



Paper for discussion: Energy Report Chapters

Agenda Item: 4

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- For information:
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Draft outline of the Energy Requirements Working Group report

Membership of the Scientific Advisory Committee on Nutrition: Energy requirements working group

Membership of the Scientific Advisory Committee on Nutrition

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Introduction

Background

1. The Dietary Reference Values (DRV) for food energy are used to provide guidance to consumers about how much energy they should consume to meet their needs, and, as a benchmark, to judge the adequacy of the food energy intake of the population. They are also used in clinical settings, by caterers and in food labelling (e.g. as a basis for Guideline Daily Amounts). Population targets for fat and sugar intakes are also expressed as a percentage of the DRV for food energy. The DRV for food energy, therefore, needs to be as accurate as possible and any changes to the DRV would have implications for the delivery of Government policy, e.g. in relation to food producer and consumer guidance on energy and other macronutrients, fat and sugar targets, labelling and catering.
2. The National Diet and Nutrition Survey (NDNS) series has consistently show average energy intakes to be below the DRV . The NDNS, and other surveys of the UK population, have also show that the number of people classified as overweight or obese is increasing (data from NDNS series is given in Appendix 1). These data indicate that on average habitual energy intake is in fact above energy need. Although it is recognized that under-reporting of food intake is responsible for at least some of the discrepancy between measured energy intake and recommended energy intake , these observations have led the Food Standards Agency to question the robustness of the DRV for energy, which were last reviewed in 1991 .
3. The FAO/WHO/UNU has also published updated recommendations for energy intake and expenditure in the report Consultation on Human Energy Requirements . The Working Group was asked to review that FAO/WHO/UNU Energy Consultation, the evidence used in the report and its implications for UK energy recommendation.

Terms of reference

The Terms of Reference for the Energy Requirements Working Group are to:

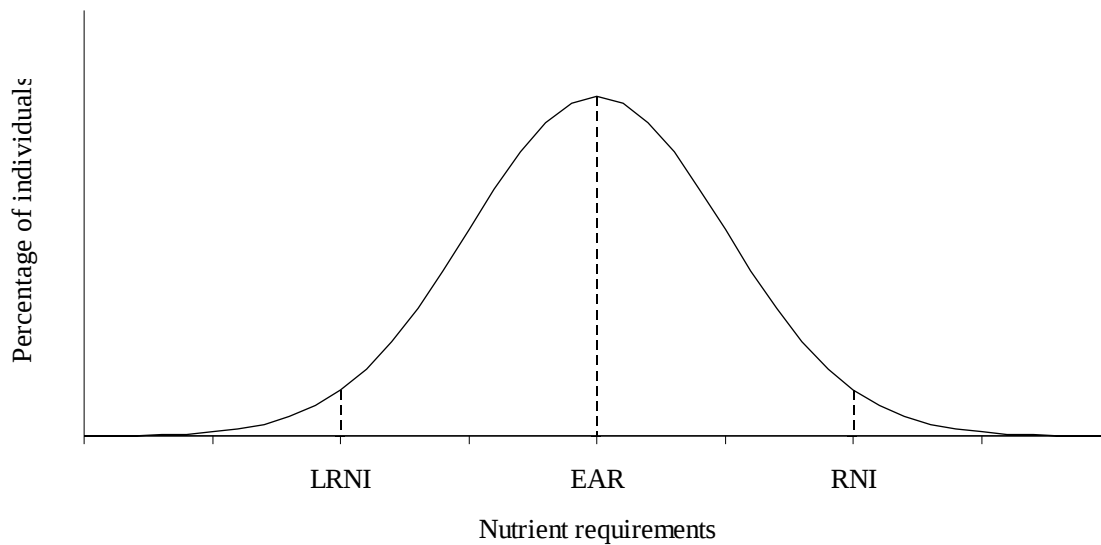
- Review and agree on the interpretation of the methods, definitions and assumptions used by Committee on Medical Aspects of Food Policy (COMA) and the FAO/WHO/UNU expert consultation to agree energy requirements.
- Agree a framework by which to arrive at energy requirements for the UK population and 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.

Dietary reference values

Definition

4. Information is usually inