in terms of number of studies, although there are five or less studies for the age groups of 3, 11, 13, 15 and 16 years. Thus, there were relatively few subjects studied at the ages of 3, 4, 12, 14, 16-18 years and a relative excess of subjects (>600) at 10 years of age. Older adolescents (>15 years of age) are under-represented. Although most data points do relate to discreet age groups, in some cases data points are the mean of an age range. For example, three data points for eight year olds include PAL values from a total of 149 children with ages ranging from 5-10.5 years.

Figure 10 – Mean physical activity level (PAL) study values in the SACN children's data set as a function of age and gender





443. The scatterplot of TEE on BMR is shown in Figure 12. The increase in PAL after the age of four years means that the slope of the relationship of TEE with BMR increases after this time. This relationship is discussed in more detail in Appendix 5.

Figure 11 – Scatterplot of total energy expenditure (TEE) on basal metabolic rate (BMR) (SACN children's data set)

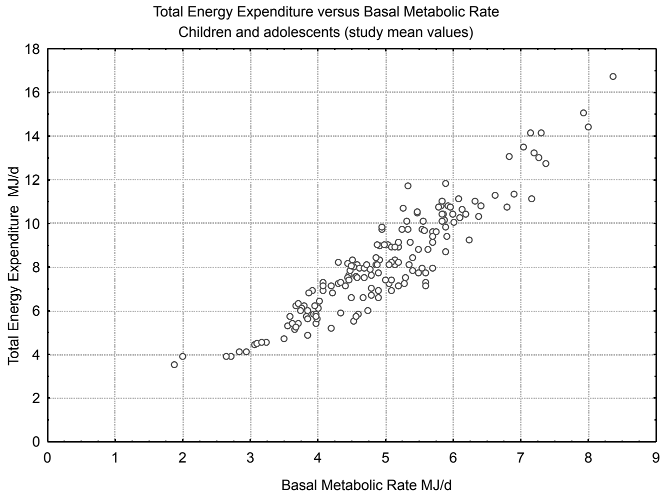
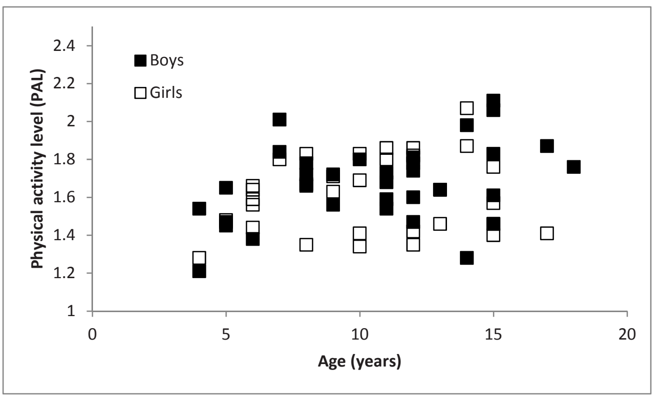


Figure 12 – Physical activity level (PAL) values in the NDNS children's data set as a fucntion of age and gender



Grouping by age

444. Overall, PAL appears to increase with age (see Figure 12) from 1.33 to 1.81 between the ages of 1 and 18 years. However, there was a clustering of the youngest children (aged 1-3 years) at the lower range (PAL ≈ 1.4), with an increase in the overall range starting at school age and with fewer studies with mean PAL values below 1.6 (n = 2) in adolescents. From an early age (≈5 years) there is a wide range of mean study PAL values for boys and girls around the regression and as discussed below (see paragraph 448), it is not entirely clear whether the apparent change with age for school-age children is real. One possibility was to exclude those studies with particularly low mean study values but it was decided that there was insufficient

evidence to do this. Therefore, all studies shown in Figure 10 were included in the analysis. The NDNS data set, of which individual values are shown in figure 12 and the median values for 4-9 and 10-18 year olds are shown in Figure 13, also displays an increase in PAL between these two age groups (although as shown in both Figures, the increase is slightly less than in the main data set). Grouping of all study means into three age groups was identified as a valid way of expressing the increase in PAL with age, thereby simplifying the calculation of energy reference values for children and adolescents.

445. Grouping of PAL values within the ages for which BMR is estimated (i.e. 1-3, 3-<10 and 10-18 years) is shown in Figure 13 and Table 34. Median PAL values for the three age groups are 1.39 (≤3 years), 1.57 (>3-<10 years) and 1.73 (>10-18 years). For the age groups ≤3 years and >10-18 years and for the ages of 5.5-8 years, the median values fall within or close to the 95th CI values of the regression. Clearly, the step changes in PAL and consequent energy reference values at the age group boundaries are a disadvantage to this approach which needs to be balanced against the simplifying effect of not using age specific PAL values predicted from the regression.

Figure 13 – Grouping of physical activity level (PAL) values by ages 0-3, >3-<10 and >10-18 years (SACN children's data set)

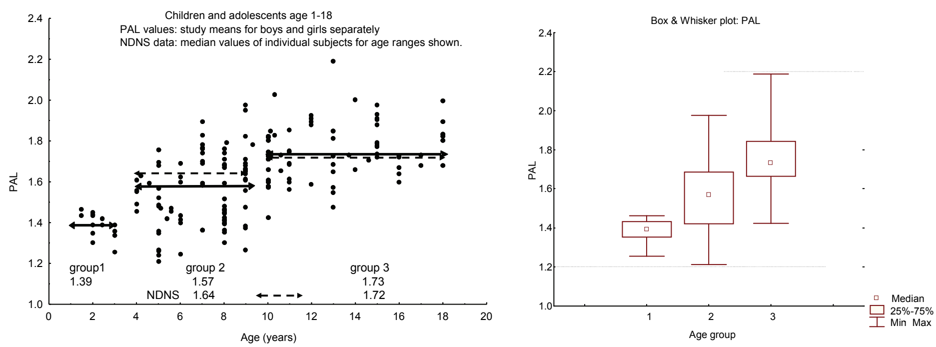


Table 34 Grouping of physical activity level (PAL) values within the ages ≤3, >3-<10 and 10-18 years (SACN children's data set)

		Age				PAL						
	N	Mean	sd	Min	Max	Mean	sd	Min	Max	Q25	Median	Q75
1	14	2.3	0.54	1.5	3.0	1.39	0.06	1.26	1.46	1.35	1.39	1.43
2	85	7.0	1.87	4.0	9.3	1.56	0.14	1.21	1.98	1.42	1.57	1.69
3	71	13.0	2.38	10.0	18.0	1.75	0.13	1.42	2.19	1.66	1.73	1.85

Influence of gender

446. Factorial ANOVA of PAL by age group and gender shows that gender is not a significant influence on PAL but there are significant differences between each of the three age groups. No gender differences were observed within the NDNS data set, shown in Figure 12.

Table 35 Influences of gender on physical activity level (PAL) values within the ages 1-3,>3-<10 and 10-18 years (SACN children's data set)

Gender			Age		PAL			
	Group	N	Means	Std.Dev.	Means	Std.Dev.	P values	
Girls	1	5	2.0	0.34	1.41	0.06	Gender	0.28
	2	23	5.4	1.23	1.50	0.18	Group	<0.0001
	3	63	11.2	2.94	1.67	0.15	Gender x group	0.55
Boys	1	5	2.0	0.34	1.40	0.04		
	2	25	5.4	1.24	1.54	0.18		
	3	49	11.5	3.19	1.71	0.20		

Variation of PAL with age within the BMR age groups

447. PAL varies with age within both the 3-<10 and 10-18 year age groups but the influence of age is minor compared with the overall variability (R^2=0.098 and 0.057) especially in the 10-18 year group (p=0.04). The slopes imply increases in PAL of 0.14 and 0.1 PAL units over the age ranges (3-<10 and 10-18 years respectively), a very small change compared with the wide within-group overall range as shown above (Table 35).

448. It is by no means certain whether the changes with age for school-age children are real or reflect selection bias or other between-study factors. Indeed, on the basis of the association of PAL values with activity levels as shown in Table 22 (Appendix 5), many of the mean PAL values for younger school children (PAL<1.5) would imply very low activity levels compared with other children at the same age. An analysis of the data set by DLW methodology (multipoint, two point or other) indicates that the PAL values for those involving the two point method, are almost invariably lower (≈ 0.2 PAL units) than the other methodologies and include all the very low activity groups. Because these studies represent a considerable fraction of studies on children aged from 5-9 years but relatively few involve adolescents, their inclusion in the data set does tend to increase the age-related changes in PAL. However, as discussed in Appendix 6 (paragraph 318), although some reviewers suggest that the multipoint approach may decrease the error to a small degree, overall the different approaches are in principle equally valid.

449. Examination of the few studies which report the range of PAL values in randomly selected pre-adolescent school children shows that such children do exhibit the same wide range of physical activity as the adult population. This is observed in the NDNS data set from the age of 7 (see Figure 12). In a study of 47 randomly selected Australian school children aged 5-10.5 years, PAL varied from 1.32-2.18 (mean =1.71: tertiles of 1.32-1.63, 1.64-1.80, 1.81-2.18) (Abbott and Davies, 2004). The same group reported mean PAL values of a group of 106 school children aged 6.0-9.6 years (Ball et al., 2001) which varied over a very wide range of 1.2-2.32, mean = 1.70. The combined data from these two studies (n=149) indicated a median PAL of 1.69 (range 1.19-2.34, 25th and 75th centiles of 1.56 and 1.83) with ages from 5-10.5 years. Although individual PAL values are not shown as a function of age, the authors

analysis of variation in PAL values (in relation to adiposity), implied that age was not an influence.

Adjustment of PAL to account for growth costs

450. Actual growth costs calculated by SACN differ only slightly from those reported by FAO/WHO/UNU (FAO, 2004). In the US DRI report (IoM, 2005) a simplified estimate is reported with single values for the 3-7 and 8-18 year age groups. Within the FAO/WHO/UNU report (FAO, 2004) growth costs are also reported as an adjustment to average PAL values involving an increase of 1% i.e. an assumption that growth costs (deposition) are equivalent to 1% of the energy requirement.

451. It is the case that if growth costs are calculated as a percentage of the energy requirement, the values indicated are an overall average (1-16 years) of 0.98% of the energy requirement (range, boys: 0.4% – 1.37%; girls: 0.05% – 1.59% girls). If a single average growth value as a percentage of the energy requirement was used throughout the age range, the maximum errors would be during the peak growth phase for older children where growth would be underestimated by up to 0.6% of the energy requirement for girls or 0.4% for boys and then overestimated by similar amounts as growth slows at the end of adolescence. Relatively, these are very small amounts (i.e. 0.01PAL units) given the variation in PAL and overall TEE which occurs at all ages.

452. Thus, growth costs can be included within a factorial model as an adjustment to PAL values of an increase of 1% (i.e. =1.01 x PAL).

Table 36 PAL values for use in calculation of energy requirements of children and adolescents, adjusted for growth

Age group	PAL[a]		
mean	Q25	Median	Q75
1-3	1.36	1.40	1.45
>3-<10	1.43	1.58	1.70
10-18	1.68	1.75	1.86

[a] PAL as indicated in Table 34 adjusted for growth (=PALx1.01)

Table 37 SACN data set of all published doubly-labelled water (DLW) studies of children aged over one year

Study	n	Sex	Age years	Weight kg	Height cm	BMI	TEE MJ/d	BMR MJ/d	PAL
Abbott *et al*., 2004	24	F	8	30.1	131.0	17.4	7.90	4.61	1.71
Abbott *et al*., 2004	23	M	9	30.0	133.0	16.7	8.30	4.92	1.69
Anderson *et al*., 2004	172	F	10	33.1	141.0	16.6	8.10	5.14	1.58
Arvidsson *et al*., 2005	16	F	16	56.4	164.0	21.0	9.10	5.70	1.60
Arvidsson *et al*., 2005	17	M	16	64.1	175.0	21.0	11.30	6.90	1.64
Ball *et al*., 2001	54	F	8	28.1	127.0	17.2	7.50	4.45	1.69
Ball *et al*., 2001	52	M	8	27.6	128.0	16.7	7.90	4.70	1.68

Table 37 (continued) SACN data set of all published doubly-labelled water (DLW) studies of children aged over one year

Study	n	Sex	Age years	Weight kg	Height cm	BMI	TEE MJ/d	BMR MJ/d	PAL
Bandini *et al*., 1990b	14	F	14	55.7	162.0	21.2	10.00	6.02	1.66
Bandini *et al*., 1990b	14	M	15	56.4	167.0	20.2	13.00	7.28	1.79
Bandini *et al*., 2002	123	F	10	30.1	137.0	16.0	7.70	4.90	1.57
Bandini *et al*., 2002	73	F	11	37.9	146.0	17.8	8.80	5.50	1.60
Bratteby *et al*., 1997	25	F	15	58.4	167.0	20.9	10.70	5.97	1.79
Bratteby *et al*., 1997	25	M	15	61.3	174.0	20.2	14.10	7.31	1.93
Bratteby *et al*., 2005	89	F	15	56.0	165.0	20.4	11.00	5.85	1.88
Bratteby *et al*., 2005	71	M	15	60.7	175.0	19.7	14.10	7.14	1.97
Butte *et al*., 2000a	43	F	2	10.9	82.0	16.3	3.50	1.88	1.86
Butte *et al*., 2000a	33	M	2	11.4	83.0	16.8	3.90	2.01	1.94
Butte *et al*., 2000a	43	F	2	12.0	88.0	16.6	4.10	2.85	1.44
Butte *et al*., 2000a	33	M	2	12.5	88.0	16.2	4.10	2.95	1.39
Champagne *et al*., 1998	31	F	10	39.3	144.0	19.0	9.70	5.34	1.82
Champagne *et al*., 1998	27	F	10	39.2	146.0	18.0	9.00	4.99	1.80
Champagne *et al*., 1998	29	M	10	46.4	147.0	21.0	10.60	6.14	1.73
Champagne *et al*., 1998	31	M	10	42.9	148.0	19.8	10.80	5.93	1.82
Champagne *et al*., 1998	15	F	10	28.9			8.28	4.52	1.83
Champagne *et al*., 1998	15	M	10	36.8			10.66	5.26	2.03
Craig *et al*., 1996	49	F	10	32.6	140.0	16.4	8.40	4.86	1.73
Davies *et al*., 1991b	16	F	5	18.5			6.18	3.68	1.68
Davies *et al*., 1991b	12	M	5	18.9			6.88	3.92	1.75
Davies *et al*., 1991b	15	F	7	26.0			8.17	4.31	1.90
Davies *et al*., 1991b	10	M	7	24.6			8.15	4.46	1.83
Davies *et al*., 1991b	15	F	9	29.1			7.57	4.57	1.66
Davies *et al*., 1991b	14	M	9	29.5			8.95	4.91	1.82
Davies *et al*., 1991b	10	F	12	49.3			10.53	5.47	1.92
Davies *et al*., 1991b	8	M	12	39.7			10.48	5.48	1.91
Davies *et al*., 1991b	11	F	15	58.0			10.12	5.88	1.72
Davies *et al*., 1991b	12	M	15	60.1			13.47	7.05	1.91
Davies *et al*., 1991b	11	F	18	62.4			11.09	6.08	1.82
Davies *et al*., 1991b	12	M	18	71.6			15.05	7.94	1.90
Davies & White 1995	11	M	2	12.7	88.0	16.3	4.50	3.11	1.45
Davies & White, 1995	12	F	2	13.0	89.0	16.4	4.40	3.07	1.43
Davies & White, 1995	16	F	3	14.9	96.0	16.2	4.70	3.51	1.34
Davies & White, 1995	15	M	3	15.0	96.0	16.3	5.10	3.67	1.39
Davies & White, 1995	16	M	4	16.9	104.0	15.6	5.40	3.71	1.46
Davies & White, 1995	11	F	4	17.1			5.30	3.56	1.49

Table 37 (continued) SACN data set of all published doubly-labelled water (DLW) studies of children aged over one year

Study	n	Sex	Age years	Weight kg	Height cm	BMI	TEE MJ/d	BMR MJ/d	PAL
DeLany *et al*., 2002	65	F	11	37.7	144.0	18.4	8.80	5.63	1.56
DeLany *et al*., 2002	66	M	11	43.9	146.0	20.1	10.30	6.38	1.61
DeLany *et al*., 2004	53	F	13	49.1	156.0	23.7	9.20	6.24	1.47
DeLany *et al*., 2004	61	M	13	55.3	159.0	25.6	11.10	7.17	1.55
DeLany *et al*., 2006	28	F	11	37.9	145.0	18.0	9.10	5.38	1.69
DeLany *et al*., 2006	25	F	11	39.4	147.0	18.9	9.70	5.55	1.75
DeLany *et al*., 2006	31	M	11	45.0	146.0	21.0	10.80	5.83	1.85
DeLany *et al*., 2006	29	M	11	42.6	143.0	19.5	10.80	6.42	1.68
Ekelund *et al*., 2001	11	F	9	37.0	139.0	19.2	8.30	5.14	1.61
Ekelund *et al*., 2001	15	M	9	33.0	140.0	16.8	8.90	5.20	1.71
Ekelund *et al*., 2002	10	F	17	61.4	166.0	22.3	10.40	6.00	1.73
Ekelund *et al*., 2002	8	M	18	73.7	180.0	22.7	13.20	7.20	1.83
Ekelund *et al*., 2004	11	F	10	37.0	139.0	19.2	8.20	5.10	1.61
Ekelund *et al*., 2004	15	M	10	33.0	140.0	16.8	8.90	5.20	1.71
Ekelund *et al*., 2004	10	F	17	62.3	166.0	22.6	10.40	6.20	1.68
Ekelund *et al*., 2004	15	M	18	71.4	179.0	22.3	14.40	8.00	1.80
Fontvieille *et al*., 1993	15	M	5	21.1	114.0	16.2	5.90	4.34	1.36
Fontvieille *et al*., 1993	13	F	6	18.9	112.0	15.1	5.60	4.00	1.40
Goran *et al*., 1993b	14	F	5	21.0	113.0	16.4	5.50	4.54	1.21
Goran *et al*., 1993b	16	M	5	20.3	112.0	16.2	6.00	4.74	1.27
Goran *et al*., 1995	34	F	5	20.1	110.0	16.4	5.20	4.20	1.24
Goran *et al*., 1995	36	M	5	20.1	111.0	16.1	5.80	4.60	1.26
Goran *et al*., 1998c	11	M	5	21.3	113.0	16.7	6.60	4.90	1.35
Goran *et al*., 1998c	11	F	6	21.5	114.0	16.5	5.70	4.57	1.25
Goran *et al*., 1998c	11	M	6	25.2	120.0	17.5	7.50	5.30	1.42
Goran *et al*., 1998c	11	F	7	24.8	121.0	16.9	7.60	4.80	1.58
Goran *et al*., 1998c	11	M	9	37.8	138.0	19.8	8.70	5.90	1.47
Hernandez-Triana *et al*., 2002	5	M	5	18.0	110.0	15.0	6.30	3.72	1.69
Hernandez-Triana *et al*., 2002	6	F	6	19.5			6.10	3.76	1.62
Hoffman *et al*., 2000	14	F	10	30.9			8.08	4.72	1.71
Hoffman *et al*., 2000	14	M	10	32.0			9.03	4.89	1.85
Hoos *et al*., 2003	8	F	6	18.6	109.0	15.7	5.70	3.98	1.43
Hoos *et al*., 2003	3	M	9	21.8	121.0	14.9	7.30	4.34	1.68
Kaskoun *et al*., 1994	23	F	6	20.7			5.63	3.86	1.46
Kaskoun *et al*., 1994	22	M	6	19.5			5.84	3.98	1.47
Lindquist *et al*., 2000	17	M	9	41.2	139.0	20.9	7.90	5.70	1.38

Table 37 (continued) SACN data set of all published doubly-labelled water (DLW) studies of children aged over one year

Study	n	Sex	Age years	Weight kg	Height cm	BMI	TEE MJ/d	BMR MJ/d	PAL
Lindquist *et al.*, 2000	13	F	10	42.9	141.0	20.8	7.90	5.56	1.42
Livingstone *et al.*, 1992	6	M	3	16.4	100.1		5.4	3.98	1.36
Livingstone *et al.*, 1992	2	F	3	15.9	103.0		4.83	3.85	1.26
Livingstone *et al.*, 1992	6	F	5	18.1	110.2		5.38	3.63	1.48
Livingstone *et al.*, 1992	6	M	5	17.9	111.6		6.8	3.87	1.76
Livingstone *et al.*, 1992	6	F	7	23.5	120.4		7.27	4.08	1.78
Livingstone *et al.*, 1992	6	M	7	25.4	125.8		7.98	4.53	1.76
Livingstone *et al.*, 1992	6	F	9	32.2	134.0		7.89	4.78	1.65
Livingstone *et al.*, 1992	6	M	9	30.2	135.2		9.68	4.96	1.95
Livingstone *et al.*, 1992	6	M	12	44.5	152.1		10.98	5.84	1.88
Livingstone *et al.*, 1992	6	F	12	44.8	157.3		10.1	5.33	1.90
Livingstone *et al.*, 1992	6	F	15	57.2	162.2		10.39	5.86	1.77
Livingstone *et al.*, 1992	6	M	15	56.4	173.1		13.03	6.84	1.90
Livingstone *et al.*, 1992	5	F	18	63.9	159.9		10.26	6.10	1.68
Livingstone *et al.*, 1992	5	M	18	78.5	181.3		16.73	8.37	2.00
Livingstone *et al.*, 1992	5	F	8	23.5	120.0	16.3	7.10	4.08	1.74
Livingstone *et al.*, 1992	6	M	8	25.4	126.0	16.0	8.00	4.54	1.76
Livingstone *et al.*, 1992	4	F	9	33.4	134.0	18.6	8.10	4.86	1.67
Livingstone *et al.*, 1992	5	M	9	30.2	135.0	16.6	9.80	4.96	1.98
Livingstone *et al.*, 1992	5	M	13	43.8	152.0	19.0	10.70	5.79	1.85
Livingstone *et al.*, 1992	5	F	13	45.1	158.0	18.1	11.70	5.35	2.19
Livingstone *et al.*, 1992	3	M	15	50.7			11.00	6.33	1.74
Livingstone *et al.*, 1992	3	F	16	55.4			9.60	5.76	1.67
Lopez-Alarcon *et al.*, 2004	5	F	4	21.3	111.0	17.3	6.00	3.85	1.56
Lopez-Alarcon *et al.*, 2004	7	M	5	19.7	109.0	16.6	6.20	3.95	1.57
Lopez-Alarcon *et al.*, 2004	10	M	5	20.0	112.0	15.9	6.10	4.01	1.52
Lopez-Alarcon *et al.*, 2004	7	F	5	20.7	113.0	16.2	5.70	3.83	1.49
Montgomery *et al.*, 2005	31	F	6	19.6	112.0	15.4	6.00	3.75	1.60
Montgomery *et al.*, 2005	32	M	6	20.6	115.0	16.3	6.90	4.09	1.69
Nagy *et al.*, 1997	22	M	7	31.6	129.0	18.4	7.10	5.20	1.37
Nagy *et al.*, 1997	9	F	8	28.8	125.0	18.2	6.70	4.80	1.40
Nagy *et al.*, 1997	25	F	8	32.8	129.0	19.1	6.90	4.90	1.41
Nagy *et al.*, 1997	20	M	8	29.7	130.0	17.4 6.90	5.10	1.35	

Table 37 (continued) SACN data set of all published doubly-labelled water (DLW) studies of children aged over one year

Study	n	Sex	Age years	Weight kg	Height cm	BMI	TEE MJ/d	BMR MJ/d	PAL
O'Connor *et al*., 2001	22	M	7	25.0	123.0	16.4	7.60	4.47	1.70
O'Connor *et al*., 2001	25	F	8	26.5	124.0	17.1	7.20	4.31	1.67
Perks *et al*., 2000	27	F	13	49.4	154.0	20.5	9.60	5.70	1.68
Perks *et al*., 2000	23	M	13	45.1	140.0	18.3	10.10	5.86	1.72
Prentice *et al*., 1988	8	M	2	11.2			3.89	2.72	1.43
Prentice *et al*., 1988	8	F	2	11.2			3.89	2.66	1.46
Prentice *et al*., 1988	6	F	2	13.3			4.51	3.18	1.42
Prentice *et al*., 1988	6	M	2	13.3			4.51	3.25	1.39
Rennie *et al*., 2005	21	F	7	22.4	122.0	15.1	6.40	4.02	1.59
Rennie *et al*., 2005	19	F	7	25.0	123.0	16.7	7.10	4.20	1.69
Rennie *et al*., 2005	29	M	7	24.5	126.0	15.5	7.80	4.48	1.74
Rennie *et al*., 2005	31	M	7	26.1	126.0	16.3	8.10	4.58	1.77
Roemmich *et al*., 1998	12	F	11	36.6			8.07	4.88	1.65
Roemmich *et al*., 1998	18	M	11	35.3			8.91	5.14	1.73
Roemmich *et al*., 1998	18	F	14	51.8			9.67	5.59	1.73
Roemmich *et al*., 1998	11	M	15	54.5			11.28	6.62	1.70
Roemmich *et al*., 2000	13	F	10	34.7	137.0	18.5	8.90	5.09	1.75
Roemmich *et al*., 2000	14	M	11	34.8	143.0	17.0	9.10	5.20	1.75
Roemmich *et al*., 2000	18	F	13	51.2	158.0	20.5	9.40	5.70	1.65
Roemmich *et al*., 2000	14	M	13	52.0	162.0	19.8	10.70	6.80	1.57
Rush *et al*., 2003	13	M	10	35.4	140.0	17.5	9.80	5.90	1.66
Rush *et al*., 2003	13	F	10	35.5	140.0	17.4	8.10	5.05	1.60
Salazar *et al*., 2000	14	M	4	17.6			6.19	3.80	1.63
Salazar *et al*., 2000	14	F	5	17.6			5.73	3.60	1.59
Saris *et al*., 1989	10	F	8	28.2			8.06	4.49	1.79
Saris *et al*., 1989	9	M	9	30.9			9.00	5.05	1.78
Sjoberg *et al*., l 2003	17	F	16	56.4	164.0	20.8	10.10	5.85	1.73
Sjoberg *et al*., 2003	18	M	16	64.1	176.0	20.8	12.70	7.38	1.72
Spadano *et al*., l 2005	28	F	10	33.7	141.0	16.9	8.20	5.20	1.58
Spadano *et al*., 2005	28	F	12	45.3	154.0	19.0	9.40	5.92	1.59
Spadano *et al*., 2005	24	F	15	58.4	165.0	21.6	10.40	5.85	1.78
Sun *et al*., 1998	30	M	7	30.5	128.0	18.6	7.20	5.29	1.36
Sun *et al*., 1998	29	F	8	34.5	129.0	20.7	7.20	5.06	1.42
Sun *et al*., 1998	21	M	8	29.3	130.0	17.3	7.10	5.20	1.37
Sun *et al*., 1998	18	F	8	41.2	132.0	23.6	8.10	5.35	1.51
Sun *et al*., 1999	30	F	8	31.6	128.0	18.7	7.00	4.80	1.46
Sun *et al*., 1999	34	M	8	34.9	133.0	21.8	7.30	5.60	1.30

Table 37 (continued) SACN data set of all published doubly-labelled water (DLW) studies of children aged over one year

Study	n	Sex	Age years	Weight kg	Height cm	BMI	TEE MJ/d	BMR MJ/d	PAL
Sun *et al.*, 1999	30	M	9	34.4	132.0	19.3	7.10	5.60	1.27
Sun *et al.*, 1999	18	F	9	40.5	134.0	19.2	7.70	5.60	1.38
Treuth *et al.*, 1998	12	F	8	28.5	129.0	17.1	6.59	4.50	1.46
Treuth *et al.*, 1998	12	F	9	46.5	134.0	25.9	8.40	5.40	1.56
Treuth *et al.*, 2000b	30	F	9	27.2	130.0	15.9	7.10	4.41	1.61
Treuth *et al.*, 2000b	44	F	9	28.0	130.0	16.5	7.40	4.47	1.66
Treuth *et al.*, 2000b	27	F	9	29.6	131.0	17.2	7.50	4.58	1.64
Trowbridge *et al.*, 1997	18	F	8	40.3			7.40	5.00	1.48
Trowbridge *et al.*, 1997	27	F	8	33.8			7.40	5.10	1.45
Trowbridge *et al.*, 1997	13	M	8	28.5			7.80	5.40	1.44
Trowbridge *et al.*, 1997	17	M	8	30.8			7.70	5.50	1.40
Valencia *et al.*, 1995	10	M	8	26.9			6.60	4.67	1.41
Valencia *et al.*, 1995	10	M	8	27.2			7.49	4.70	1.59
Vasquez *et al.*, 2006	12	F	4	23.1	108.0	20.6	6.20	4.00	1.55
Vasquez *et al.*, 2006	12	M	4	22.3	108.0	19.1	6.80	4.23	1.61
Wong *et al.*, 1994	9	F	13	43.3	155.0	17.8	9.70	5.24	1.85
Wong *et al.*, 1999	41	F	13	57.5	160.0	22.5	10.10	5.57	1.81
Wong *et al.*, 1999	40	F	14	53.2	159.0	20.9	11.80	5.90	2.00
Wren 1997	8	M	5	19.1			5.80	3.94	1.47
Wren 1997	8	F	5	18.5			5.22	3.68	1.42

Appendix 13 – Comparisons with the existing DRVs for energy from the 1991 COMA report 'Dietary Reference Values for Energy and Nutrients for the United Kingdom'.

Overarching differences

453. The energy reference values detailed in this SACN report derive from a methodology which differs from that employed in 1991 by the Committee on Medical Aspects of Food Policy (COMA) (Department of Health (DH) 1991) in several ways. In contrast to the COMA report (DH, 1991):

- Energy reference values have been calculated from rates of total energy expenditure (TEE) assessed by the doubly labelled water (DLW) method. This has been used either directly as a measure of TEE for infants or, in all other cases, to identify suitable physical activity level (PAL) values which have been employed within a factorial calculation as basal metabolic rate (BMR) x PAL.
- BMR has been estimated using the Henry prediction equations (Henry 2005), resulting in slightly lower BMR values than would have been estimated from the Schofield equations (Schofield *et al.*, 1985) used by COMA in 1991.
- The population PAL values identified are higher. In this report, a value of 1.63 has been used as the population PAL value for adults compared to a PAL value of 1.4 commonly cited from the COMA report (Table 2.8 DH, 1991).
- A prescriptive approach has been adopted, calculating energy reference values on the basis of healthy body weights (i.e. using a desirable body mass index – BMI) which gives slightly lower values than those used by COMA.

Infants aged 1-12 months

454. COMA defined energy reference values for infants aged 0-12 months on the basis of available evidence on TEE and energy intakes. Energy reference values for infants were only defined for breast milk substitute-fed infants as it was felt that a reference value for breast-fed infants was meaningless in practice.

455. SACN followed the approach of the FAO/WHO/UNU (Butte, 2005; FAO, 2004) using more recent infant growth data from the UK-WHO Growth Standards (RCPCH, 2011). Separate values are provided for breast-fed and breast milk substitute-fed infants, and values are also given for when the method of feeding is mixed or not known. The new energy reference values described in this SACN report are

10-14% higher at 0-3 months but are lower by between 7-18% for infants after three months of age compared to the COMA values (see Table 38).

Children and adolescents aged 1-18 years

456. For children aged 1-10 years, in the absence of sufficient TEE data, COMA based its reference values on energy intake data. For older children and adolescents (aged 10-18 years), COMA defined energy reference values with a factorial model assigning PAL values of 1.56 for boys and 1.48 for girls with small additions made for growth costs. The PAL values used were lower for girls compared to boys, with the gender differences based on the view that girls exhibited lower levels of physical activity than boys. Average weights and heights from studies available at the time were used by COMA to calculate BMR using the Schofield equations.

457. SACN calculated energy reference values for boys and girls aged 1-18 years using a factorial model. PAL values were identified for three age groups (1-<3, 3-<10 10-18 years) from an analysis of DLW measures of TEE and adjusted for growth in terms of a 1% increase. As discussed in Appendix 5 (paragraph 276), in the DLW data sets examined in this SACN report no differences in PAL with gender were identified for children (or adults) and the same PAL values are therefore used for boys and girls. Three PAL values are presented in the current report for the three age groups of children representing "less active", "typically active" and "more active" (see paragraph 461 for further detail under the adults section). The new energy reference values were calculated from BMR values estimated using Henry equations based on what can be considered best estimates of healthy body weights i.e. mean weights from the UK-WHO Growth Standards (RCPCH, 2011) (ages 1 to 4 years) and from the 50th centile of the UK 1990 reference for children and adolescents (Freeman *et al*., 1995) for children aged more than four years.

458. Table 38 shows energy reference values for infants, children and adolescents in the current SACN report based on median PAL values for the various age groups compared with summary values reported by COMA (in Table 2.6 DH, 1991), with the change in this report shown as a percentage. Some of the body weights at the various ages used to calculate values in the two reports vary slightly and this explains some of the differences although most are due to the different methods of calculation. The overall pattern of differences is for, lower values from 3 months to 10 years, with higher values for adolescents, especially girls (up to 16% higher) due to use of a higher PAL value.

Table 38 Energy reference values for infants, children and adolescents in the current report compared with values reported by COMA[a]

Age	Energy reference values (MJ/d)					
	COMA (1991)[a]		SACN 2011		Change (±%)	
	Boys	Girls	Boys	Girls	Boys	Girls
0-3 months	2.3	2.2	2.6[b]	2.4[b]	14	10
4-6 months	2.9	2.7	2.7[b]	2.5[b]	-8	-7
7-9 months	3.4	3.2	2.9[b]	2.7[b]	-15	-16
10-12 months	3.9	3.6	3.2[b]	3.0[b]	-16	-18
1-3 years	5.2	4.9	4.1	3.8	-20	-22
4-6 years	7.2	6.5	6.2	5.8	-14	-11
7-10 years	8.2	7.3	7.6	7.2	-8	-2
11-14 years	9.3	7.7	9.8	9.1	6	18
15-18 years	11.5	8.8	12.6	10.2	9	15

[a] DH, 1991

[b] Using the comparable values for breast milk substitute fed infants (see Table 5).

Adults

459. The COMA report listed EAR values for energy for men and women aged 19-29 years and 30-59 years calculated as BMR x PAL. BMR was calculated from modified Schofield equations (Schofield *et al.*, 1985) for a wide range of body weights, and EAR values were listed for nine PAL values ranging from 1.4 to 2.2. Summary values were then presented for men and women aged 19-49 years and 50-59 years using a the PAL value of 1.4 (Table 2.8 DH, 1991).

460. COMA derived its PAL values from an analysis of PAR values for specific activities because at that time more reliable information on PAL values was unavailable. COMA assumed that much of the population had an inactive lifestyle and recommended that when activity patterns were unknown, energy reference values for the adult population should be based on a PAL value of 1.4.

461. This SACN report employs a different approach to identifying PAL values and to formulating specific recommendations for population subgroups. Thus, the magnitude and distribution of PAL values reflect measured values of TEE and BMR in reference populations. For adults in this report, PAL values of 1.49, 1.63 and 1.78 equate to the 25th, median and 75th centile. These values represent the less active, typically active, and the more active, and can only be equated to lifestyles in very general categories: i.e. sedentary, low and moderate activity. In practice, some sedentary individuals may have energy requirements slightly lower than implied by the less active PAL value and some active individuals may have energy requirements higher than implied by the more active PAL value. Judgements must be made where it is deemed necessary to identify energy requirements more precisely. The likely extra energy required for specific activities can aid such judgements (see Table 13).

462. The EAR values for adults reported by COMA were calculated from median body weights from a 1980 survey of adult heights and weights in Great Britain equivalent to BMI values of 24.1 and 24. 9 kg/m^2 for men and 23.1 and 24.7 kg/m^2 for women, for ages 19-49 and 50-59 years respectively. Although not discussed as such, COMA identified weight-maintaining energy reference values and gave only brief consideration to a healthy PAL value (i.e. an additional 0.1 PAL was suggested for exercise associated with maintenance of cardiovascular health, increasing PAL from 1.4 in a previous non-active group to 1.5).

463. In contrast, in this SACN report the increasing prevalence of overweight and obesity has been recognised and the new energy reference values are prescriptive i.e. they are calculated for body weights which are consistent with long-term good health. In adults, a healthy body weight range is generally defined as a BMI between 18.5-24.9kg/m^2, however, for the purposes of calculating the revised EAR values, healthy body weights are identified as body weights equivalent to a BMI of 22.5kg/m^2 from current mean heights for men and women (see paragraph 92). This approach is similar in principle to that taken in the FAO/WHO/UNU report (FAO, 2004) which was also prescriptive (or "normative"), recommending that reference EAR values should be identified in relation to height and listing values which would maintain BMI between 18.5 and 24.9 kg/m^2.

464. In this report, the influence of age on energy requirements is limited to that associated with the fall with age in BMR per kg body weight. The recommendations in terms of PAL values, therefore, are the same for healthy mobile free-living older adults as for younger adults. This is in recognition that an increasing fraction of older adults can and do remain physically active. Evidence suggests that individuals who are able to maintain higher levels of physical activity of any sort will gain benefit in terms of lower mortality (Manini *et al*., 2006). With an increasing lack of mobility, energy expenditure and requirements will fall so that PAL values at or below the lower quartile (i.e. PAL=1.49) will become more appropriate. For those who are immobile, falling food intakes associated with reduced energy requirements increase the potential for nutrient deficiencies. As a result nutrient dense food becomes particularly important for this population group.

465. Despite being calculated using a PAL value about 16% higher than that used by COMA (DH, 1991), the revised all-adult energy reference values reported here are 3% higher for men and 7-9% higher for women (see Table 39). This is explained by the use of a 5-7% lower average body weight for the all-age category of adult men and women. It should be noted that these revised values are within the range of measurement error.

Table 39 Comparison of COMA[a] and new SACN mean adult population EAR values

Age group (years)	Weight (kg)	COMA EAR (MJ/d)[c]	SACN EAR (MJ/d)[d]	% change for SACN values
All men	69.2[b]	–	10.9	–
19-49	74.0	10.6	–	2.8
50-59	74.0	10.6	–	2.8
All women	58.7[b]	–	8.7	–
19-49	60.0	8.1	–	7.4
50-59	63.0	8.0	–	8.7

[a] *DH, 1991*

[b] Weights calculated for a Body Mass Index of 22.5kg.m^2 at mean heights reported in the 2009 Health Survey for England (NHS IC, 2010).

[c] From Table 2.8 COMA (DH, 1991) based on PAL=1.4 and BMR calculated from mean bodyweights from National Diet and Nutrition Survey 1986/1987

[d] New SACN summary values for adults at PAL = 1.63

466. Table 40 compares the new SACN EAR values with values calculated using the approach adopted by COMA at the same healthy weights and PAL of 1.63 used by SACN to allow a more like for like comparison of the different methodologies used. The differences are the result of the Henry BMR prediction equations (Henry, 2005) used in the present SACN report (based on height and weight) providing slightly lower estimates of BMR than the weight-based Schofield equations (about 4% for men under 65 years and 3% for women).

Table 40 Comparison of the new SACN EAR values with values calculated using the approach adopted by COMA[a] at the same weights and PAL value used by SACN

Age group (years)	Height (cm)	Weight (kg) BMI[b] = 22.5 kg/m²	COMA EAR (MJ/d)[c]	SACN EAR (MJ/d)[d]	% change for SACN values
Men					
19-24	178	71.5	12.1	11.6	-4.1
25-34	178	71.0	12.0	11.5	-4.2
35-44	176	69.7	11.4	11.0	-3.5
45-54	175	68.8	11.3	10.8	-4.4
55-64	174	68.3	11.3	10.8	-4.4
65-74	173	67.0	9.4	9.8	4.3
75+	170	65.1	9.2	9.6	4.3
Women					
19-24	163	59.9	9.4	9.1	-3.2
25-34	163	59.7	9.4	9.1	-3.2
35-44	163	59.9	9.1	8.8	-3.3
45-54	162	59.0	9.0	8.8	-2.2
55-64	161	58.0	9.0	8.7	-3.3
65-74	159	57.2	8.0	8.0	0
75+	155	54.3	7.9	7.7	-2.5

[a] DH, 1991

[b] Body Mass index (BMI)

[c] EAR= PAL (1.63) x Basal Metabolic Rate (BMR), with BMR calculated using the modified Schofield equations (DH, 1991)

[d] EAR= PAL (1.63) x BMR, with BMR calculated using the Henry equation (see Appendix 4)

Comparison with COMA report for pregnancy

467. The COMA report recommended an increment in EAR of 0.8MJ/d above the pre-pregnant EAR only during the last trimester. In the absence of sufficient evidence to revise this recommendation, SACN considered it prudent to retain the EAR for pregnancy set by COMA. For pregnant women, unlike other adults, SACN has set energy requirements (EAR's) at actual pre-conceptional body weights, rather than at healthy body weights (see paragraph 129).

Comparison with COMA report for lactation

468. The COMA report recommended an increment of 1.9-2.4MJ/day (454-574kcal/day additional energy for the first six months of lactation and recognised two distinctive groups of breastfeeding mothers firstly, women who practised exclusive or almost exclusive breastfeeding until the baby was 3-4 months old and then progressively introduced complementary foods as part of an active complementary feeding process which often lasted only a few months (Group 1). Secondly, women who introduced only limited complementary feeds after 3-4 months and whose intention was that breast milk should provide the primary source of nourishment for 6 months or more (Group 2).

469. The energy reference value identified in this report for lactation, an increment of 1.38MJ/day (330kcal/day) for the first six months, is the same as previously recommended in the US DRI report (IoM, 2005), but considerably lower than that recommended for the first six months by COMA. The COMA values are higher due to the application of an efficiency factor of 0.8, to adjust for the efficiency of conversion of maternal energy intake to milk, which is insecure and likely to overestimate the true synthetic cost.

Glossary

Accelerometry	A non-calorimetric method of assessing free living energy expenditure by monitoring activity and movement.
Adenosine triphosphate (ATP)	The cofactor that acts as an intermediate between catabolic, anabolic and energy expenditure reactions
Adipose tissue	Body fat storage tissue. Distributed under the skin and around body organs. Composed of cells that synthesise and store fat, releasing it for metabolism in fasting.
Anthropometry	Body measurements made non-invasively to assess body composition, physiological development and nutritional status.
Average	Used here as a general term embracing both median and mean
Basal Metabolic Rate (BMR)	Rate at which the body uses energy when it is at complete rest, when food and physical activity have minimal influence on metabolism.
Body Mass Index (BMI)	An index of fatness and obesity of older children and adults. Weight in kilograms divided by the square of height in meters.
Dietary Reference Value (DRV)	A term used to define the various expressions of estimated dietary requirements in individuals and population groups. DRVs comprise 3 levels of intake Lower Reference Nutrient Intake, Reference Nutrient Intake and Estimated Average Requirement (see glossary entry).
Direct calorimetry	Calorimetry is a method of energy expenditure measurement. Direct calorimetry is a measure of heat output from the body, as an index of energy expenditure.
Doubly labelled water (DLW)	The stable (non radioactive) isotope method for estimating energy expenditure in free living individuals over extended periods up to several weeks. Subjects consume water containing isotopes hydrogen ($^2H_2$) and oxygen (^{18}O).
Energy balance	The difference between metabolisable energy intake and total energy expenditure. A neutral energy balance occurs when energy intake is equal to energy expenditure.

Energy flux	Energy flux or turnover is the rate at which energy in all its forms flows through the body on a daily basis (chemical, work, thermal). For an individual in energy balance the energy flux is numerically the same as energy expenditure or energy intake. Provided energy intake matches energy expenditure then energy balance will be maintained whether the energy flux itself is at a higher or lower level.
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.
Excess Post exercise oxygen consumption (EPOC)	Excess oxygen consumption following exercise. Small increase in energy expenditure following exercise which persists for some time after the exertion itself has been completed.
Fat Mass	The component of body composition made up of fat.
Fat Free Mass (FFM)	The non fat component of body composition comprising muscle, bone, skin and organs.
Food Frequency Questionnaire	A method for assessing past dietary intake. A questionnaire asking the frequency of consumption of foods over a day/week/month etc.
Gross Energy (GE)	The total maximum amount of energy contained within food, determined by measuring the heat released after complete combustion to carbon dioxide and water.
Glycogen	The storage carbohydrate in the liver and muscles. A branched polymer of glucose units.
Heart rate monitoring (HRM)	A method for measuring free living energy expenditure. Heart rate is monitored minute by minute throughout the day using portable meters. The energy expenditure at a given heart rate can be estimated using an individual linear regression line of the relationship between oxygen consumption and heart rate.
Heat Increment of Feeding	See Themic Effect of Food.
Homeostasis	The control of key components, (such as temperature and blood constituent concentrations) to ensure consistency and physiological normalisation.
Hyperglycaemia	Elevated plasma concentration of glucose, caused by failure of the normal hormonal mechanisms of blood glucose control.
Hypoglycaemia	Abnormally low concentration of plasma glucose.

Indirect calorimetry	Calorimetry is the measurement of energy expenditure. Indirect calorimetry is the most commonly used approach. It is the calculation of energy expenditure by the measurement of oxygen consumption and carbon dioxide production.
Insulin resistance	Reduction in the biological activity of insulin sensitive peripheral tissues, which results in reduced disposal of glucose from plasma for any given concentration of insulin.
Kilocalorie (kcal)	Units used to measure the energy value of food, 1kcal = 4.18kJ
kilojoule (kJ)/ megajoule (MJ)	Units used to measure the energy value of food, 1kJ=1000 joules, 1MJ = 1 million joules
Lipolysis	The breakdown of fat molecules e.g. triglycerides.
Metabolisable energy (ME)	The energy contained within food that is available to human metabolism as ATP after digestion and absorption.
Metabolic equivalent (MET)	Specific unit of measurement of energy expenditure by the body: equivalent to the BMR of an average adult; 1 MET = 3.5 ml $O_2 . kg^{-1} . min^{-1}$
Net Metabolisable energy (NME)	The ATP producing capacity of foods which is available to the body excluding unavoidable energy use in nutrient absorption and excretion of waste products.
Non exercise activity thermogenesis (NEAT)	Increase in energy expenditure due to non volitional activity/ behaviours such as fidgeting, muscle tone, posture maintenance.
Normative	See prescriptive.
Physical Activity Level (PAL)	Daily total energy expenditure (TEE) expressed as multiple of Basal metabolic rate (BMR). It is calculated as TEE divided by BMR.
Physical activity-related energy expenditure (PAEE)	The component of energy expenditure related to physical activity.
Physical Activity Ratio (PAR)	Energy cost of different physical activities per unit of time expressed as a multiple of BMR.
Prescriptive	Relating to an ideal standard: e.g. of body weight or physical activity. Sometimes used interchangeably with normative. As distinct from *status quo*.

Reference Nutrient Intake (RNI)	The Reference Nutrient Intake for a nutient is the amount of the nutrient that is enough, or more than enough, for about 97% of people in a group. If the average intake of a group is at the RNI, then the risk of deficiency in the group is very small.
Resting metabolic rate (RMR)	Rate at which the body uses energy when it is at rest. Sometimes used interchangeably with BMR but RMR is not measured at the standardised metabolic state and can include TEF and EPOC.
Thermic Effect of Food (TEF)	The increase in heat production by the body after eating, due to both the metabolic energy cost of ingestion, digestion and the energy cost of forming tissue reserves of fat, glycogen and protein.
Total Energy Expenditure (TEE)	The sum of all the energy expended by an individual over the course of one day. It includes BMR, PAEE and TEF and represents the average amount of energy spent in a typical day.
US DRI (dietary reference intakes)	US term for dietary reference values (includes the terms average requirement, Recommended Daily Amount and tolerable upper levels for supplements)

Glossary of statistical terms

Confidence intervals	Gives a range around the estimate of a mean from a sample such that if samples of the same size are taken repeatedly from the same population and a confidence interval is calculated for each sample then 95% of these intervals should contain the true population mean.
Determination, Coefficient of	Also referred to as R-squared value. The square of the product moment correlation between two variables, so called because it expresses the proportion of the variance of one variable, Y, given by the other, X, when Y is expressed as a linear regression on X. More generally, if a dependent variable has multiple correlation R with a set of independent variables, R-squared is known as the coefficient of determination.
Linear Regression Analysis	• Simple linear regression – A statistical method that attempts to explain the relationship between a dependent variable and a single independent variable by fitting a linear equation to the observed data • Multiple linear regression – The regression of a dependent variable on more than one independent variable.
Mean	The sum of the observations divided by the number of observations: similar to the median only if the data set is normally distributed
Unweighted mean	The mean of a set of observations in which no weights are attached to them, except in the trivial sense that each is weighted equally
Median	The midpoint or 50^{th} centile of a distribution
Multiple regression techniques	Techniques by which multiple linear regression models are assessed to determine the independent variables that have the greatest influence on the dependent variable
Post-hoc Analysis	Refers to statistical analysis after the data has been collected and investigating trends that were not specified before the study was conducted.
Residuals	A term denoting a quantity remaining after some other quantity has been subtracted. In terms of regression modelling the values by which the observations differ from the model values are called residuals.

Standard Error of Estimates (SEE)	A measure (estimate) of the accuracy of predictions (the estimated standard deviation of the error in the model). Note that the true value is unknown, by definition, so the standard error of an estimate is itself an estimate.

Abbreviations

ATP	Adenosine Triphosphate
BMI	Body Mass Index
BMR	Basal Metabolic Rate
COMA	Committee on Medical Aspects of Food and Nutrition Policy
CV	Coefficient of Variation
DLW	Doubly Labelled Water
DRI	Dietary Reference Intake
DRV	Dietary Reference Value
EAR	Estimated Average Requirement
EE	Energy Expenditure
EFS	Expenditure Food Survey
EI	Energy Intake
EPOC	Excess Post Exercise Oxygen Consumption
FAO/WHO/UNU	Food and Agriculture Organization/World Health Organization/United Nations University
FFM	Fat Free Mass
FFQ	Food Frequency Questionnaire
FM	Fat Mass
GDA	Guideline Daily Amount
GE	Gross Energy
GWG	Gestational Weight Gain
HR	Heart Rate
HRM	Heart Rate Monitor
HSE	Health Survey for England
J	Joule
kJ	Kilojoule
ME	Metabolisable Energy
MET	Metabolic Energy Equivalent
MJ	Megajoule
NDNS	National Diet and Nutrition Survey
NFS	National Food Survey
NME	Net Metabolisable Energy
NEAT	Non-Exercise Activity Thermogenesis
PA	Physical Activity
PAEE	Physical Activity Energy Expenditure
PAL	Physical Activity Level
PAR	Physical Activity Ratio
REE	Resting Energy Expenditure
RMR	Resting Metabolic Rate
RQ	Respiration Quotient
SACN	Scientific Advisory Committee on Nutrition
SEE	Standard Error of Estimate

SI	System of Units
SPA	Spontaneous Physical Activity
TEE	Total Energy Expenditure
TEF	Thermic Effect of Food

Reference List

Abbott RA, Davies PS (2004) Habitual physical activity and physical activity intensity: their relation to body composition in 5.0-10.5-y-old children. *Eur J Clin Nutr* **58**(2): 285-291.

Addolorato G, Capristo E, Greco AV, Stefanini GF & Gasbarrini G (1998) Influence of chronic alcohol abuse on body weight and energy metabolism: is excess ethanol consumption a risk factor for obesity or malnutrition? *Journal of Internal Medicine* **244**, 387-395.

Al Adsani H, Hoffer LJ & Silva JE (1997) Resting Energy Expenditure is Sensitive to Small Dose Changes in Patients on Chronic Thyroid Hormone Replacement. *J Clin Endocrinol Metab* **82**, 1118-1125.

Albu J, Shur M, Curi M, Murphy L, Heymsfield SB & Pi-Sunyer FX (1997) Resting metabolic rate in obese, premenopausal black women. *Am J Clin Nutr* **66**, 531-538.

Alfonzo-Gonzalez G, Doucet E, Almeras N, Bouchard C & Tremblay A (2004) Estimation of daily energy needs with the FAO/WHO/UNU 1985 procedures in adults: comparison to whole-body indirect calorimetry measurements. *Eur J Clin Nutr* **58**, 1125-1131.

Anderson SE, Bandini LG, Dietz WH & Must A (2004) Relationship between temperament, nonresting energy expenditure, body composition, and physical activity in girls. *Int J Obes Relat Metab Disord* **28**, 300-306.

Andreasen CH, Stender-Petersen KL, Mogensen MS et al (2008) Low Physical Activity Accentuates the Effect of the FTO rs9939609 Polymorphism on Body Fat Accumulation. *Diabetes* **57**(1), 95-101.

Arciero PJ, Gardner AW, Calles-Escandon J, Benowitz NL & Poehlman ET (1995) Effects of caffeine ingestion on NE kinetics, fat oxidation, and energy expenditure in younger and older men. *Am J Physiol* **268**, E1192-E1198.

Arciero PJ, Goran MI & Poehlman ET (1993) Resting metabolic rate is lower in women than in men. *J Appl Physiol* **75**, 2514-2520.

Aresu M, Bécares L, Brage S*, et al.* (2009) *Health Survey for England - 2008: Physical activity and fitness*: The NHS Information Centre for Health and Social Care.

Arthur PG, Hartmann PE & Smith M (1987) Measurement of the milk intake of breast-fed infants. *J Pediatr Gastroenterol Nutr* **6**, 758-763.

Arvidsson D, Slinde F & Hulthen L (2005) Physical activity questionnaire for adolescents validated against doubly labelled water. *Eur J Clin Nutr* **59**, 376-383.

Astrup A (2006) How to maintain a healthy body weight. *Int J Vitam Nutr Res* **76**, 208-215.

Astrup A, Buemann B, Christensen NJ, Madsen J, Gluud C, Bennett P & Svenstrup B (1992) The contribution of body composition, substrates, and hormones to the variability in energy expenditure and substrate utilization in premenopausal women. *J Clin Endocrinol Metab* **74**, 279-286.

Astrup A, Toubro S, Cannon S, Hein P, Breum L & Madsen J (1990) Caffeine: a double-blind, placebo-controlled study of its thermogenic, metabolic, and cardiovascular effects in healthy volunteers. *Am J Clin Nutr* **51**, 759-767.

Astrup A, Toubro S, Dalgaard LT, Urhammer SA, Sorensen TI & Pedersen O (1999) Impact of the v/v 55 polymorphism of the uncoupling protein 2 gene on 24-h energy expenditure and substrate oxidation. *Int J Obes Relat Metab Disord* **23**, 1030-1034.

Atlantis E, Barnes EH & Singh MAF (2006) Efficacy of exercise for treating overweight in children and adolescents: a systematic review. *Int J Obes* **30**, 1027-1040.

Bailey BW, Tucker LA, Peterson TR & LeCheminant JD (2007) A prospective study of physical activity intensity and change in adiposity in middle-aged women. *Am J Health Promot* **21**, 492-497.

Ball EJ, O'Connor J, Abbott R, Steinbeck KS, Davies PS, Wishart C, Gaskin KJ & Baur LA (2001) Total energy expenditure, body fatness, and physical activity in children aged 6-9 y. *Am J Clin Nutr* **74**, 524-528.

Ball K, Mishra G & Crawford D (2002) Which aspects of socioeconomic status are related to obesity among men and women? *Int J Obes Relat Metab Disord* **26**, 559-565.

Bandini LG, Must A, Spadano JL & Dietz WH (2002) Relation of body composition, parental overweight, pubertal stage, and race-ethnicity to energy expenditure among premenarcheal girls. *Am J Clin Nutr* **76**, 1040-1047.

Bandini LG, Schoeller DA, Cyr HN & Dietz WH (1990a) Validity of reported energy intake in obese and nonobese adolescents. *Am J Clin Nutr* **52**, 421-425.

Bandini LG, Schoeller DA & Dietz WH (1990b) Energy expenditure in obese and nonobese adolescents. *Pediatr Res* **27**, 198-203.

Baranowski T, Baranowski JC, Cullen KW, Thompson DI, Nicklas T, Zakeri IE & Rochon J (2003) The Fun, Food, and Fitness Project (FFFP): the Baylor GEMS pilot study. *Ethn Dis* **13**, S30-S39.

Basterfield L, Adamson AJ, Parkinson KN, Maute U, Li PX, Reilly JJ & the Gateshead Millennium Study Core Team (2008) Surveillance of physical activity in the UK is flawed: validation of the Health Survey for England Physical Activity Questionnaire. *Arch Dis Child* **93**, 1054-1058.

Bell AC, Ge K & Popkin BM (2001) Weight gain and its predictors in Chinese adults. *Int J Obes Relat Metab Disord* **25**, 1079-1086.

Bensimhon DR, Kraus WE & Donahue MP (2006) Obesity and physical activity: a review. *Am Heart J* **151**, 598-603.

Berentzen T, Kring SII, Holst C, Zimmermann E, Jess T, Hansen T, Pedersen O, Toubro S, Astrup A & Sorensen TIA (2008) Lack of Association of Fatness-Related FTO Gene Variants with Energy Expenditure or Physical Activity. *J Clin Endocrinol Metab* **93**, 2904-2908.

Berkey CS, Rockett HR, Field AE, Gillman MW, Frazier AL, Camargo CA, Jr. & Colditz GA (2000) Activity, dietary intake, and weight changes in a longitudinal study of preadolescent and adolescent boys and girls. *Pediatrics* **105**, E56.

Berkey CS, Rockett HRH, Gillman MW & Colditz GA (2003) One-Year Changes in Activity and in Inactivity Among 10- to 15-Year-Old Boys and Girls: Relationship to Change in Body Mass Index. *Pediatrics* **111**, 836-843.

Bernstein RS, Thornton JC, Yang MU, Wang J, Redmond AM, Pierson RN, Jr., Sunyer FX & Van Itallie TB (1983) Prediction of the resting metabolic rate in obese patients. *Am J Clin Nutr* **37**, 595-602.

Bessey PQ & Wilmore DW (1988) The burned patient. In *Nutrition and Metabolism in Patient Care*, pp. 672-700. London: W.B. Saunders.

Bingham SA & Day N (2006) Commentary: Fat and breast cancer: time to re-evaluate both methods and results? *Int J Epidemiol* **35**, 1022-1024

Bingham SA, Goldberg GR, Coward WA, Prentice AM & Cummings JH (1989) The effect of exercise and improved physical fitness on basal metabolic rate. *Br J Nutr* **61**, 155-173.

Bingham SA, Luben R, Welch A, Wareham N, Khaw KT & Day N (2003) Are imprecise methods obscuring a relation between fat and breast cancer? *Lancet* **362**, 212-214.

Bingham SA, Welch AA, McTaggart A, Mulligan AA, Runswick SA, Luben R, Oakes S, Khaw KT, Wareham N & Day NE (2001) Nutritional methods in the European Prospective Investigation of Cancer in Norfolk. *Public Health Nutrition* **4**, 847-858.

Bisdee JT, James WP & Shaw MA (1989) Changes in energy expenditure during the menstrual cycle. *Br J Nutr* **61**, 187-199.

Bitar A, Fellmann N, Vernet J, Coudert J & Vermorel M (1999) Variations and determinants of energy expenditure as measured by whole-body indirect calorimetry during puberty and adolescence. *Am J Clin Nutr* **69**, 1209-1216.

Blaak EE, Westerterp KR, Bar-Or O, Wouters LJ & Saris WH (1992) Total energy expenditure and spontaneous activity in relation to training in obese boys. *Am J Clin Nutr* **55**, 777-782.

Black AE, Coward WA, Cole TJ & Prentice AM (1996) Human energy expenditure in affluent societies: an analysis of 574 doubly-labelled water measurements. *Eur J Clin Nutr* **50**, 72-92.

Black AE (1996) Physical activity levels from a meta-analysis of doubly labeled water studies for validating energy intake as measured by dietary assessment. *Nutr Rev* **54**, 170-174.

Black AE & Cole TJ (2000) Within- and between-subject variation in energy expenditure measured by the doubly-labelled water technique: implications for validating reported dietary energy intake. *Eur J Clin Nutr* **54**, 386-394.

Black AE, Prentice AM, Goldberg GR, Jebb SA, Bingham SA, Livingstone MB & Coward WA (1993) Measurements of total energy expenditure provide insights into the validity of dietary measurements of energy intake. *J Am Diet Assoc* **93**, 572-579.

Blair SN, LaMonte MJ & Nichaman MZ (2004) The evolution of physical activity recommendations: how much is enough? *Am J Clin Nutr* **79**, 913S-9920.

Blanc S, Schoeller DA, Bauer D, Danielson ME, Tylavsky F, Simonsick EM, Harris TB, Kritchevsky SB & Everhart JE (2004) Energy requirements in the eighth decade of life. *Am J Clin Nutr* **79**, 303-310.

Blaza S & Garrow JS (1983) Thermogenic response to temperature, exercise and food stimuli in lean and obese women, studied by 24 h direct calorimetry. *Br J Nutr* **49**, 171-180.

Blundell JE, Stubbs RJ, Hughes DA, Whybrow S & King NA (2003) Cross talk between physical activity and appetite control: does physical activity stimulate appetite? *Proc Nutr Soc* **62**, 651-661.

Bobbioni-Harsch E, Assimacopoulos-Jeannet F, Lehmann T, Munger R, Allaz AF & Golay A (1999) Leptin plasma levels as a marker of sparing-energy mechanisms in obese women. *Int J Obes Relat Metab Disord* **23**, 470-475.

Bogaert N, Steinbeck KS, Baur LA, Brock K & Bermingham MA (2003) Food, activity and family – environmental vs biochemical predictors of weight gain in children. *Eur J Clin Nutr* **57**, 1242-1249.

Bogardus C, Lillioja S, Ravussin E, Abbott W, Zawadzki JK, Young A, Knowler WC, Jacobowitz R & Moll PP (1986) Familial dependence of the resting metabolic rate. *N Engl J Med* **315**, 96-100.

Boreham C, Robson PJ, Gallagher AM, Cran GW, Savage JM & Murray LJ (2004) Tracking of physical activity, fitness, body composition and diet from adolescence to young adulthood: The Young Hearts Project, Northern Ireland. *Int J Behav Nutr Phys Act* **1**, 14.

Bosy-Westphal A, Eichhorn C, Kutzner D, Illner K, Heller M & Muller MJ (2003) The Age-Related Decline in Resting Energy Expenditure in Humans Is Due to the Loss of Fat-Free Mass and to Alterations in Its Metabolically Active Components. *J Nutr* **133**, 2356-2362.

Bouchard C, Tremblay A, Nadeau A, Despres JP, Theriault G, Boulay MR, Lortie G, Leblanc C & Fournier G (1989) Genetic effect in resting and exercise metabolic rates. *Metabolism* **38**, 364-370.

Bratteby LE, Sandhagen B, Fan H & Samuelson G (1997) A 7-day activity diary for assessment of daily energy expenditure validated by the doubly labelled water method in adolescents. *Eur J Clin Nutr* **51**, 585-591.

Bratteby LE, Sandhagen B & Samuelson G (2005) Physical activity, energy expenditure and their correlates in two cohorts of Swedish subjects between adolescence and early adulthood. *Eur J Clin Nutr* **59**, 1324-1334.

Bryant MJ, Lucove JC, Evenson KR & Marshall S (2007) Measurement of television viewing in children and adolescents: a systematic review. *Obes Rev* **8**, 197-209.

Buemann B, Astrup A, Christensen NJ & Madsen J (1992) Effect of moderate cold exposure on 24-h energy expenditure: similar response in postobese and nonobese women. *Am J Physiol* **263**, E1040-E1045.

Buhl KM, Gallagher D, Hoy K, Matthews DE & Heymsfield SB (1995) Unexplained disturbance in body weight regulation: diagnostic outcome assessed by doubly labeled water and body composition analyses in obese patients reporting low energy intakes. *J Am Diet Assoc* **95**, 1393-1400.

Burke V, Giangiulio N, Gillam HF, Beilin LJ & Houghton S (2003) Physical activity and nutrition programs for couples: a randomized controlled trial. *J Clin Epidemiol* **56**, 421-432.

Butte NF (2005) Energy requirements of infants. *Public Health Nutr* **8**, 953-967.

Butte NF & Ellis KJ (2003) Comment on "Obesity and the Environment: Where Do We Go from Here?". *Science* **301**, 598b.

Butte NF, Ellis KJ, Wong WW, Hopkinson JM, Smith EO (2003a) Composition of gestational weight gain impacts maternal fat retention and infant birth weight. *Am J Obstet Gynecol* **189**(5):1423-1432.

Butte NF & Hopkinson JM (1998) Body composition changes during lactation are highly variable among women. *J Nutr* **128**, 381S-385S.

Butte NF, Hopkinson JM, Wong WW, Smith EO & Ellis KJ (2000b) Body composition during the first 2 years of life: an updated reference. *Pediatr Res* **47**, 578-585.

Butte NF & King JC (2002) *Energy requirements during pregnancy and lactation. Energy background paper prepared for the joint FAO/WHO/UNU Consultation on Energy in Human Nutrition.*

Butte NF & King JC (2005) Energy requirements during pregnancy and lactation. *Public Health Nutr* **8**, 1010-1027.

Butte NF, Treuth MS, Mehta NR, Wong WW, Hopkinson JM & Smith EO (2003b) Energy requirements of women of reproductive age. *Am J Clin Nutr* **77**, 630-638.

Butte NF, Wong WW, Ferlic L, Smith EO, Klein PD & Garza C (1990) Energy expenditure and deposition of breast-fed and formula-fed infants during early infancy. *Pediatr Res* **28**, 631-640.

Butte NF, Wong WW & Garza C (1989) Energy cost of growth during infancy. *Proceedings of the Nutrition Society* **48**, 303-312.

Butte NF, Wong WW & Hopkinson JM (2001) Energy requirements of lactating women derived from doubly labeled water and milk energy output. *J Nutr* **131**, 53-58.

Butte NF, Wong WW, Hopkinson JM, Heinz CJ, Mehta NR & Smith EO (2000a) Energy requirements derived from total energy expenditure and energy deposition during the first 2 y of life. *Am J Clin Nutr* **72**, 1558-1569.

Butte NF, Wong WW, Treuth MS, Ellis KJ & O'Brian Smith E (2004) Energy requirements during pregnancy based on total energy expenditure and energy deposition. *Am J Clin Nutr* **79**, 1078-1087.

Byrne NM, Hills AP, Hunter GR, Weinsier RL & Schutz Y (2005) Metabolic equivalent: one size does not fit all. *J Appl Physiol* **99**, 1112-1119.

Byrne NM, Weinsier RL, Hunter GR, Desmond R, Patterson MA, Darnell BE & Zuckerman PA (2003) Influence of distribution of lean body mass on resting metabolic rate after weight loss and weight regain: comparison of responses in white and black women. *Am J Clin Nutr* **77**, 1368-1373.

Caballero B, Davis S, Davis CE, Ethelbah B, Evans M, Lohman T, Stephenson L, Story M, White J (1998) Pathways: A school-based program for the primary prevention of obesity in American Indian chidlren *J Nutr Biochem* **9**, 535-543.

Caballero B, Clay T, Davis SM*, et al.* (2003) Pathways: a school-based, randomized controlled trial for the prevention of obesity in American Indian schoolchildren. *Am J Clin Nutr* **78**, 1030-1038.

Cai G, Cole SA, Butte NF, Voruganti VS, Comuzzie AG (2008) Genome-wide scan revealed genetic loci for energy metabolism in Hispanic children and adolescents. *Int J Obes* **32**(4):579-585.

Cairella G, Romagnoli F, Cantarelli P, Valentini P & Tarsitani G (1998) School oriented intervention on dietary education: Results of phase 1. *Int J Obes Relat Metab Disord* **22**, S254 (Abstract).

Carpenter WH, Fonong T, Toth MJ, Ades PA, Calles-Escandon J, Walston JD & Poehlman ET (1998) Total daily energy expenditure in free-living older African-Americans and Caucasians. *Am J Physiol Endocrinol Metab* **274**, 96-101.

Carpenter WH, Poehlman ET, O'Connell M & Goran MI (1995) Influence of body composition and resting metabolic rate on variation in total energy expenditure: a meta-analysis. *Am J Clin Nutr* **61**, 4-10.

Casper RC, Schoeller DA, Kushner R, Hnilicka J, Gold ST (1991) Total daily energy expenditure and activity level in anorexia nervosa. *Am J Clin Nutr* **53**(5), 1143-1150.

Caspersen CJ, Powell KE & Christenson GM (1985) Physical activity, exercise, and physical fitness: definitions and distinctions for health-related research. *Public Health Rep* **100**, 126-131.

Cecil JE, Tavendale R, Watt P, Hetherington MM & Palmer CNA (2008) An Obesity-Associated FTO Gene Variant and Increased Energy Intake in Children. *N Engl J Med* **359**, 2558-2566.

Champagne CM, Baker NB, DeLany JP, Harsha DW & Bray GA (1998) Assessment of energy intake underreporting by doubly labeled water and observations on reported nutrient intakes in children. *J Am Diet Assoc* **98**, 426-433.

Chief Medical Officers of England, Scotland, Wales and Northern Ireland (2011). *Start Active, Stay Active. A report on physical activity for health from the four home countries' Chief Medical Officers.* Department of Health, Welsh Government, The Scottish Government, Department of Health SSaPS.

Chitwood LF, Brown SP, Lundy MJ & Dupper MA (1996) Metabolic propensity toward obesity in black vs white females: responses during rest, exercise and recovery. *Int J Obes Relat Metab Disord* **20**, 455-462.

Chong PK, Jung RT, Scrimgeour CM & Rennie MJ (1994) The effect of pharmacological dosages of glucocorticoids on free living total energy expenditure in man. *Clin Endocrinol (Oxf)* **40**, 577-581.

Cole TJ (1995) Conditional reference charts to assess weight gain in British infants. *Arch Dis Child* **73**, 8-16.

Cole TJ, Bellizzi MC, Flegal KM, Dietz WH (2000) Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ* **320** (7244), 1240-1243.

Cole TJ, Faith MS, Pietrobelli A & Heo M (2005) What is the best measure of adiposity change in growing children: BMI, BMI %, BMI z-score or BMI centile? *Eur J Clin Nutr* **59**, 419-425.

Collins LC, Cornelius MF, Vogel RL, Walker JF & Stamford BA (1994) Effect of caffeine and/or cigarette smoking on resting energy expenditure. *Int J Obes Relat Metab Disord* **18**, 551-556.

Cooper AR, Moore LA, McKenna J & Riddoch C (2000) What is the magnitude of blood pressure response to a programme of moderate intensity exercise? Randomised controlled trial among sedentary adults with unmedicated hypertension. *Br J Gen Pract* **50**, 958-962.

Corbett J, Dobbie F, Doig M*, et al.* (2010) *Scottish Health Survey 2009 - Volume 1: Main report*: The Scottish Government.

Corder K, Brage S, Mattocks C, Ness A, Riddoch C, Wareham NJ & Ekelund U (2007) Comparison of two methods to assess PAEE during six activities in children. *Med Sci Sports Exerc* **39**, 2180-2188.

Correa A, Gilboa SM, Besser LM et al. Diabetes mellitus and birth defects. (2008) *Am J Obstet Gynecol* **199**(3):237-239.

Craig SB, Bandini LG, Lichtenstein AH, Schaefer EJ & Dietz WH (1996) The impact of physical activity on lipids, lipoproteins, and blood pressure in preadolescent girls. *Pediatrics* **98**, 389-395.

Cunningham JJ (1980) A reanalysis of the factors influencing basal metabolic rate in normal adults. *Am J Clin Nutr* **33**, 2372-2374.

Cunningham JJ (1991) Body composition as a determinant of energy expenditure: a synthetic review and a proposed general prediction equation. *Am J Clin Nutr* **54**, 963-969.

Danforth E Jr & Burger A (1984) The role of thyroid hormones in the control of energy expenditure. *Clin Endocrinol Metab* **13**, 581-595.

Das SK, Moriguti JC, McCrory MA, Saltzman E, Mosunic C, Greenberg AS & Roberts SB (2001) An Underfeeding Study in Healthy Men and Women Provides Further Evidence of Impaired Regulation of Energy Expenditure in Old Age. *J Nutr* **131**, 1833-1838.

Das SK, Saltzman E, McCrory MA, Hsu LK, Shikora SA, Dolnikowski G, Kehayias JJ & Roberts SB (2004) Energy expenditure is very high in extremely obese women. *J Nutr* **134**, 1412-1416.

Dauncey MJ (1981) Influence of mild cold on 24 h energy expenditure, resting metabolism and diet-induced thermogenesis. *Br J Nutr* **45**, 257-267.

Davidson L, McNeill G, Haggarty P, Smith JS & Franklin MF (1997) Free-living energy expenditure of adult men assessed by continuous heart-rate monitoring and doubly-labelled water. *Br J Nutr* **78**, 695-708.

Davies PS, Day JM & Lucas A (1991a) Energy expenditure in early infancy and later body fatness. *Int J Obes* **15**, 727-731.

Davies PS, Livingstone MB, Prentice AM, Coward WA, Jagger SE, Stewart CM & Strain JJ (1991b) Total energy expenditure during childhood and adolescence. *Proc Nutr Soc* **50**, 1-14A.

Davies PS & White A (1995) Energy expenditure in children aged 1.5 to 4.5 years: a comparison with current recommendations for energy intake. *Eur J Clin Nutr*, 360-364.

Davis S, Gomez Y, Lambert L & Skipper B (1993) Primary prevention of obesity in American Indian children. In *Pevention and treatment of childhood obesity*, pp. 167-180 [CL Williams and SYS Kimm, editors]. New York: New York Academy of Sciences.

Davison KK & Birch LL (2001) Child and parent characteristics as predictors of change in girls' body mass index. *Int J Obes Relat Metab Disord* **25**, 1834-1842.

Davison KK, Marshall SJ & Birch LL (2006) Cross-sectional and longitudinal associations between TV viewing and girls' body mass index, overweight status, and percentage of body fat. *J Pediatr* **149**, 32-37.

Day DS, Gozansky WS, Van Pelt RE, Schwartz RS & Kohrt WM (2005) Sex hormone suppression reduces resting energy expenditure and {beta}-adrenergic support of resting energy expenditure. *J Clin Endocrinol Metab* **90**, 3312-3317.

DeLany JP, Bray GA, Harsha DW & Volaufova J (2002) Energy expenditure in preadolescent African American and white boys and girls: the Baton Rouge Children's Study. *Am J Clin Nutr* **75**, 705-713.

DeLany JP, Bray GA, Harsha DW & Volaufova J (2004) Energy expenditure in African American and white boys and girls in a 2-y follow-up of the Baton Rouge Children's Study. *Am J Clin Nutr* **79**, 268-273.

DeLany JP, Bray GA, Harsha DW & Volaufova J (2006) Energy expenditure and substrate oxidation predict changes in body fat in children. *Am J Clin Nutr* **84**, 862-870.

Demattia L, Lemont L & Meurer L (2007) Do interventions to limit sedentary behaviours change behaviour and reduce childhood obesity? A critical review of the literature. *Obes Rev* **8**, 69-81.

Dempsey DT, Guenter P, Mullen JL, Fairman R, Crosby LO, Spielman G & Gennarelli T (1985) Energy expenditure in acute trauma to the head with and without barbiturate therapy. *Surg Gynecol Obstet* **160**, 128-134.

Dennison BA, Russo TJ, Burdick PA & Jenkins PL (2004) An intervention to reduce television viewing by preschool children. *Arch Pediatr Adolesc Med* **158**, 170-176.

Department of Health (1991) *Dietary Reference Values for Food Energy and Nutrients for the United Kingdom* no. 41. London: HMSO.

Department of Health (2004) *At least five a week. Evidence on the impact of physical activity and its relationship to health. A report from the Chief Medical Officer.* London: Department of Health.

Deriaz O, Dionne F, Perusse L et al. (1994) DNA variation in the genes of the Na,K-adenosine triphosphatase and its relation with resting metabolic rate, respiratory quotient, and body fat. J Clin Invest **93**(2):838-843.

Di Blasio AM, van Rossum EF, Maestrini S et al (2003) The relation between two polymorphisms in the glucocorticoid receptor gene and body mass index, blood pressure and cholesterol in obese patients. *Clin Endocrinol* (Oxf) **59**(1), 68-74.

Diepvens K, Westerterp KR & Westerterp-Plantenga MS (2007) Obesity and thermogenesis related to the consumption of caffeine, ephedrine, capsaicin, and green tea. *Am J Physiol Regul Integr Comp Physiol* **292**, R77-R85.

Dietz WH (1998) Does energy expenditure affect changes in body fat in children? *Am J Clin Nutr* **67**, 190-191.

Dietz WH & Robinson TN (1998) Use of the body mass index (BMI) as a measure of overweight in children and adolescents. *J Pediatr* **132**, 191-193.

Diffey B, Piers LS, Soares MJ & O'Dea K (1997) The effect of oral contraceptive agents on the basal metabolic rate of young women. *Br J Nutr* **77**, 853-862.

Dina C, Meyre D, Gallina S*, et al*. (2007) Variation in FTO contributes to childhood obesity and severe adult obesity. *Nat Genet* **39**, 724-726.

Dionne I, Despres JP, Bouchard C & Tremblay A (1999) Gender difference in the effect of body composition on energy metabolism. *Int J Obes Relat Metab Disord* **23**, 312-319.

Dionne IJ, Garant MJ, Nolan AA, Pollin TI, Lewis DG, Shuldiner AR & Poehlman ET (2002) Association between obesity and a polymorphism in the beta(1)-adrenoceptor gene (Gly389Arg ADRB1) in Caucasian women. *Int J Obes Relat Metab Disord* **26**, 633-639.

Dionne IJ, Turner AN, Tchernof A, Pollin TI, Avrithi D, Gray D, Shuldiner AR & Poehlman ET (2001) Identification of an interactive effect of beta3- and alpha2b-adrenoceptor gene polymorphisms on fat mass in Caucasian women. *Diabetes* **50**, 91-95.

Do R, Bailey SD, Desbiens K, Belisle A, Montpetit A, Bouchard C, Perusse L, Vohl MC & Engert JC (2008) Genetic Variants of FTO Influence Adiposity, Insulin Sensitivity, Leptin Levels, and Resting Metabolic Rate in the Quebec Family Study. *Diabetes* **57**, 1147-1150.

Donnelly JE, Jacobsen DJ, Whatley JE, Hill JO, Swift LL, Cherrington A, Polk B, Tran ZV & Reed G (1996) Nutrition and physical activity program to attenuate obesity and promote physical and metabolic fitness in elementary school children. *Obes Res* **4**, 229-243.

Drøyvold WB, Holmen J, Midthjell K & Lydersen S (2004) BMI change and leisure time physical activity (LTPA): an 11-y follow-up study in apparently healthy men aged 20-69 y with normal weight at baseline. *Int J Obes Relat Metab Disord* **28**, 410-417.

Durnin JV (1996) Energy requirements: general principles. *Eur J Clin Nutr* **50 Suppl 1**, S2-S9.

Eck LH, Bennett AG, Egan BM, Ray JW, Mitchell CO, Smith MA & Klesges RC (1997) Differences in macronutrient selections in users and nonusers of an oral contraceptive. *Am J Clin Nutr* **65**, 419-424.

Eddy TP, Nicholson AL & Wheeler EF (1965) Energy expenditures and dietary intakes in cerebral palsy. *Dev Med Child Neurol* **7**, 377-386.

Ekelund U, Aman J, Yngve A, Renman C, Westerterp K & Sjostrom M (2002) Physical activity but not energy expenditure is reduced in obese adolescents: a case-control study. *Am J Clin Nutr* **76**, 935-941.

Ekelund U, Brage S, Besson H, Sharp S, Wareham NJ (2008) Time spent being sedentary and weight gain in healthy adults: reverse or bidirectional causality? *Am J Clin Nutr* **88**(3), 612-617.

Ekelund U, Sardinha LB, Anderssen SA, Harro M, Franks PW, Brage S, Cooper AR, Andersen LB, Riddoch C & Froberg K (2004b) Associations between objectively assessed physical activity and indicators of body fatness in 9- to 10-y-old European children: a population-based study from 4 distinct regions in Europe (the European Youth Heart Study). *Am J Clin Nutr* **80**, 584-590.

Ekelund U, Yngve A, Brage S, Westerterp K & Sjostrom M (2004a) Body movement and physical activity energy expenditure in children and adolescents: how to adjust for differences in body size and age. *Am J Clin Nutr* **79**, 851-856.

Ekelund U, Sjostrom M, Yngve A et al. Physical activity assessed by activity monitor and doubly labeled water in children. Med Sci Sports Exerc 2001;33(2):275-281.

Elder SJ & Roberts SB (2007) The effects of exercise on food intake and body fatness: a summary of published studies. *Nutr Rev* **65**, 1-19.

Elia M (1992) Organ and tissue contribution to metabolic rate. In *Energy Metabolism: Tissue Determinants and Cellular Corollaries*, pp. 61-79. New York: Raven Press.

Elia M (1995) Changing concepts of nutrient requirements in disease: implications for artificial nutritional support. *Lancet* **345**, 1279-1284.

Elia M (1997) Tissue distribution and energetics in weigh loss and undernutrition. In *Physiology,Stress and Malnutrition: Functional Correlates and Nutritional Intervention* [HN Tucker and JM Kinney, editors]. New York: Lipcott-Raven.

Elia M (2000) Hunger disease: metabolic response to starvation, injury and sepsis. *Clin Nutr* **19**, 379-386.

Elia M (2005) Insights into energy requirements in disease. *Public Health Nutr* **8**, 1037-1052.

Elia M, Ritz P & Stubbs RJ (2000) Total energy expenditure in the elderly. *Eur J Clin Nutr* **54 Suppl 3**, S92-103.

Erlichman J, Kerbey AL & James WP (2002) Physical activity and its impact on health outcomes. Paper 2: Prevention of unhealthy weight gain and obesity by physical activity: an analysis of the evidence. *Obes Rev* **3**, 273-287.

FAO (2003) *Food energy - methods of analysis and conversion factors. Report of a Technical Workshop* no. 77. Rome: FAO.

FAO (2004) *Human energy requirements. Report of a Joint FAO/WHO/UNU Expert Consultation* no. 1. Rome: FAO.

Fellows IW, Bennett T & Macdonald IA (1985) The effect of adrenaline upon cardiovascular and metabolic functions in man. *Clin Sci (Lond)* **69**, 215-222.

Ferraro R, Lillioja S, Fontvieille AM, Rising R, Bogardus C & Ravussin E (1992) Lower sedentary metabolic rate in women compared with men. *J Clin Invest* **90**, 780-784.

Ferraro R & Ravussin E (1992) Fat mass in predicting resting metabolic rate. *Am J Clin Nutr* **56**, 460-461.

Figueroa-Colon R, Arani RB, Goran MI & Weinsier RL (2000) Paternal body fat is a longitudinal predictor of changes in body fat in premenarcheal girls. *Am J Clin Nutr* **71**, 829-834.

Filozof CM, Murua C, Sanchez MP, Brailovsky C, Perman M, Gonzalez CD & Ravussin E (2000) Low plasma leptin concentration and low rates of fat oxidation in weight-stable post-obese subjects. *Obes Res* **8**, 205-210.

Finch S, Doyle W, Lowe C, Gregory J, Bates CJ, Prentice A, Smithers G & Clarke PC (1998) *National Diet and Nutrition Survey: people aged 65 years and over. Volume 1: report of the diet and nutrition survey*. London: The Stationary Office.

Fitzgibbon ML, Stolley MR & Kirschenbaum DS (1995) An obesity prevention pilot program in African-American mothers and daughters. *J Nutr Educ* **27**, 93-97.

Fogelholm M & Kukkonen-Harjula K (2000) Does physical activity prevent weight gain – a systematic review. *Obes Rev* **1**, 95-111.

Fomom SJ, Haschke F, Ziegler EE & Nelson SE (1982) Body composition of reference children from birth to age 10 years. *Am J Clin Nutr* **35**, 1169-1175.

Fontvieille AM, Harper IT, Ferraro RT, Spraul M & Ravussin E (1993) Daily energy expenditure by five-year-old children, measured by doubly labeled water. *J Pediatr* **123**, 200-207.

Food Standards Agency (2002) McCance and Widdowson's the composition of foods, 6 ed. Cambridge: Royal Society of Chemistry.

Foresight (2007) Tackling obesities: future choices. Government Office of Science. www.foresight.gov.uk/Obesity/14.pdf.

Forman JN, Miller WC, Szymanski LM & Fernhall B (1998) Differences in resting metabolic rates of inactive obese African-American and Caucasian women. *Int J Obes Relat Metab Disord* **22**, 215-221.

Forster JL, Jeffery RW, Schmid TL & Kramer FM (1988) Preventing weight gain in adults: a pound of prevention. *Health Psychol* **7**, 515-525.

Forsum E, Kabir N, Sadurskis A & Westerterp K (1992) Total energy expenditure of healthy Swedish women during pregnancy and lactation. *Am J Clin Nutr* **56**, 334-342.

Forsum E, Lof M & Schoeller DA (2006) Calculation of energy expenditure in women using the MET system. *Med Sci Sports Exerc* **38**, 1520-1525.

Foster GD, Wadden TA, Swain RM, Anderson DA & Vogt RA (1999) Changes in resting energy expenditure after weight loss in obese African American and white women. *Am J Clin Nutr* **69**, 13-17.

Foster GD, Wadden TA & Vogt RA (1997) Resting energy expenditure in obese African American and Caucasian women. *Obes Res* **5**, 1-8.

Fox KR & Hillsdon M (2007) Physical activity and obesity. *Obes Rev* **8 Suppl 1**, 115-121.

Francis LA, Lee Y & Birch LL (2003) Parental weight status and girls' television viewing, snacking, and body mass indexes. *Obes Res* **11**, 143-151.

Frayling TM, Timpson NJ, Weedon MN*, et al.* (2007) A common variant in the FTO gene is associated with body mass index and predisposes to childhood and adult obesity. *Science* **316**, 889-894.

Freedson P, Pober D & Janz KF (2005) Calibration of accelerometer output for children. *Med Sci Sports Exerc* **37**, S523-S530.

Freeman JV, Cole TJ, Chinn S, Jones PR, White EM & Preece MA (1995) Cross sectional stature and weight reference curves for the UK, 1990. *Arch Dis Child* **73**, 17-24.

Fukagawa NK, Bandini LG & Young JB (1990) Effect of age on body composition and resting metabolic rate. *Am J Physiol* **259**, E233-E238.

Fuller NJ, Sawyer MB, Coward WA, Paxton P & Elia M (1996) Components of total energy expenditure in free-living elderly men (over 75 years of age): measurement, predictability and relationship to quality-of-life indices. *Br J Nutr* **75**, 161-173.

Gagnon J, Mauriege P, Roy S, Sjostrom D, Chagnon YC, Dionne FT, Oppert JM, Perusse L, Sjostrom L & Bouchard C (1996) The Trp64Arg mutation of the beta3 adrenergic receptor gene has no effect on obesity phenotypes in the Quebec Family Study and Swedish Obese Subjects cohorts. *J Clin Invest* **98**, 2086-2093.

Gallagher D, Belmonte D, Deurenberg P, Wang Z, Krasnow N, Pi-Sunyer FX & Heymsfield SB (1998) Organ-tissue mass measurement allows modeling of REE and metabolically active tissue mass. *Am J Physiol* **275**, E249-E258.

Gallagher D, Albu J, He Q et al. Small organs with a high metabolic rate explain lower resting energy expenditure in African American than in white adults. Am J Clin Nutr 2006;83(5):1062-1067.

Gannon B, DiPietro L & Poehlman ET (2000) Do African Americans have lower energy expenditure than Caucasians? *Int J Obes Relat Metab Disord* **24**, 4-13.

Garby L & Lammert O (1994) Between-subjects variation in energy expenditure: estimation of the effect of variation in organ size. *Eur J Clin Nutr* **48**, 376-378.

Garza C & Butte NF (1986) Energy concentration of human milk estimated from 24-h pools and various abbreviated sampling schemes. *J Pediatr Gastroenterol Nutr* **5**, 943-948.

Gerken T, Girard CA, Tung YC et al (2007) The obesity-associated FTO gene encodes a 2-oxoglutarate-dependent nucleic acid demethylase. *Science* **318**(5855), 1469-1472.

Gibney ER, Murgatroyd P, Wright A, Jebb S & Elia M (2003) Measurement of total energy expenditure in grossly obese women: comparison of the bicarbonate-urea method with whole-body calorimetry and free-living doubly labelled water. *Int J Obes Relat Metab Disord* **27**, 641-647.

Gittelsohn J, Evans M, Helitzer D, Anliker J, Story M, Metcalfe L, Davis S & Iron CP (1998) Formative research in a school-based obesity prevention program for Native American school children (Pathways). *Health Educ Res* **13**, 251-265.

Goldberg GR, Black AE, Jebb SA, Cole TJ, Murgatroyd PR, Coward WA & Prentice AM (1991a) Critical evaluation of energy intake data using fundamental principles of energy physiology: 1. Derivation of cut-off limits to identify under-recording. *Eur J Clin Nutr* **45**, 569-581.

Goldberg GR, Prentice AM, Coward WA, Davies HL, Murgatroyd PR, Sawyer MB, Ashford J & Black AE (1991b) Longitudinal assessment of the components of energy balance in well-nourished lactating women. *Am J Clin Nutr* **54**, 788-798.

Goldberg GR, Prentice AM, Coward WA, Davies HL, Murgatroyd PR, Wensing C, Black AE, Harding M & Sawyer M (1993) Longitudinal assessment of energy expenditure in pregnancy by the doubly labeled water method. *Am J Clin Nutr* **57**, 494-505.

Goran MI (2005) Estimating energy requirements: regression based prediction equations or multiples of resting metabolic rate. *Public Health Nutr* **8**, 1184-1186.

Goran MI, Beer WH, Wolfe RR, Poehlman ET & Young VR (1993a) Variation in total energy expenditure in young healthy free-living men. *Metabolism* **42**, 487-496.

Goran MI, Calles-Escandon J, Poehlman ET, O'Connell M & Danforth E Jr (1994b) Effects of increased energy intake and/or physical activity on energy expenditure in young healthy men. *J Appl Physiol* **77**, 366-372.

Goran MI, Carpenter WH & Poehlman ET (1993b) Total energy expenditure in 4- to 6-yr-old children. *Am J Physiol* **264**, E706-E711.

Goran MI, Gower BA, Nagy TR & Johnson RK (1998c) Developmental changes in energy expenditure and physical activity in children: evidence for a decline in physical activity in girls before puberty. *Pediatrics* **101**, 887-891.

Goran MI, Kaskoun M & Johnson R (1994a) Determinants of resting energy expenditure in young children. *J Pediatr* **125**, 362-367.

Goran MI, Kaskoun M, Johnson R, Martinez C, Kelly B & Hood V (1995) Energy expenditure and body fat distribution in Mohawk children. *Pediatrics* **95**, 89-95.

Goran MI, Nagy TR, Gower BA, Mazariegos M, Solomons N, Hood V & Johnson R (1998a) Influence of sex, seasonality, ethnicity, and geographic location on the components of total energy expenditure in young children: implications for energy requirements. *Am J Clin Nutr* **68**, 675-682.

Goran MI, Shewchuk R, Gower BA, Nagy TR, Carpenter WH & Johnson RK (1998b) Longitudinal changes in fatness in white children: no effect of childhood energy expenditure. *Am J Clin Nutr* **67**, 309-316.

Gordon-Larsen P, Hou N, Sidney S, Sternfeld B, Lewis CE, Jacobs DR, Jr. & Popkin BM (2009) Fifteen-year longitudinal trends in walking patterns and their impact on weight change. *Am J Clin Nutr* **89**, 19-26.

Goris AH, Westerterp-Plantenga MS & Westerterp KR (2000) Undereating and underrecording of habitual food intake in obese men: selective underreporting of fat intake1. *Am J Clin Nutr* **71**, 130-134.

Grediagin A, Cody M, Rupp J, Benardot D & Shern R (1995) Exercise intensity does not effect body composition change in untrained, moderately overfat women. *J Am Diet Assoc* **95**, 661-665.

Green JH. Asessment of energy requirements (1994). In: *Consensus in Clinical Nutrition*. Ed. Heatley RV, Green JH, Losowsky MS. Cambridge University Press.

Gregory J, Bates CJ, Lowe S, Prentice A, Jackson LV, Smithers G, Wenlock R & Farron M (2000) *National diet and nutrition survey: Young people aged 4 to 18 years: Volume 1: Report of the diet and nutrition survey*, Volume 1 ed. London: The Stationery Office.

Gregory JR, Collins DL, Davies PSW, Hughes JM & Clarke PC (1995) *National Diet and Nutrition Survey: children aged 1½-4½ years. Volume 1: report of the diet and nutrition survey*. London: HMSO.

Gregory J, Foster K, Tyler H & Wiseman M (1990) *The Dietary and Nutritional Survey of British Adults*. London: HMSO.

Guinhouya CB, Hubert H, Soubrier S, Vilhelm C, Lemdani M & Durocher A (2006) Moderate-to-vigorous physical activity among children: discrepancies in accelerometry-based cut-off points. *Obesity (Silver Spring)* **14**, 774-777.

Haas V, Onur S, Paul T, Nutzinger DO, Bosy-Westphal A, Hauer M, Brabant G, Klein H & Muller MJ (2005) Leptin and body weight regulation in patients with anorexia nervosa before and during weight recovery. *Am J Clin Nutr* **81**, 889-896.

Haggarty P, McNeill G, Manneh MK, Davidson L, Milne E, Duncan G & Ashton J (1994) The influence of exercise on the energy requirements of adult males in the UK. *Br J Nutr* **72**, 799-813.

Haggarty P, Valencia ME, McNeill G, Gonzales NL, Moya SY, Pinelli A, Quihui L, Saucedo MS, Esparxa J, Ashton J, Milne E, James WPT (1997) Energy expenditure during heavy work and its interaction with body weight. *Br J Nutr* **77**, 359-373.

Hall MB & Elliman D (2003) Health for all children. 4th Edition. Oxford University Press.

Haman F (2006) Shivering in the cold: from mechanisms of fuel selection to survival. *J Appl Physiol* **100**, 1702-1708.

Han Z, Mulla S, Beyene J, Liao G & McDonald SD (2011) Maternal underweight and the risk of preterm birth and low birth weight: a systematic review and meta-analyses. *Int J Epidemiol* **40**, 65-101.

Hancox RJ, Milne BJ & Poulton R (2004) Association between child and adolescent television viewing and adult health: a longitudinal birth cohort study. *Lancet* **364**, 257-262.

Hancox RJ & Poulton R (2006) Watching television is associated with childhood obesity: but is it clinically important? *Int J Obes (Lond)* **30**, 171-175.

Hansen M, Morthorst R, Larsson B, Flyvbjerg A, Rasmussen MH, Orskov H, Astrup A, Kjaer M & Lange KHW (2005) Effects of 2 wk of GH administration on 24-h indirect calorimetry in young, healthy, lean men. *Am J Physiol Endocrinol Metab* **289**, E1030-E1038.

Hardeman W, Griffin S, Johnston M, Kinmonth AL & Wareham NJ (2000) Interventions to prevent weight gain: a systematic review of psychological models and behaviour change methods. *Int J Obes Relat Metab Disord* **24**, 131-143.

Haslam D, Sattar N & Lean M (2006) Obesity--time to wake up. *BMJ* **333**, 640-642.

Heal DJ, Aspley S, Prow MR, Jackson HC, Martin KF & Cheetham SC (1998) Sibutramine: a novel anti-obesity drug. A review of the pharmacological evidence to differentiate it from d-amphetamine and d-fenfluramine. *Int J Obes Relat Metab Disord* **22 Suppl 1**, S18-S28.

Henderson L, Gregory J, Irving K & Swan G (2003a) *The National Diet and Nutrition Survey: adults aged 19 to 64 years. Volume 2: Energy, protein, carbohydrate, fat and alcohol intake*. London: TSO.

Henderson L, Gregory J & Swan G (2002) *The National Diet and Nutrition Survey: adults aged 19 to 64 years. Volume 1: Types and quantities of foods consumed*. London: TSO.

Henderson L, Irving K, Gregory J, Bates CJ, Prentice A, Perks J, Swan G & Farron M (2003b) *The National Diet and Nutrition Survey: adults aged 19 to 64 years. Volume 3: Vitamin and mineral intake and urinary analytes*. London: TSO.

Henry CJ (2005) Basal metabolic rate studies in humans: measurement and development of new equations. *Public Health Nutr* **8**, 1133-1152.

Hernandez-Triana M, Salazar G, Diaz E, Sanchez V, Basabe B, Gonzalez S & Diaz ME (2002) Total energy expenditure by the doubly-labeled water method in rural preschool children in Cuba. *Food Nutr Bull* **23**, 76-81.

Heslehurst N, Ells LJ, Simpson H, Batterham A, Wilkinson J, Summerbell CD (2007) Trends in maternal obesity incidence rates, demographic predictors, and health inequalities in 36,821 women over a 15-year period. *BJOG* **114**(2):187-194.

Hessemer V & Bruck K (1985) Influence of menstrual cycle on thermoregulatory, metabolic, and heart rate responses to exercise at night. *J Appl Physiol* **59**, 1911-1917.

Heymsfield SB, Gallagher D, Kotler DP, Wang Z, Allison DB & Heshka S (2002) Body-size dependence of resting energy expenditure can be attributed to nonenergetic homogeneity of fat-free mass. *Am J Physiol Endocrinol Metab* **282**, E132-E138.

Hill JO (2006) Understanding and Addressing the Epidemic of Obesity: An Energy Balance Perspective. *Endocr Rev* **27**, 750-761.

Hill JO (2009) Can a small-changes approach help address the obesity epidemic? A report of the Joint Task Force of the American Society Nutrition, Institute of Food Technologists, and International Food Information Council. *Am J Clin Nutr* **89**, 477-484.

Hill JO, Wyatt HR, Reed GW & Peters JC (2003) Obesity and the Environment: Where Do We Go from Here? *Science* **299**, 853-855.

Hinney A, Nguyen TT, Scherag A*, et al.* (2007) Genome wide association (GWA) study for early onset extreme obesity supports the role of fat mass and obesity associated gene (FTO) variants. *PLoS ONE* **2**, e1361.

Hoare J, Henderson L, Bates CJ, Prentice A, Birch M, Swan G & Farron M (2004) *The National Diet and Nutrition Survey: adults aged 19 to 64 years. Volume 5: Summary report*. London: TSO.

Hoffman DJ, Sawaya AL, Coward WA, Wright A, Martins PA, de Nascimento C, Tucker KL & Roberts SB (2000) Energy expenditure of stunted and nonstunted boys and girls living in the shantytowns of Sao Paulo, Brazil. *Am J Clin Nutr* **72**, 1031.

Hojlund K, Christiansen C, Bjornsbo KS, Poulsen P, Bathum L, Henriksen JE, Lammert O & Beck-Nielsen H (2006) Energy expenditure, body composition and insulin response to glucose in male twins discordant for the Trp64Arg polymorphism of the beta3-adrenergic receptor gene. *Diabetes Obes Metab* **8**, 322-330.

Hoos MB, Plasqui G, Gerver WJ & Westerterp KR (2003) Physical activity level measured by doubly labeled water and accelerometry in children. *Eur J Appl Physiol* **89**, 624-626.

Horn OK, Paradis G, Potvin L, Macaulay AC & Desrosiers S (2001) Correlates and predictors of adiposity among Mohawk children. *Prev Med* **33**, 274-281.

Howe JC, Rumpler WV & Seale JL (1993) Energy expenditure by indirect calorimetry in premenopausal women: variation within one menstrual cycle. *Journal of Nutritional Biochemistry* **4**, 268-273.

Hoyt RW & Friedl KE (2006) Field studies of exercise and food deprivation. *Curr Opin Clin Nutr Metab Care* **9**, 685-690.

Hu FB, Li TY, Colditz GA, Willett WC & Manson JE (2003) Television watching and other sedentary behaviors in relation to risk of obesity and type 2 diabetes mellitus in women. *JAMA* **289**, 1785-1791.

Hukshorn CJ, Menheere PP, Westerterp-Plantenga MS & Saris WH (2003a) The effect of pegylated human recombinant leptin (PEG-OB) on neuroendocrine adaptations to semi-starvation in overweight men. *Eur J Endocrinol* **148**, 649-655.

Hukshorn CJ, Saris WHM, Westerterp-Plantenga MS, Farid AR, Smith FJ & Campfield LA (2000) Weekly Subcutaneous Pegylated Recombinant Native Human Leptin (PEG-OB) Administration in Obese Men. *J Clin Endocrinol Metab* **85**, 4003-4009.

Hukshorn CJ, Westerterp-Plantenga MS & Saris WH (2003b) Pegylated human recombinant leptin (PEG-OB) causes additional weight loss in severely energy-restricted, overweight men. *Am J Clin Nutr* **77**, 771-776.

Hunt SC, Stone S, Xin Y, Scherer CA, Magness CL, Iadonato SP, Hopkins PN & Adams TD (2008) Association of the FTO gene with BMI. *Obesity (Silver Spring)* **16**, 902-904.

Hunter GR, Weinsier RL, Darnell BE, Zuckerman PA, Goran MI. Racial differences in energy expenditure and aerobic fitness in premenopausal women1. Am J Clin Nutr 2000;71(2):500-506.

Hytten FE & Chamberlain G (1991) *Clinical physiology in obstetrics.* Oxford, United Kingdom: Blackwell Scientific Publications.

IAEA (2009) *Assessment of body composition and total energy expenditure in humans using stable isotope techniques*. Vienna: International Atomic Energy Agency.

Illner K, Brinkmann G, Heller M, Bosy-Westphal A & Muller MJ (2000) Metabolically active components of fat free mass and resting energy expenditure in nonobese adults. *Am J Physiol Endocrinol Metab* **278**, E308-E315.

Institute of Medicine Food and Nutrition Board (1990) *Nutrition During Pregnancy: Part I: Weight Gain, Part II: Nutrient Supplements*. Washington DC: National Academy Press.

Institute of Medicine (2005) *Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids*. Washington, D.C.: National Academy Press.

Institute of Medicine Food and Nutrition Board (1990) *Nutrition During Pregnancy: Part I: Weight Gain, Part II: Nutrient Supplements*. Washington DC: National Academy Press.

Jago R, Baranowski T, Baranowski JC, Thompson D & Greaves KA (2005) BMI from 3-6 y of age is predicted by TV viewing and physical activity, not diet. *Int J Obes (Lond)* **29**, 557-564.

Jakicic JM & Wing RR (1998) Differences in resting energy expenditure in African-American vs Caucasian overweight females. *Int J Obes Relat Metab Disord* **22**, 236-242.

Jebb SA (2007) Dietary determinants of obesity. *Obes Rev* **8 Suppl 1**, 93-97.

Jeffery RW & French SA (1997) Preventing weight gain in adults: design, methods and one year results from the Pound of Prevention study. *Int J Obes Relat Metab Disord* **21**, 457-464.

Jessen AB, Toubro S & Astrup A (2003) Effect of chewing gum containing nicotine and caffeine on energy expenditure and substrate utilization in men. *Am J Clin Nutr* **77**, 1442-1447.

Jiang Z, Yan Q, Su Y, Acheson KJ, Thelin A, Piguet-Welsch C, Ritz P & Ho ZC (1998) Energy expenditure of Chinese infants in Guangdong Province, south China, determined with use of the doubly labeled water method. *Am J Clin Nutr* **67**, 1256-1264.

Johnson MS, Figueroa-Colon R, Herd SL, Fields DA, Sun M, Hunter GR & Goran MI (2000) Aerobic Fitness, Not Energy Expenditure, Influences Subsequent Increase in Adiposity in Black and White Children. *Pediatrics* **106**, e50.

Johnson RK, Russ J & Goran MI (1998) Physical activity related energy expenditure in children by doubly labeled water as compared with the Caltrac accelerometer. *Int J Obes Relat Metab Disord* **22**, 1046-1052.

Johnstone AM, Murison SD, Duncan JS, Rance KA & Speakman JR (2005) Factors influencing variation in basal metabolic rate include fat-free mass, fat mass, age, and circulating thyroxine but not sex, circulating leptin, or triiodothyronine. *Am J Clin Nutr* **82**, 941-948.

Jones A, Jr., Shen W, St Onge MP, Gallagher D, Heshka S, Wang Z & Heymsfield SB (2004) Body-composition differences between African American and white women: relation to resting energy requirements. *Am J Clin Nutr* **79**, 780-786.

Jorgensen JO, Vahl N, Dall R & Christiansen JS (1998) Resting metabolic rate in healthy adults: relation to growth hormone status and leptin levels. *Metabolism* **47**, 1134-1139.

Jung RT, Shetty PS & James WP (1980) The effect of beta-adrenergic blockade on metabolic rate and peripheral thyroid metabolism in obesity. *Eur J Clin Invest* **10**, 179-182.

Kain J, Uauy R, Albala, Vio F, Cerda R & Leyton B (2004) School-based obesity prevention in Chilean primary school children: methodology and evaluation of a controlled study. *Int J Obes Relat Metab Disord* **28**, 483-493.

Kaplan AS, Zemel BS & Stallings VA (1996) Differences in resting energy expenditure in prepubertal black children and white children. *J Pediatr* **129**, 643-647.

Karhunen L, Franssila-Kallunki A, Rissanen A, Kervinen K, Kesaniemi YA & Uusitupa M (1997) Determinants of resting energy expenditure in obese non-diabetic caucasian women. *Int J Obes Relat Metab Disord* **21**, 197-202.

Kaskoun MC, Johnson RK & Goran MI (1994) Comparison of energy intake by semiquantitative food-frequency questionnaire with total energy expenditure by the doubly labeled water method in young children. *Am J Clin Nutr* **60**, 43-47.

Kautiainen S, Koivusilta L, Lintonen T, Virtanen SM & Rimpela A (2005) Use of information and communication technology and prevalence of overweight and obesity among adolescents. *Int J Obes (Lond)* **29**, 925-933.

Kennedy A, Gettys TW, Watson P, Wallace P, Ganaway E, Pan Q & Garvey WT (1997) The metabolic significance of leptin in humans: gender-based differences in relationship to adiposity, insulin sensitivity, and energy expenditure. *J Clin Endocrinol Metab* **82**, 1293-1300.

Key TJ, Schatzkin A, Willett WC, Allen NE, Spencer EA & Travis RC (2004) Diet, nutrition and the prevention of cancer. *Public Health Nutr* **7**, 187-200.

Keys A, Fidanza F, Karvonen MJ, Kimura N & Taylor HL (1972) Indices of relative weight and obesity. *J Chronic Dis* **25**, 329-343.

Kim CH, Yun SK, Byun DW, Yoo MH, Lee KU, Suh KI (2001) Codon 54 polymorphism of the fatty acid binding protein 2 gene is associated with increased fat oxidation and hyperinsulinemia, but not with intestinal fatty acid absorption in Korean men. Metabolism **50**(4), 473-476.

Kim J, Wang Z, Heymsfield SB, Baumgartner RN & Gallagher D (2002) Total-body skeletal muscle mass: estimation by a new dual-energy X-ray absorptiometry method. *Am J Clin Nutr* **76**, 378-383.

Kimm SY, Barton BA, Obarzanek E, McMahon RP, Sabry ZI, Waclawiw MA, Schreiber GB, Morrison JA, Similo S & Daniels SR (2001) Racial divergence in adiposity during adolescence: The NHLBI Growth and Health Study. *Pediatrics* **107**, E34.

Kimm SY, Glynn NW, Obarzanek E, Kriska AM, Daniels SR, Barton BA & Liu K (2005) Relation between the changes in physical activity and body-mass index during adolescence: a multicentre longitudinal study. *Lancet* **366**, 301-307.

King NA, Appleton K, Rogers PJ & Blundell JE (1999) Effects of sweetness and energy in drinks on food intake following exercise. *Physiol Behav* **66**, 375-379.

King NA, Caudwell P, Hopkins M, Byrne NM, Colley R, Hills AP, Stubbs JR & Blundell JE (2007) Metabolic and Behavioral Compensatory Responses to Exercise Interventions: Barriers to Weight Loss. *Obesity Res* **15**, 1373-1383.

Kipnis V, Subar AF & Midthune D (2003) Structure of dietary measurement error: results of the OPEN biomarker study. *Am J Epidemiol* **158**, 14-21.

Kirkby J, Metcalf BS, Jeffery AN, O'Riordan CF, Perkins J, Voss LD & Wilkin TJ (2004) Sex differences in resting energy expenditure and their relation to insulin resistance in children (EarlyBird 13). *Am J Clin Nutr* **80**, 430-435.

Klannemark M, Orho M & Groop L (1998) No relationship between identified variants in the uncoupling protein 2 gene and energy expenditure. *Eur J Endocrinol* **139**, 217-223.

Klausen B, Toubro S & Astrup A (1997) Age and sex effects on energy expenditure. *Am J Clin Nutr* **65**, 895-907.

Kleiber M (1975) *The Fire of Life. An Introduction to Animal Energetics*. New York: Robert E. Krieger Publishing.

Klesges RC, Klesges LM, Eck LH & Shelton ML (1995) A longitudinal analysis of accelerated weight gain in preschool children. *Pediatrics* **95**, 126-130.

Koh-Banerjee P, Chu NF, Spiegelman D, Rosner B, Colditz G, Willett W & Rimm E (2003) Prospective study of the association of changes in dietary intake, physical activity, alcohol consumption, and smoking with 9-y gain in waist circumference among 16 587 US men. *Am J Clin Nutr* **78**, 719-727.

Kopp-Hoolihan LE, van Loan MD, Wong WW & King JC (1999) Longitudinal assessment of energy balance in well-nourished, pregnant women. *Am J Clin Nutr* **69**, 697-704.

Krakoff J, Ma L, Kobes S et al (2008) Lower Metabolic Rate in Individuals Heterozygous for Either a Frameshift or a Functional Missense MC4R Variant. *Diabetes* **57**(12), 3267-3272.

Kramer MS & Kakuma R (2003) Energy and protein intake in pregnancy (Review). *Cochrane Database of Systematic Reviews*, Art. No.:CD000032. DOI: 10.1002/14651858/CD000032.

Kring SII, Holst C, Zimmermann E, Jess T, Berentzen T, Toubro S, ansen T, strup A, edersen O & orensen T (2008) FTO Gene Associated Fatness in Relation to Body Fat Distribution and Metabolic Traits throughout a Broad Range of Fatness. *PLoS ONE* **3**, e2958.

Kubaszek A, Pihlajamaki J, Punnonen K, Karhapaa P, Vauhkonen I, Laakso M (2003) The C-174G promoter polymorphism of the IL-6 gene affects energy expenditure and insulin sensitivity. *Diabetes* **52**(2), 558-561.

Kuip M, Hoos MB, Forget PP, Westerterp KR, Gemke RJ & Meer KD (2003) Energy expenditure in infants with congenital heart disease, including a meta-analysis. *Acta Paediatrica* **92**, 921-927.

Kunz I, Klaus S, Kallies B, Schorr U & Sharma AM (2000) Kinetic analysis of the thermic effect of food and its relationship to body composition in humans. *Metabolism* **49**, 1340-1345.

Kushner RF, Racette SB, Neil K & Schoeller DA (1995) Measurement of physical activity among black and white obese women. *Obes Res* **3 Suppl 2**, 261s-265s.

Kvaavik E, Tell GS & Klepp KI (2003) Predictors and tracking of body mass index from adolescence into adulthood: follow-up of 18 to 20 years in the Oslo Youth Study. *Arch Pediatr Adolesc Med* **157**, 1212-1218.

Kyle UG, Genton L, Hans D, Karsegard L, Slosman DO & Pichard C (2001) Age-related differences in fat-free mass, skeletal muscle, body cell mass and fat mass between 18 and 94 years. *Eur J Clin Nutr* **55**, 663-672.

Lariviere F, Moussalli R & Garrel DR (1994) Increased leucine flux and leucine oxidation during the luteal phase of the menstrual cycle in women. *Am J Physiol Endocrinol Metab* **267**, E422-E428.

Lawrence M, Thongprasert K & Durnin JV (1988) Between-group differences in basal metabolic rates: an analysis of data collected in Scotland, the Gambia and Thailand. *Eur J Clin Nutr* **42**, 877-891.

Lean ME, Murgatroyd PR, Rothnie I, Reid IW & Harvey R (1988) Metabolic and thyroidal responses to mild cold are abnormal in obese diabetic women. *Clin Endocrinol (Oxf)* **28**, 665-673.

Leenders NY, Sherman WM, Nagaraja HN & Kien CL (2001) Evaluation of methods to assess physical activity in free-living conditions. *Med Sci Sports Exerc* **33**, 1233-1240.

Lemura LM & Maziekas MT (2002) Factors that alter body fat, body mass, and fat-free mass in pediatric obesity. *Med Sci Sports Exerc* **34**, 487-496.

Levine JA (2005) Measurement of energy expenditure. *Public Health Nutr* **8**, 1123-1132.

Levine JA (2007) Nonexercise activity thermogenesis-liberating the life-force. *J Intern Med* **262**, 273-287.

Levine JA, Eberhardt NL & Jensen MD (1999) Role of nonexercise activity thermogenesis in resistance to fat gain in humans. *Science* **283**, 212-214.

Levine JA, Harris MM & Morgan MY (2000) Energy expenditure in chronic alcohol abuse. *Eur J Clin Invest* **30**, 779-786.

Levine JA (2004) Nonexercise activity thermogenesis (NEAT): environment and biology. *Am J Physiol Endocrinol Metab* **286**, E675-E685.

Li ET, Tsang LB & Lui SS (1999) Resting metabolic rate and thermic effects of a sucrose-sweetened soft drink during the menstrual cycle in young Chinese women. *Can J Physiol Pharmacol* **77**, 544-550.

Lichtman SW, Pisarska K, Berman ER, Pestone M, Dowling H, Offenbacher E, Weisel H, Heshka S, Matthews DE & Heymsfield SB (1992) Discrepancy between self-reported and actual caloric intake and exercise in obese subjects. *N Engl J Med* **327**, 1893-1898.

Lindquist CH, Cummings T & Goran MI (2000) Use of tape-recorded food records in assessing children's dietary intake. *Obes Res* **8**, 2-11.

Littrell KH, Hilligoss NM, Kirshner CD, Petty RG & Johnson CG (2003) The effects of an educational intervention on antipsychotic-induced weight gain. *J Nurs Scholarsh* **35**, 237-241.

Livingstone MB (1995) Assessment of food intakes: are we measuring what people eat? *Br J Biomed Sci* **52**, 58-67.

Livingstone MB & Black AE (2003) Markers of the validity of reported energy intake. *J Nutr* **133 Suppl 3**, 895S-920S.

Livingstone MB, Prentice AM, Coward WA, Strain JJ, Black AE, Davies PS, Stewart CM, McKenna PG & Whitehead RG (1992) Validation of estimates of energy intake by weighed dietary record and diet history in children and adolescents. *Am J Clin Nutr* **56**, 29-35.

Long SJ, Hart K & Morgan LM (2002) The ability of habitual exercise to influence appetite and food intake in response to high- and low-energy preloads in man. *Br J Nutr* **87**, 517-523.

Loos RJF, Rankinen T, Chagnon Y, Tremblay A, Perusse L & Bouchard C (2006) Polymorphisms in the leptin and leptin receptor genes in relation to resting metabolic rate and respiratory quotient in the Quebec Family Study. *Int J Obes Relat Metab Disord* **30**, 183-190.

Lopez-Alarcon M, Merrifield J, Fields DA, Hilario-Hailey T, Franklin FA, Shewchuk RM, Oster RA & Gower BA (2004) Ability of the actiwatch accelerometer to predict free-living energy expenditure in young children. *Obes Res* **12**, 1859-1865.

Lovejoy JC, Champagne CM, Smith SR, de Jonge L & Xie H (2001) Ethnic differences in dietary intakes, physical activity, and energy expenditure in middle-aged, premenopausal women: the Healthy Transitions Study. *Am J Clin Nutr* **74**, 90-95.

Lovelady CA, Meredith CN, McCrory MA, Nommsen LA, Joseph LJ & Dewey KG (1993) Energy expenditure in lactating women: a comparison of doubly labeled water and heart-rate-monitoring methods. *Am J Clin Nutr* **57**, 512-518.

Macdonald HM, New SA, Campbell MK & Reid DM (2003) Longitudinal changes in weight in perimenopausal and early postmenopausal women: effects of dietary energy intake, energy expenditure, dietary calcium intake and hormone replacement therapy. *Int J Obes Relat Metab Disord* **27**, 669-676.

Mackintosh RM & Hirsch J (2001) The Effects of Leptin Administration in Non-obese Human Subjects. *Obesity Res* **9**, 462-469.

Maes HH, Neale MC & Eaves LJ (1997) Genetic and environmental factors in relative body weight and human adiposity. *Behav Genet* **27**, 325-351.

Maestrini S, Podesta F, Di Blasio AM, Savia G, Brunani A, Tagliaferri A, Mencarelli M, Chiodini I & Liuzzi A (2003) Lack of association between UCP2 gene polymorphisms and obesity phenotype in Italian Caucasians. *J Endocrinol Invest* **26**, 985-990.

Maffeis C, Talamini G, Tato L (1998) Influence of diet, physical activity and parents' obesity on children's adiposity: a four-year longitudinal study. *Int J Obes Relat Metab Disord* **22**(8), 758-764.

Magarey AM, Daniels LA, Boulton TJ & Cockington RA (2003) Predicting obesity in early adulthood from childhood and parental obesity. *Int J Obes Relat Metab Disord* **27**, 505-513.

Magoffin A, Allen JR, McCauley J, Gruca MA, Peat J, Van Asperen P & Gaskin K (2008) Longitudinal analysis of resting energy expenditure in patients with cystic fibrosis. *J Pediatr* **152**, 703-708.

Mamalakis G, Kafatos A, Manios Y, Anagnostopoulou T & Apostolaki I (2000) Obesity indicies in a cohort of primary school children in Crete: a six year prospective study. *International Journal of Obesity and Related Metabolic Disorders* **24**, 765-771.

Manini TM, Everhart JE, Patel KV, Schoeller DA, Colbert LH, Visser M, Tylavsky F, Bauer DC, Goodpaster BH & Harris TB (2006) Daily Activity Energy Expenditure and Mortality Among Older Adults. *JAMA* **296**, 171-179.

Mansell PI & Macdonald IA (1990) Reappraisal of the Weir equation for calculation of metabolic rate. *Am J Physiol* **258**(6 Pt 2), R1347-54.

March of Dimes (2002) *Nutrition Today Matters Tomorrow. A Report from The March of Dimes Task Force on Nutrition and Optimal Human Development.*: March of Dimes Fulfillment Center, Pennsylvania.

Marken Lichtenbelt WD, Heidendal GA & Westerterp KR (1997) Energy expenditure and physical activity in relation to bone mineral density in women with anorexia nervosa. *Eur J Clin Nutr* **51**, 826-830.

Marshall SJ, Biddle SJ, Gorely T, Cameron N & Murdey I (2004) Relationships between media use, body fatness and physical activity in children and youth: a meta-analysis. *Int J Obes Relat Metab Disord* **28**, 1238-1246.

Martins C, Morgan L & Truby H (2008a) A review of the effects of exercise on appetite regulation: an obesity perspective. *Int J Obes (Lond)* **32**, 1337-1347.

Martins C, Robertson MD & Morgan LM (2008b) Effects of exercise and restrained eating behaviour on appetite control. *Proceedings of the Nutrition Society* **67**, 28-41.

Martins C, Truby H & Morgan LM (2007) Short-term appetite control in response to a 6-week exercise programme in sedentary volunteers. *Br J Nutr* **98**, 834-842.

Mâsse LC, Fulton JE, Watson KL, Mahar MT, Meyers MC & Wong WW (2004) Influence of body composition on physical activity validation studies using doubly labeled water. *J Appl Physiol* **96**, 1357-1364.

Mattocks C, Leary S, Ness A, Deere K, Saunders J, Kirkby J, Blair SN, Tilling K & Riddoch C (2007) Intraindividual variation of objectively measured physical activity in children. *Med Sci Sports Exerc* **39**, 622-629.

Mayer J (1966) Some aspects of the problem of regulation of food intake and obesity. *N Engl J Med* **274**, 722-731.

Mayer J & Thomas DW (1967) Regulation of food intake and obesity. *Science* **156**, 328-337.

McDonald SD, Han Z, Mulla S & Beyene J (2010) Overweight and obesity in mothers and risk of preterm birth and low birth weight infants: systematic review and meta-analyses. *BMJ* **341**, c3428.

McGloin AF, Livingstone MB, Greene LC, Webb SE, Gibson JM, Jebb SA, Cole TJ, Coward WA, Wright A & Prentice AM (2002) Energy and fat intake in obese and lean children at varying risk of obesity. *Int J Obes Relat Metab Disord* **26**, 200-207.

McMurray RG, Harrell JS, Bangdiwala SI, Bradley CB, Deng S, Levine A (2002) A school-based intervention can reduce body fat and blood pressure in young adolescents. *J Adolesc Health* **31**(2), 125-132.

Meijer GA, Westerterp KR, Saris WH & Ten Hoor F (1992) Sleeping metabolic rate in relation to body composition and the menstrual cycle. *Am J Clin Nutr* **55**, 637-640.

Melanson KJ, Saltzman E, Russell R & Roberts SB (1996) Postabsorptive and postprandial energy expenditure and substrate oxidation do not change during the menstrual cycle in young women. *J Nutr* **126**, 2531-2538.

Melanson KJ, Saltzman E, Vinken AG, Russell R & Roberts SB (1998) The effects of age on postprandial thermogenesis at four graded energetic challenges: findings in young and older women. *J Gerontol A Biol Sci Med Sci* **53**, B409-B414.

Melzer K, Kayser B, Saris WH & Pichard C (2005) Effects of physical activity on food intake. pp. 885-895.

Miller WC, Koceja DM & Hamilton EJ (1997) A meta-analysis of the past 25 years of weight loss research using diet, exercise or diet plus exercise intervention. *Int J Obes Relat Metab Disord* **21**, 941-947.

Molnar D & Livingstone B (2000) Physical activity in relation to overweight and obesity in children and adolescents. *Eur J Pediatr* **159 Suppl 1**, S45-S55.

Montgomery C, Reilly JJ, Jackson DM, Kelly LA, Slater C, Paton JY & Grant S (2005) Validation of energy intake by 24-hour multiple pass recall: comparison with total energy expenditure in children aged 5-7 years. *Br J Nutr* **93**, 671-676.

Moore LL, Gao D, Bradlee ML, Cupples LA, Sundarajan-Ramamurti A, Proctor MH, Hood MY, Singer MR & Ellison RC (2003) Does early physical activity predict body fat change throughout childhood? *Prev Med* **37**, 10-17.

Moore LL, Nguyen US, Rothman KJ, Cupples LA & Ellison RC (1995) Preschool Physical Activity Level and Change in Body Fatness in Young Children: The Framingham Children's Study. *Am J Epidemiol* **142**, 982-988.

Morrison JA, Alfaro MP, Khoury P, Thornton BB & Daniels SR (1996) Determinants of resting energy expenditure in young black girls and young white girls. *J Pediatr* **129**, 637-642.

Moshfegh AJ, Rhodes DG, Baer DJ*, et al.* (2008) The US Department of Agriculture Automated Multiple-Pass Method reduces bias in the collection of energy intakes. *Am J Clin Nutr* **88**, 324-332.

Muller MJ, Bosy-Westphal A, Klaus S*, et al.* (2004) World Health Organization equations have shortcomings for predicting resting energy expenditure in persons from a modern, affluent population: generation of a new reference standard from a retrospective analysis of a German database of resting energy expenditure. *Am J Clin Nutr* **80**, 1379-1390.

Muller MJ, Burger AG, Ferrannini E, Jequier E & Acheson KJ (1989) Glucoregulatory function of thyroid hormones: role of pancreatic hormones. *Am J Physiol* **256**, E101-E110.

Mundt CA, Baxter-Jones AD, Whiting SJ, Bailey DA, Faulkner RA & Mirwald RL (2006) Relationships of activity and sugar drink intake on fat mass development in youths. *Med Sci Sports Exerc* **38**, 1245-1254.

Murgatroyd PR, Goldberg GR, Leahy FE, Gilsenan MB & Prentice AM (1999) Effects of inactivity and diet composition on human energy balance. *Int J Obes Relat Metab Disord* **23**, 1269-1275.

Must A, Bandini LG, Tybor DJ, Phillips SM, Naumova EN, Dietz WH (2007) Activity, inactivity, and screen time in relation to weight and fatness over adolescence in girls. *Obesity* (Silver Spring) **15**(7), 1774-1781.

Must A & Tybor DJ (2005) Physical activity and sedentary behavior: a review of longitudinal studies of weight and adiposity in youth. *Int J Obes (Lond)* **29 Suppl 2**, S84-S96.

Muto T, Yamauchi K (2001) Evaluation of a multicomponent workplace health promotion program conducted in Japan for improving employees' cardiovascular disease risk factors. *Prev Med* **33**(6), 571-577.

Nagai N, Sakane N, Ueno LM, Hamada T & Moritani T (2003) The -3826 A-->G variant of the uncoupling protein-1 gene diminishes postprandial thermogenesis after a high fat meal in healthy boys. *J Clin Endocrinol Metab* **88**, 5661-5667.

Nagy TR, Gower BA, Shewchuk RM & Goran MI (1997) Serum Leptin and Energy Expenditure in Children. *J Clin Endocrinol Metab* **82**, 4149-4153.

NAHRES-4 (1990) *The Doubly-labelled Water Method for Measuring Energy Expenditure: A consensus Report by the IDECG working group*. Vienna: IAEA.

National Health Service (NHS) Information Centre (2010) *Health Survey for England - 2009 trend tables*. The NHS Information Centre for health and social care.

Nelson KM, Weinsier RL, Long CL & Schutz Y (1992) Prediction of resting energy expenditure from fat-free mass and fat mass. *Am J Clin Nutr* **56**, 848-856.

Nelson MC, Gordon-Larsen P, Adair LS & Popkin BM (2005) Adolescent physical activity and sedentary behavior: patterning and long-term maintenance. *Am J Prev Med* **28**, 259-266.

Nelson MJ, Erens B, Bates B, Church S & Boshier T (2007) *Low income diet and nutrition survey*. London: TSO.

Neuhauser-Berthold M, Herbert BM, Luhrmann PM, Sultemeier AA, Blum WF, Frey J & Hebebrand J (2000) Resting metabolic rate, body composition, and serum leptin concentrations in a free-living elderly population. *Eur J Endocrinol* **142**, 486-492.

Neumark-Sztainer D, Story M, Hannan PJ & Rex J (2003) New Moves: a school-based obesity prevention program for adolescent girls. *Prev Med* **37**, 41-51.

NICE (2006) Obesity: the prevention, identification, assessment and management of overweight and obesity in adults and children.

NICE (2010) Weight management before, during and after pregnancy.

Nicklas BJ, Toth MJ, Goldberg AP & Poehlman ET (1997) Racial Differences in Plasma Leptin Concentrations in Obese Postmenopausal Women. *J Clin Endocrinol Metab* **82**, 315-317.

Norman RA, Tataranni PA, Pratley R et al (1998) Autosomal genomic scan for loci linked to obesity and energy metabolism in Pima Indians. *Am J Hum Genet* **62**(3):659-668.

Northern Ireland Statistics and Research Agency (2006) Health and Social Wellbeing Survey. Topline results 2005/06.

O'Connor J, Ball EJ, Steinbeck KS, Davies PS, Wishart C, Gaskin KJ & Baur LA (2001) Comparison of total energy expenditure and energy intake in children aged 6-9 y. *Am J Clin Nutr* **74**, 643-649.

O'Loughlin J, Gray-Donald K, Paradis G & Meshefedjian G (2000) One- and two-year predictors of excess weight gain among elementary schoolchildren in multiethnic, low-income, inner-city neighborhoods. *Am J Epidemiol* **152**, 739-746.

O'Rahilly S & Farooqi IS (2006) Genetics of obesity. *Philos Trans R Soc Lond B Biol Sci* **361**, 1095-1105.

Obarzanek E, Lesem MD & Jimerson DC (1994) Resting metabolic rate of anorexia nervosa patients during weight gain. *Am J Clin Nutr* **60**, 666-675.

Onur S, Haas V, Bosy-Westphal A, Hauer M, Paul T, Nutzinger D, Klein H & Muller MJ (2005) L-Tri-iodothyronine is a major determinant of resting energy expenditure in underweight patients with anorexia nervosa and during weight gain. *Eur J Endocrinol* **152**, 179-184.

Oomen JM, Waijers PM, van Rossum C, Hoebee B, Saris WH & van Baak MA (2005) Influence of beta(2)-adrenoceptor gene polymorphisms on diet-induced thermogenesis. *Br J Nutr* **94**, 647-654.

Oppert JM, Vohl MC, Chagnon M, Dionne FT, Cassard-Doulcier AM, Ricquier D, Perusse L & Bouchard C (1994) DNA polymorphism in the uncoupling protein (UCP) gene and human body fat. *Int J Obes Relat Metab Disord* **18**, 526-531.

Pangrazi RP, Beighle A, Vehige T & Vack C (2003) Impact of Promoting Lifestyle Activity for Youth (PLAY) on children's physical activity. *J Sch Health* **73**, 317-321.

Pannemans DL & Westerterp KR (1995) Energy expenditure, physical activity and basal metabolic rate of elderly subjects. *Br J Nutr* **73**, 571-581.

Parsons TJ, Manor O & Power C (2006) Physical activity and change in body mass index from adolescence to mid-adulthood in the 1958 British cohort. *Int J Epidemiol* **35**, 197-204.

Parsons TJ, Manor O & Power C (2008) Television viewing and obesity: a prospective study in the 1958 British birth cohort. *Eur J Clin Nutr* **62**, 1355-1363.

Pauly RP, Lear SA, Hastings FC & Birmingham CL (2000) Resting energy expenditure and plasma leptin levels in anorexia nervosa during acute refeeding. *Int J Eat Disord* **28**, 231-234.

Pelkman CL, Chow M, Heinbach RA & Rolls BJ (2001) Short-term effects of a progestational contraceptive drug on food intake, resting energy expenditure, and body weight in young women. *Am J Clin Nutr* **73**, 19-26.

Perkins KA (1992) Metabolic effects of cigarette smoking. *J Appl Physiol* **72**, 401-409.

Perkins KA, Sexton JE & DiMarco A (1996) Acute thermogenic effects of nicotine and alcohol in healthy male and female smokers. *Physiol Behav* **60**, 305-309.

Perkins KA, Sexton JE, Epstein LH, DiMarco A, Fonte C, Stiller RL, Scierka A & Jacob RG (1994) Acute thermogenic effects of nicotine combined with caffeine during light physical activity in male and female smokers. *Am J Clin Nutr* **60**, 312-319.

Perks SM, Roemmich JN, Sandow-Pajewski M, Clark PA, Thomas E, Weltman A, Patrie J & Rogol AD (2000) Alterations in growth and body composition during puberty. IV. Energy intake estimated by the youth-adolescent food-frequency questionnaire: validation by the doubly labeled water method. *Am J Clin Nutr* **72**, 1455-1460.

Petersen L, Schnohr P & Sorensen TI (2004) Longitudinal study of the long-term relation between physical activity and obesity in adults. *Int J Obes Relat Metab Disord* **28**, 105-112.

Piers LS, Diggavi SN, Rijskamp J, van Raaij JM, Shetty PS & Hautvast JG (1995) Resting metabolic rate and thermic effect of a meal in the follicular and luteal phases of the menstrual cycle in well-nourished Indian women. *Am J Clin Nutr* **61**, 296-302.

Piers LS, Soares MJ, McCormack LM & O'Dea K (1998) Is there evidence for an age-related reduction in metabolic rate? *J Appl Physiol* **85**, 2196-2204.

Pietilainen KH, Kaprio J, Borg P, Plasqui G, Yki-Jarvinen H, Kujala UM, Rose RJ, Westerterp KR & Rissanen A (2008) Physical Inactivity and Obesity: A Vicious Circle. *Obesity (Silver Spring)* **16**, 409-414.

Pietrobelli A, Faith MS, Allison DB, Gallagher D, Chiumello G, Heymsfield SB (1998) Body mass index as a measure of adiposity among children and adolescents: a validation study. *J Pediatr* **132**(2), 204-210.

Plasqui G, Joosen AM, Kester AD, Goris AH & Westerterp KR (2005) Measuring free-living energy expenditure and physical activity with triaxial accelerometry. *Obes Res* **13**, 1363-1369.

Plasqui G, Kester ADM & Westerterp KR (2003) Seasonal variation in sleeping metabolic rate, thyroid activity, and leptin. *Am J Physiol Endocrinol Metab* **285**, E338-E343.

Plasqui G & Westerterp KR (2004) Seasonal Variation in Total Energy Expenditure and Physical Activity in Dutch Young Adults. *Obesity Res* **12**, 688-694.

Poehlman ET, Melby CL & Badylak SF (1991) Relation of age and physical exercise status on metabolic rate in younger and older healthy men. *J Gerontol* **46**, B54-B58.

Poehlman ET, Toth MJ, Ades PA & Calles-Escandon J (1997) Gender differences in resting metabolic rate and noradrenaline kinetics in older individuals. *Eur J Clin Invest* **27**, 23-28.

Polley BA, Wing RR & Sims CJ (2002) Randomized controlled trial to prevent excessive weight gain in pregnant women. *Int J Obes Relat Metab Disord* **26**, 1494-1502.

Poppitt SD, Swann D, Black AE, Prentice AM (1998) Assessment of selective under-reporting of food intake by both obese and non-obese women in a metabolic facility. *Int J Obesity* **22**, 303-31.

Prentice AM (1995) Alcohol and obesity. *Int J Obes Relat Metab Disord* **19 Suppl 5**, S44-S50.

Prentice AM (1998) Manipulation of dietary fat and energy density and subsequent effects on substrate flux and food intake. *Am J Clin Nutr* **67**, 535S-5541.

Prentice AM, Black AE, Coward WA & Cole TJ (1996a) Energy expenditure in overweight and obese adults in affluent societies: an analysis of 319 doubly-labelled water measurements. *Eur J Clin Nutr* **50**, 93-97.

Prentice AM, Black AE, Coward WA, Davies HL, Goldberg GR, Murgatroyd PR, Ashford J, Sawyer M & Whitehead RG (1986) High levels of energy expenditure in obese women. *Br Med J (Clin Res Ed)* **292**, 983-987.

Prentice AM & Goldberg GR (2000) Energy adaptations in human pregnancy: limits and long-term consequences. *Am J Clin Nutr* **71**, 1226S-1232

Prentice AM, Goldberg GR, Murgatroyd PR & Cole TJ (1996c) Physical activity and obesity: problems in correcting expenditure for body size. *Int J Obes Relat Metab Disord* **20**, 688-691.

Prentice AM & Jebb SA (2004) Energy intake/physical activity interactions in the homeostasis of body weight regulation. *Nutr Rev* **62**, S98-104.

Prentice AM, Lucas A, Vasquez-Velasquez L, Davies PS & Whitehead RG (1988) Are current dietary guidelines for young children a prescription for overfeeding? *Lancet* **2**, 1066-1069.

Prentice AM, Spaaij CJ, Goldberg GR, Poppitt SD, van Raaij JM, Totton M, Swann D & Black AE (1996b) Energy requirements of pregnant and lactating women. *Eur J Clin Nutr* **50 Suppl 1**, S82-110.

Proctor MH, Moore LL, Gao D, Cupples LA, Bradlee ML, Hood MY & Ellison RC (2003) Television viewing and change in body fat from preschool to early adolescence: The Framingham Children's Study. *Int J Obes Relat Metab Disord* **27**, 827-833.

Proper KI, Hildebrandt VH, van der Beek AJ, Twisk JW, van Mechelen W (2003) Effect of individual counseling on physical activity fitness and health: a randomized controlled trial in a workplace setting. *Am J Prev Med* **24**(3), 218-226.

Prospective Studies Collaboration, Whitlock G, Lewington S, Sherliker P, Clarke R, Emberson J, Halsey J, Qizilbash N, Collins R & Peto R (2009) Body-mass index and cause-specific mortality in 900 000 adults: collaborative analyses of 57 prospective studies *Lancet* **373,** 1083-1096.

Raben A, Agerholm-Larsen L, Flint A, Holst JJ & Astrup A (2003) Meals with similar energy densities but rich in protein, fat, carbohydrate, or alcohol have different effects on energy expenditure and substrate metabolism but not on appetite and energy intake. *Am J Clin Nutr* **77**, 91-100.

Rainwater DL, Mitchell BD, Comuzzie AG, VandeBerg JL, Stern MP & Maccluer JW (2000) Association among 5-year changes in weight, physical activity, and cardiovascular disease risk factors in Mexican Americans. *Am J Epidemiol* **152**, 974-982.

Rampersaud E, Mitchell BD, Pollin TI et al (2008) Physical Activity and the Association of Common FTO Gene Variants With Body Mass Index and Obesity. *Arch Intern Med* **168**(16), 1791-1797.

Ravussin E, Harper IT, Rising R & Bogardus C (1991) Energy expenditure by doubly labeled water: validation in lean and obese subjects. *Am J Physiol* **261**, E402-E409.

Ravussin E, Lillioja S, Anderson T, Christin L & Bogardus C (1986) Determinants of 24-hour energy expenditure in man. Methods and results using a respiratory chamber. pp. 1562-1578.

Rawson ES, Nolan A, Silver K, Shuldiner AR & Poehlman ET (2002) No effect of the Trp64Arg beta(3)-adrenoceptor gene variant on weight loss, body composition, or energy expenditure in obese, caucasian postmenopausal women. *Metabolism* **51**, 801-805.

Reilly JJ, Armstrong J, Dorosty AR, Emmett PM, Ness A, Rogers I, Steer C & Sherriff A (2005) Early life risk factors for obesity in childhood: cohort study. *BMJ* **330**, 1357.

Reilly JJ, Corbett J, Given L, Gray L, Leyland A, MacGregor A, Marryat L, Miller M & Reid S (2009) *The Scottish Health Survey 2008*. Edinburgh: The Scottish Government.

Reimer RA, Debert CT, House JL & Poulin MJ (2005) Dietary and metabolic differences in pre- versus postmenopausal women taking or not taking hormone replacement therapy. *Physiol Behav* **84**, 303-312.

Renehan AG, Tyson M, Egger M, Heller RF & Zwahlen M (2008) Body-mass index and incidence of cancer: a systematic review and meta-analysis of prospective observational studies. *Lancet* **371**, 569-578.

Rennie KL, Coward A & Jebb SA (2007) Estimating under-reporting of energy intake in dietary surveys using an individualised method. *Br J Nutr* **97**, 1169-1176.

Rennie KL, Jebb SA, Wright A & Coward WA (2005) Secular trends in under-reporting in young people. *Br J Nutr* **93**, 241-247.

Rennie KL, Wells JC, McCaffrey TA & Livingstone MB (2006) The effect of physical activity on body fatness in children and adolescents. *Proc Nutr Soc* **65**, 393-402.

Riddoch CJ, Mattocks C, Deere K, Saunders J, Kirkby J, Tilling K, Leary SD, Blair SN & Ness AR (2007) Objective measurement of levels and patterns of physical activity. *Arch Dis Child* **92**, 963-969.

Rissanen J, Kuopusjarvi J, Pihlajamaki J, Sipilainen R, Heikkinen S, Vanhala M, Kekalainen P, Kuusisto J & Laakso M (1997) The Trp64Arg polymorphism of the beta 3-Adrenergic receptor gene. Lack of association with NIDDM and features of insulin resistance syndrome. *Diabetes Care* **20**, 1319-1323.

Roberts SB & Dallal GE (1998) Effects of age on energy balance. *Am J Clin Nutr* **68**, 975S-979S.

Roberts SB, Dietz W, Sharp T, Dallal GE & Hill JO (1995a) Multiple laboratory comparison of the doubly labeled water technique. *Obes Res* **3 Suppl 1**, 3-13.

Roberts SB, Fuss P, Heyman MB & Young VR (1995b) Influence of age on energy requirements. *Am J Clin Nutr* **62**, 1053S-1058S.

Roberts SB, Nicholson M, Staten M, Dallal GE, Sawaya AL, Heyman MB, Fuss P & Greenberg AS (1997) Relationship between circulating leptin and energy expenditure in adult men and women aged 18 years to 81 years. *Obesity Res* **5**, 459-463.

Roberts SB & Rosenberg I (2006) Nutrition and Aging: Changes in the Regulation of Energy Metabolism With Aging. *Physiol Rev* **86**, 651-667.

Roberts SB, Savage J, Coward WA, Chew B & Lucas A (1988) Energy expenditure and intake in infants born to lean and overweight mothers. *N Engl J Med* **318**, 461-466.

Robinson TN, Killen JD, Kraemer HC*, et al.* (2003) Dance and reducing television viewing to prevent weight gain in African-American girls: the Stanford GEMS pilot study. *Ethn Dis* **13**, S65-S77.

Roemmich JN, Clark PA, Mai V, Berr SS, Weltman A, Veldhuis JD & Rogol AD (1998) Alterations in growth and body composition during puberty: III. Influence of maturation, gender, body composition, fat distribution, aerobic fitness, and energy expenditure on nocturnal growth hormone release. *J Clin Endocrinol Metab* **83**, 1440-1447.

Roemmich JN, Clark PA, Walter K, Patrie J, Weltman A & Rogol AD (2000) Pubertal alterations in growth and body composition. V. Energy expenditure, adiposity, and fat distribution. *Am J Physiol Endocrinol Metab* **279**, E1426-E1436.

Rosenbaum M, Goldsmith R, Bloomfield D, Magnano A, Weimer L, Heymsfield S, Gallagher D, Mayer L, Murphy E & Leibel RL (2005) Low-dose leptin reverses skeletal muscle, autonomic, and neuroendocrine adaptations to maintenance of reduced weight. *J Clin Invest* **115**, 3579-3586.

Rosenbaum M, Hirsch J, Murphy E & Leibel RL (2000) Effects of changes in body weight on carbohydrate metabolism, catecholamine excretion, and thyroid function. *Am J Clin Nutr* **71**, 1421-1432.

Rosenbaum M, Murphy EM, Heymsfield SB, Matthews DE & Leibel RL (2002) Low Dose Leptin Administration Reverses Effects of Sustained Weight-Reduction on Energy Expenditure and Circulating Concentrations of Thyroid Hormones. *J Clin Endocrinol Metab* **87**, 2391.

Rothenberg EM, Bosaeus IG, Westerterp KR & Steen BC (2000) Resting energy expenditure, activity energy expenditure and total energy expenditure at age 91-96 years. *Br J Nutr* **84**, 319-324.

Rosenbaum M, Vandenborne K, Goldsmith R*, et al.* (2003) Effects of experimental weight perturbation on skeletal muscle work efficiency in human subjects. *Am J Physiol Regul Integr Comp Physiol* **285**, R183-R192.

Royal College of Paediatrics and Child Health (2011) UK-WHO Growth Charts: early years. Available at: http://www.rcpch.ac.uk/growthcharts

Royal College of Physicians (2004) *Storing up problems: The medical case for a slimmer nation*. London: RCP.

Rush EC, Plank LD & Coward WA (1999) Energy expenditure of young Polynesian and European women in New Zealand and relations to body composition. *Am J Clin Nutr* **69**, 43-48.

Rush EC, Plank LD, Davies PS, Watson P & Wall CR (2003) Body composition and physical activity in New Zealand Maori, Pacific and European children aged 5-14 years. *Br J Nutr* **90**, 1133-1139.

Ruston D, Hoare J, Henderson L, Gregory J, Bates CJ, Prentice A, Birch M, Swan G & Farron M (2004) *The National Diet and Nutrition Survey: adults aged 19 to 64 years. Volume 4: Nutritional Status (anthropometry and blood analytes), blood pressure and physical activity*. London: TSO.

Rutanen J, Pihlajamaki J, Karhapaa P et al (2004) The Val103Ile polymorphism of melanocortin-4 receptor regulates energy expenditure and weight gain. *Obes Res* **12**(7), 1060-1066.

Sahota P, Rudolf MC, Dixey R, Hill AJ, Barth JH & Cade J (2001) Randomised controlled trial of primary school based intervention to reduce risk factors for obesity. *BMJ* **323**, 1029-1032.

Salazar G, Vio F, Aguirre E & Coward WA (2000) Energy requirements in Chilean infants. *Arch Dis Child Fetal Neonatal Ed* **83**, F123.

Salbe AD, Nicolson M & Ravussin E (1997) Total energy expenditure and the level of physical activity correlate with plasma leptin concentrations in five-year-old children. *J Clin Invest* **99**, 592-595.

Sallis JF, McKenzie TL, Conway TL, Elder JP, Prochaska JJ, Brown M, Zive MM, Marshall SJ & Alcaraz JE (2003) Environmental interventions for eating and physical activity: a randomized controlled trial in middle schools. *Am J Prev Med* **24**, 209-217.

Saris WHM, Emons HJG, Groenenboom DC & Westerterp KR (1989) *Discrepancy between FAO/WHO energy requirements and actual energy expenditure in healthy 7-11 year old children*. Lueven, Belgium: 14th International Seminar on Pediatric Work Physiology.

Satoh Y, Shimizu T, Lee T, Nishizawa K, Iijima M & Yamashiro Y (2003) Resting energy expenditure and plasma leptin levels in adolescent girls with anorexia nervosa. *Int J Eat Disord* **34**, 156-161.

Schmitz KH, Jacobs DR, Jr., Leon AS, Schreiner PJ & Sternfeld B (2000) Physical activity and body weight: associations over ten years in the CARDIA study. Coronary Artery Risk Development in Young Adults. *Int J Obes Relat Metab Disord* **24**, 1475-1487.

Schoeller DA (1998) Balancing energy expenditure and body weight. *Am J Clin Nutr* **68**, 956S-961S.

Schoeller DA & Hnilicka JM (1996) Reliability of the doubly labeled water method for the measurement of total daily energy expenditure in free-living subjects. *J Nutr* **126**, 348S-354S.

Schoeller DA, Leitch CA & Brown C (1986) Doubly labeled water method: in vivo oxygen and hydrogen isotope fractionation. *Am J Physiol Regul Integr Comp Physiol* **251**, R1137-R1143.

Schofield WN, Schofield C & James WPT (1985) Basal metabolic rate - review and prediction, together with an annotated bibliography of source material. *Hum Nutr Clin Nutr* **39C Suppl 1**, 1-96.

Schulz LO & Schoeller DA (1994) A compilation of total daily energy expenditures and body weights in healthy adults. *Am J Clin Nutr* **60**, 676-681.

Schutz Y (2000) Role of substrate utilization and thermogenesis on body-weight control with particular reference to alcohol. *Proceedings of the Nutrition Society* **59**, 511-517.

Schutz Y, Bessard T & Jequier E (1984) Diet-induced thermogenesis measured over a whole day in obese and nonobese women. *Am J Clin Nutr* **40**, 542-552.

Schutz Y & Garrow JS (2000) Energy and substrate balance and weight regulation. In *Human nutrition and dietetics*, pp. 137-148 [JS Garrow, WPT James, and A Ralph, editors]. Edinburgh: Churchill Livingstone.

Scientific Advisory Committee on Nutrition (2002) *A framework for the Evaluation of Evidence* no. http://www.sacn.gov.uk/pdfs/sacn_02_02a.pdf.

Scientific Advisory Committee on Nutrition (2008) *The Nutritional Wellbeing of the British Population.* London: TSO.

Scientific Advisory Committee on Nutrition & The Royal College of Paediatrics and Child Health (2007) *Application of WHO Growth Standards in the UK. Report prepared by the Joint SACN/RCPCH Expert Group on Growth Standards*. London: TSO.

Scuteri A, Sanna S, Chen WM*, et al.* (2007) Genome-wide association scan shows genetic variants in the FTO gene are associated with obesity-related traits. *PLoS Genet* **3**, e115.

Segal KR, Gutin B, Albu J & Pi-Sunyer FX (1987) Thermic effects of food and exercise in lean and obese men of similar lean body mass. *Am J Physiol* **252**, E110-E117.

Sesso HD (2007) Invited Commentary: A Challenge for Physical Activity Epidemiology. *Am J Epidemiol* **165**, 1351-1353.

Sharp TA, Bell ML, Grunwald GK, Schmitz KH, Sidney S, Lewis CE, Tolan K & Hill JO (2002) Differences in Resting Metabolic Rate between White and African-American Young Adults. *Obesity Res* **10**, 726-732.

Shaw K, Gennat H, O'Rourke P & Del Mar C (2006) Exercise for overweight or obesity. *Cochrane Database Syst Rev*, CD003817.

Sherwood NE, Jeffery RW, French SA, Hannan PJ & Murray DM (2000) Predictors of weight gain in the Pound of Prevention study. *Int J Obes Relat Metab Disord* **24**, 395-403.

Sherwood NE, Morton N, Jeffery RW, French SA, Neumark-Sztainer D & Falkner NH (1998) Consumer preferences in format and type of community-based weight control programs. *Am J Health Promot* **13**, 12-18.

Shiwaku K, Nogi A, Anuurad E, Kitajima K, Enkhmaa B, Shimono K & Yamane Y (2003) Difficulty in losing weight by behavioral intervention for women with Trp64Arg polymorphism of the beta3-adrenergic receptor gene. *Int J Obes Relat Metab Disord* **27**, 1028-1036.

Siega-Riz AM, Viswanathan M, Moos MK, Deierlein A, Mumford S, Knaack J, Thieda P, Lux LJ & Lohr KN (2009) A systematic review of outcomes of maternal weight gain according to the Institute of Medicine recommendations: birthweight, fetal growth, and postpartum weight retention. *Am J Obstet Gynecol* **201**, 339-343.

Siler SQ, Neese RA & Hellerstein MK (1999) De novo lipogenesis, lipid kinetics, and whole-body lipid balances in humans after acute alcohol consumption. *Am J Clin Nutr* **70**, 928-936.

Simkin-Silverman LR, Wing RR, Boraz MA & Kuller LH (2003) Lifestyle intervention can prevent weight gain during menopause: results from a 5-year randomized clinical trial. *Ann Behav Med* **26**, 212-220.

Simon C, Schweitzer B, Oujaa M, Wagner A, Arveiler D, Triby E, Copin N, Blanc S & Platat C (2008) Successful overweight prevention in adolescents by increasing physical activity: a 4-year randomized controlled intervention. *Int J Obes (Lond)* **32**, 1489-1498.

Simonetti D'Arca A, Tarsitani G, Cairella M, Siani V, De Filippis S, Mancinelli S, Marazzi MC & Palombi L (1986) Prevention of obesity in elementary and nursery school children. *Public Health* **100**, 166-173.

Sipilainen R, Uusitupa M, Heikkinen S, Rissanen A, Laakso M (1997) Variants in the human intestinal fatty acid binding protein 2 gene in obese subjects. J Clin Endocrinol Metab;**82**(8), 2629-2632.

Sjoberg A, Slinde F, Arvidsson D, Ellegard L, Gramatkovski E, Hallberg L & Hulthen L (2003) Energy intake in Swedish adolescents: validation of diet history with doubly labelled water. *Eur J Clin Nutr* **57**, 1643-1652.

Snitker S, Tataranni PA & Ravussin E (2001) Spontaneous physical activity in a respiratory chamber is correlated to habitual physical activity. *Int J Obes Relat Metab Disord* **25**, 1481-1486.

Soares MJ, Piers LS, O'Dea K & Shetty PS (1998) No evidence for an ethnic influence on basal metabolism: an examination of data from India and Australia. *Br J Nutr* **79**, 333-341.

Solomon SJ, Kurzer MS & Calloway DH (1982) Menstrual cycle and basal metabolic rate in women. *Am J Clin Nutr* **36**, 611-616.

Spadano JL, Bandini LG, Must A, Dallal GE & Dietz WH (2005) Longitudinal changes in energy expenditure in girls from late childhood through midadolescence. *Am J Clin Nutr* **81**, 1102-1109.

Sparti A, DeLany JP, de la Bretonne JA, Sander GE & Bray GA (1997) Relationship between resting metabolic rate and the composition of the fat-free mass. *Metabolism* **46**, 1225-1230.

Speakman JR & Selman C (2003) Physical activity and resting metabolic rate. *Proc Nutr Soc* **62**, 621-634.

Statistics for Wales/Ystadegau ar gyfer Cymru (2010) *Welsh Health Survey 2009*: Welsh Assembly Government.

Stefan N, Vozarova B, Del Parigi A, Ossowski V, Thompson DB, Hanson RL, Ravussin E & Tataranni PA (2002) The Gln223Arg polymorphism of the leptin receptor in Pima Indians:

influence on energy expenditure, physical activity and lipid metabolism. *Int J Obes* Relat *Metab Disord* **26**, 1629-1632.

Stephen AM, Teucher B, Bates BJ, Bluck LJ, Mander AP (2007) UK National Diet and Nutrition Survey: a study to decide the dietary assessment method for the new rolling programme (unpublished).

Stolley MR & Fitzgibbon ML (1997) Effects of an obesity prevention program on the eating behavior of African American mothers and daughters. *Health Educ Behav* **24**, 152-164.

Stothard KJ, Tennant PW, Bell R & Rankin J (2009) Maternal overweight and obesity and the risk of congenital anomalies: a systematic review and meta-analysis. *JAMA* **301**, 636-650.

Stratz CH (1904) *Der Korper des Kindes*: Fur Eltern, Arzta und Kunstler. Ferdinland Enke. Stuttgart: Germany.

Stubbs RJ, Harbron CG, Murgatroyd PR & Prentice AM (1995b) Covert manipulation of dietary fat and energy density: effect on substrate flux and food intake in men eating ad libitum. *Am J Clin Nutr* **62**, 316-329.

Stubbs RJ, Hughes DA, Johnstone AM, Horgan GW, King N & Blundell JE (2004) A decrease in physical activity affects appetite, energy, and nutrient balance in lean men feeding ad libitum. *Am J Clin Nutr* **79**, 62-69.

Stubbs RJ, Ritz P, Coward WA & Prentice AM (1995a) Covert manipulation of the ratio of dietary fat to carbohydrate and energy density: effect on food intake and energy balance in free-living men eating ad libitum. *Am J Clin Nutr* **62**, 330-337.

Styne DM (2005) Obesity in childhood: what's activity got to do with it? *Am J Clin Nutr* **81**, 337-338.

Subar AF, Kipnis V, Troiano RP*, et al.* (2003) Using intake biomarkers to evaluate the extent of dietary misreporting in a large sample of adults: the OPEN study. *Am J Epidemiol* **158**, 1-13.

Sun M, Gower BA, Bartolucci AA, Hunter GR, Figueroa-Colon R & Goran MI (2001) A longitudinal study of resting energy expenditure relative to body composition during puberty in African American and white children. *Am J Clin Nutr* **73**, 308-315.

Sun M, Gower BA, Nagy TR, Bartolucci AA & Goran MI (1999) Do hormonal indices of maturation explain energy expenditure differences in African American and Caucasian prepubertal children? *Int J Obes Relat Metab Disord* **23**, 1320-1326.

Sun M, Gower BA, Nagy TR, Trowbridge CA, Dezenberg C & Goran MI (1998) Total, resting, and activity-related energy expenditures are similar in Caucasian and African-American children. *Am J Physiol* **274**, E232-E237.

Svendsen OL, Hassager C & Christiansen C (1993) Impact of regional and total body composition and hormones on resting energy expenditure in overweight postmenopausal women. *Metabolism* **42**, 1588-1591.

Swinamer DL, Phang PT, Jones RL, Grace M & King EG (1988) Effect of routine administration of analgesia on energy expenditure in critically ill patients. *Chest* **93**, 4-10.

Tammelin T, Laitinen J & Nayha S (2004) Change in the level of physical activity from adolescence into adulthood and obesity at the age of 31 years. *Int J Obes Relat Metab Disord* **28**, 775-782.

Tanner JM (1990) *Foetus into man: physical growth from conception to maturity*, Second ed.: Castlemead Publications, Ware.

Tataranni PA, Baier L, Jenkinson C, Harper I, Del Parigi A, Bogardus C (2001) A Ser311Cys mutation in the human dopamine receptor D2 gene is associated with reduced energy expenditure. *Diabetes* **50**(4), 901-904.

Tataranni PA, Larson DE, Snitker S, Young JB, Flatt JP & Ravussin E (1996) Effects of glucocorticoids on energy metabolism and food intake in humans. *Am J Physiol Endocrinol Metab* **271**, E317-E325.

Tchernof A, Starling RD, Walston JD, Shuldiner AR, Dvorak RV, Silver K, Matthews DE & Poehlman ET (1999) Obesity-related phenotypes and the beta3-adrenoceptor gene variant in postmenopausal women. *Diabetes* **48**, 1425-1428.

Tershakovec AM, Kuppler KM, Zemel B, Stallings VA. Age, sex, ethnicity, body composition, and resting energy expenditure of obese African American and white children and adolescents. Am J Clin Nutr 2002;75(5):867-871.

Timpson NJ, Emmett PM, Frayling TM, Rogers I, Hattersley AT, McCarthy MI & Davey Smith G (2008) The fat mass-and obesity-associated locus and dietary intake in children. *Am J Clin Nutr* **88**, 971-978.

Tooze JA, Schoeller DA, Subar AF, Kipnis V, Schatzkin A & Troiano RP (2007) Total daily energy expenditure among middle-aged men and women: the OPEN Study. *Am J Clin Nutr* **86**, 382-387.

Torun B (2005) Energy requirements of children and adolescents. *Public Health Nutr* **8**, 968-993.

Toth MJ, Gottlieb SS, Fisher ML, Ryan AS, Nicklas BJ & Poehlman ET (1997) Plasma leptin concentrations and energy expenditure in heart failure patients. *Metabolism* **46**, 450-453.

Toubro S, Sorensen TI, Ronn B, Christensen NJ & Astrup A (1996) Twenty-four-hour energy expenditure: the role of body composition, thyroid status, sympathetic activity, and family membership. *J Clin Endocrinol Metab* **81**, 2670-2674.

Trabulsi J, Troiano RP, Subar AF, Sharbaugh C, Kipnis V, Schatzkin A & Schoeller DA (2003) Precision of the doubly labeled water method in a large-scale application: evaluation of a streamlined-dosing protocol in the Observing Protein and Energy Nutrition (OPEN) study. *Eur J Clin Nutr* **57**, 1370-1377.

Trayers T, Cooper AR, Riddoch CJ, Ness AR, Fox KR, Deem R & Lawlor DA (2006) Do children from an inner city British school meet the recommended levels of physical activity? Results from a cross sectional survey using objective measurements of physical activity. *Arch Dis Child* **91**, 175-176.

Trayhurn P (2001) Biology of leptin--its implications and consequences for the treatment of obesity. *Int J Obes Relat Metab Disord* **25 Suppl 1**, S26-S28.

Tremblay A, Simoneau JA & Bouchard C (1994) Impact of exercise intensity on body fatness and skeletal muscle metabolism. *Metabolism* **43**, 814-818.

Treuth MS, Butte NF, Puyau M & Adolph A (2000b) Relations of parental obesity status to physical fitness of prepubertal girls. *Pediatrics* **106**.

Treuth MS, Butte NF & Sorkin JD (2003) Predictors of body fat gain in nonobese girls with a familial predisposition to obesity. *Am J Clin Nutr* **78**, 1212-1218.

Treuth MS, Butte NF & Wong WW (2000a) Effects of familial predisposition to obesity on energy expenditure in multiethnic prepubertal girls. *Am J Clin Nutr* **71**, 893-900.

Treuth MS, Figueroa-Colon R, Hunter GR, Weinsier RL, Butte NF & Goran MI (1998) Energy expenditure and physical fitness in overweight vs non-overweight prepubertal girls. *Int J Obes Relat Metab Disord* **22**, 440-447.

Treuth MS, Butte NF & Sorkin JD (2003) Predictors of body fat gain in nonobese girls with a familial predisposition to obesity. *Am J Clin Nutr* **78**, 1212-1218.

Trowbridge CA, Gower BA, Nagy TR, Hunter GR, Treuth MS & Goran MI (1997) Maximal aerobic capacity in African-American and Caucasian prepubertal children. *Am J Physiol* **273**, E809-E814.

Twisk JW, Kemper HC & van Mechelen W (2000) Tracking of activity and fitness and the relationship with cardiovascular disease risk factors. *Med Sci Sports Exerc* **32**, 1455-1461.

Ukkola O, Tremblay A, Sun G, Chagnon YC & Bouchard C (2001) Genetic variation at the uncoupling protein 1, 2 and 3 loci and the response to long-term overfeeding. *Eur J Clin Nutr* **55**, 1008-1015.

Valencia ME, McNeill G, Brockway JM & Smith JS (1992) The effect of environmental temperature and humidity on 24 h energy expenditure in men. *Br J Nutr* **68**, 319-327.

Valencia ME, McNeill G, Haggarty P, Moya SY, Pinelli A, Quihui L & Davalos R (1995) Energetic consequences of mild Giardia intestinalis infestation in Mexican children. *Am J Clin Nutr* **61**, 860-865.

Valve R, Heikkinen S, Rissanen A, Laakso M & Uusitupa M (1998) Synergistic effect of polymorphisms in uncoupling protein 1 and beta3-adrenergic receptor genes on basal metabolic rate in obese Finns. *Diabetologia* **41**, 357-361.

van Marken Lichtenbelt WD, Schrauwen P, van de Kerckhove S & Westerterp-Plantenga MS (2002) Individual variation in body temperature and energy expenditure in response to mild cold. *Am J Physiol Endocrinol Metab* **282**, E1077-E1083.

van Marken Lichtenbelt W, Westerterp-Plantenga MS & van Hoydonck P (2001) Individual variation in the relation between body temperature and energy expenditure in response to elevated ambient temperature. *Physiol Behav* **73**, 235-242.

van Sluijs EM, Skidmore PM, Mwanza K, *et al.* (2008) Physical activity and dietary behaviour in a population-based sample of British 10-year old children: the SPEEDY study (Sport, Physical activity and Eating behaviour: Environmental Determinants in Young people). *BMC Public Health* **8**, 388.

Van Wymelbeke V, Brondel L, Marcel Brun J & Rigaud D (2004) Factors associated with the increase in resting energy expenditure during refeeding in malnourished anorexia nervosa patients. *Am J Clin Nutr* **80**, 1469-1477.

Vasquez F, Salazar G, Andrade M, Vasquez L & Diaz E (2006) Energy balance and physical activity in obese children attending day-care centres. *Eur J Clin Nutr* **60**, 1115-1121.

Vaughan L, Zurlo F & Ravussin E (1991) Aging and energy expenditure. *Am J Clin Nutr* **53**, 821-825.

Vaz M, Karaolis N, Draper A & Shetty P (2005) A compilation of energy costs of physical activities. *Public Health Nutr* **8**, 1153-1183.

Verstraete SJ, Cardon GM, de Clercq DL & De Bourdeaudhuij IM (2007) A comprehensive physical activity promotion programme at elementary school: the effects on physical activity, physical fitness and psychosocial correlates of physical activity. *Public Health Nutr* **10**, 477-484.

Viner RM & Cole TJ (2005) Television viewing in early childhood predicts adult body mass index. *J Pediatr* **147**, 429-435.

Visser M, Deurenberg P, Van Staveren WA & Hautvast JG (1995) Resting metabolic rate and diet-induced thermogenesis in young and elderly subjects: relationship with body composition, fat distribution, and physical activity level. *Am J Clin Nutr* **61**, 772-778.

Wagner A, Simon C, Ducimetiere P, *et al.* (2001) Leisure-time physical activity and regular walking or cycling to work are associated with adiposity and 5 y weight gain in middle-aged men: the PRIME Study. *Int J Obes Relat Metab Disord* **25**, 940-948.

Walder K, Norman RA, Hanson RL, *et al.* (1998) Association between uncoupling protein polymorphisms (UCP2-UCP3) and energy metabolism/obesity in Pima indians. *Hum Mol Genet* **7**, 1431-1435.

Walker A, Maher J, Coulthard M, Goddard E & Thomas M (2001) *Living in Britain: Results from the 2000/01 General Household Survey*. London: The Stationery Office.

Wallace JD, Abbott-Johnson WJ, Crawford DHG, Barnard R, Potter JM & Cuneo RC (2002) GH Treatment in Adults with Chronic Liver Disease: A Randomized, Double-Blind, Placebo-Controlled, Cross-Over Study. *J Clin Endocrinol Metab* **87**, 2751-2759.

Waller K, Kaprio J, Kujala UM (2008) Associations between long-term physical activity, waist circumference and weight gain: a 30-year longitudinal twin study. *Int J Obes* (Lond) **32**(2), 353-361.

Wang Z, Heshka S, Gallagher D, Boozer CN, Kotler DP & Heymsfield SB (2000) Resting energy expenditure-fat-free mass relationship: new insights provided by body composition modeling. *Am J Physiol Endocrinol Metab* **279**, E539-E545.

Wardle J & Boniface D (2008) Changes in the distributions of body mass index and waist circumference in English adults, 1993//1994 to 2002//2003. *Int J Obes* **32**, 527-532.

Wardle J, Brodersen NH & Boniface D (2007) School-based physical activity and changes in adiposity. *Int J Obes*.

Wardle J, Carnell S, Haworth CM, Farooqi IS, O'Rahilly S & Plomin R (2008) Obesity-associated genetic variation in FTO is associated with diminished satiety. *J Clin Endocrinol Metab* **93**, 3640-3643.

Wardle J, Llewellyn C, Sanderson S & Plomin R (2009) The FTO gene and measured food intake in children. *Int J Obes* **33**, 42-45.

Wareham NJ, Jakes RW, Rennie KL, Mitchell J, Hennings S & Day NE (2002) Validity and repeatability of the EPIC-Norfolk Physical Activity Questionnaire. *Int J Epidemiol* **31**, 168-174.

Wareham NJ & Rennie KL (1998) The assessment of physical activity in individuals and populations: why try to be more precise about how physical activity is assessed? *Int J Obes Relat Metab Disord* **22 Suppl 2**, S30-S38.

Wareham NJ, van Sluijs EM & Ekelund U (2005) Physical activity and obesity prevention: a review of the current evidence. *Proc Nutr Soc* **64**, 229-247.

Warren JM, Henry CJ, Lightowler HJ, Bradshaw SM & Perwaiz S (2003) Evaluation of a pilot school programme aimed at the prevention of obesity in children. *Health Promot Int* **18**, 287-296.

Warwick PM (2006) Factorial estimation of daily energy expenditure using a simplified method was improved by adjustment for excess post-exercise oxygen consumption and thermic effect of food. *Eur J Clin Nutr* **60**, 1337-1340.

Warwick PM & Busby R (1990) Influence of mild cold on 24 h energy expenditure in 'normally' clothed adults. *Br J Nutr* **63**, 481-488.

Wauters M, Considine RV, Chagnon M, Mertens I, Rankinen T, Bouchard C & Van Gaal LF (2002) Leptin levels, leptin receptor gene polymorphisms, and energy metabolism in women. *Obes Res* **10**, 394-400.

Webb P (1986) 24-hour energy expenditure and the menstrual cycle. *Am J Clin Nutr* **44**, 614-619.

Weijs PJ (2008) Validity of predictive equations for resting energy expenditure in US and Dutch overweight and obese class I and II adults aged 18-65 y. *Am J Clin Nutr* **88**, 959-970.

Weinsier RL, Hunter GR, Desmond RA, Byrne NM, Zuckerman PA & Darnell BE (2002) Free-living activity energy expenditure in women successful and unsuccessful at maintaining a normal body weight. *Am J Clin Nutr* **75**, 499-504.

Weinsier RL, Hunter GR, Zuckerman PA, Redden DT, Darnell BE, Larson DE, Newcomer BR & Goran MI (2000) Energy expenditure and free-living physical activity in black and white women: comparison before and after weight loss. *Am J Clin Nutr* **71**, 1138-1146.

Weinsier RL, Schutz Y & Bracco D (1992) Reexamination of the relationship of resting metabolic rate to fat-free mass and to the metabolically active components of fat-free mass in humans. *Am J Clin Nutr* **55**, 790-794.

Welk GJ, Corbin CB & Dale D (2000) Measurement issues in the assessment of physical activity in children. *Res Q Exerc Sport* **71**, S59-S73.

Welle S, Jozefowicz R & Statt M (1990) Failure of dehydroepiandrosterone to influence energy and protein metabolism in humans. *J Clin Endocrinol Metab* **71**, 1259-1264.

Wells JC, Chomtho S, Fewtrell MS (2007b) Programming of body composition by early growth and nutrition. *Proc Nutr Soc* **66**(3), 423-434.

Wells JC, Coward WA, Cole TJ, Davies PS (2002) The contribution of fat and fat-free tissue to body mass index in contemporary children and the reference child. *Int J Obes Relat Metab Disord* **26**(10), 1323-1328.

Wells JC & Ritz P (2001) Physical activity at 9-12 months and fatness at 2 years of age. *Am J Hum Biol* **13**, 384-389.

Wells JC, Treleaven P & Cole TJ (2007a) BMI compared with 3-dimensional body shape: the UK National Sizing Survey. *Am J Clin Nutr* **85**, 419-425.

Wenche DB, Holmen J, Kruger O & Midthjell K (2004) Leisure time physical activity and change in body mass index: an 11-year follow-up study of 9357 normal weight health women 20-49 years old. *J Womens Health (Larchmt)* **13**, 55-62.

Westerterp KR & Goris AHC (2002) Validity of the assessment of dietary intake: problems of mis-reporting. *Current Opinion in Clinical Nutrition Metabolic Care.* **5**, 489-493.

Westerterp KR (1998) Alterations in energy balance with exercise. *Am J Clin Nutr* **68**, 970S-974S.

Westerterp KR, Meijer GA, Janssen EM, Saris WH & Ten Hoor F (1992) Long-term effect of physical activity on energy balance and body composition. *Br J Nutr* **68**, 21-30.

Westerterp KR & Plasqui G (2004) Physical activity and human energy expenditure. *Curr Opin Clin Nutr Metab Care* **7**, 607-613.

Westerterp-Plantenga MS, van Marken Lichtenbelt WD, Strobbe H & Schrauwen P (2002) Energy metabolism in humans at a lowered ambient temperature. *Eur J Clin Nutr* **56**, 288-296.

Weyer C, Snitker S, Bogardus C & Ravussin E (1999) Energy metabolism in African Americans: potential risk factors for obesity. *Am J Clin Nutr* **70**, 13-20.

WHO Multicentre Growth Reference Study Group (2006) Complementary feeding in the WHO Multicentre Growth Reference Study. *Acta Paediatrica* **Suppl 450**, 27-37.

Wilks DC, Besson H, Lindroos AK & Ekelund U (2011) Objectively measured physical activity and obesity prevention in children, adolescents and adults: a systematic review of prospective studies. *Obes Res* **12**, e119-e129.

Willett WC (2003) Invited Commentary: OPEN Questions. *Am J Epidemiol* **158**, 22-24.

Wilsgaard T, Jacobsen BK & Arnesen E (2005) Determining Lifestyle Correlates of Body Mass Index using Multilevel Analyses: The Tromso Study, 1979-2001. *Am J Epidemiol* **162**, 1179-1188.

Wilson MM & Morley JE (2003) Invited Review: Aging and energy balance. *J Appl Physiol* **95**, 1728-1736.

Wing RR & Hill JO (2001) Successful weight loss maintenance. *Annu Rev Nutr* **21**, 323-341.

Withers RT, Smith DA, Tucker RC, Brinkman M & Clark DG (1998) Energy metabolism in sedentary and active 49- to 70-yr-old women. *J Appl Physiol* **84**, 1333-1340.

Wong WW (1994) Energy expenditure of female adolescents. *J Am Coll Nutr* **13**, 332-337.

Wong WW, Butte NF, Ellis KJ, Hergenroeder AC, Hill RB, Stuff JE & Smith EO (1999) Pubertal African-American girls expend less energy at rest and during physical activity than Caucasian girls. *J Clin Endocrinol Metab* **84**, 906-911.

World Health Organization (1985) *Energy and protein requirements. Report of a joint FAO/WHO/UNU expert consultation* no. 724. Geneva: WHO.

World Health Organization (1995) Maternal Anthropometry and Pregnancy Outcomes – A WHO Collaborative Study. *Bulletin of the World HealthOrganization* **73**, S1-S69.

World Health Organization (1998) *Obesity – Preventing and Managing the Global Epidemic* no. 894. Geneva: World Health Organization.

World Health Organization (2006) *WHO Child Growth Standards: Methods and development: Length/height-for-age, weight-for-age, weight-for-length, weight-for-height and body mass index-for-age.* Geneva, Switzerland: World Health Organization.

Wouters-Adriaens MP & Westerterp KR (2008) Low resting energy expenditure in Asians can be attributed to body composition. *Obesity (Silver Spring)* **16**, 2212-2216.

Wren RE, Blume H, Mazariegos M, Solomons N, Alvarez JO & Goran MI (1997) Body composition, resting metabolic rate, and energy requirements of short- and normal-stature, low-income Guatemalan children. *Am J Clin Nutr* **66**, 406-412.

Wu X, Luke A, Cooper RS et al (2004) A genome scan among Nigerians linking resting energy expenditure to chromosome 16. *Obes Res* **12**(4):577-581.

Yang X, Telama R, Leskinen E, Mansikkaniemi K, Viikari J & Raitakari OT (2007) Testing a model of physical activity and obesity tracking from youth to adulthood: the cardiovascular risk in young Finns study. *Int J Obes (Lond)* **31**, 521-527.

Yang X, Telama R, Viikari J & Raitakari OT (2006) Risk of obesity in relation to physical activity tracking from youth to adulthood. *Med Sci Sports Exerc* **38**, 919-925.

Yanovski JA, Diament AL, Sovik KN, Nguyen TT, Li H, Sebring NG & Warden CH (2000) Associations between uncoupling protein 2, body composition, and resting energy expenditure in lean and obese African American, white, and Asian children. *Am J Clin Nutr* **71**, 1405-1420.

Yanovski SZ, Reynolds JC, Boyle AJ & Yanovski JA (1997) Resting metabolic rate in African-American and Caucasian girls. *Obes Res* **5**, 321-325.

Yao M, McCrory MA, Ma G, Li Y, Dolnikowski GG, Roberts SB (2002) Energy requirements of urban Chinese adults with manual or sedentary occupations, determined using the doubly labelled water method. *Eur J Clin Nutr* 56, 575-584.

Yoshioka M, Doucet E, St Pierre S, Almeras N, Richard D, Labrie A, Despres JP, Bouchard C & Tremblay A (2001) Impact of high-intensity exercise on energy expenditure, lipid oxidation and body fatness. *Int J Obes Relat Metab Disord* **25**, 332-339.

Zurlo F, Ferraro RT, Fontvielle AM, Rising R, Bogardus C & Ravussin E (1992) Spontaneous physical activity and obesity: cross-sectional and longitudinal studies in Pima Indians. *Am J Physiol* **263**, E296-E300.

TSO
information & publishing solutions

www.tso.co.uk

ISBN 978-0-10-851137-0

9 780108 511370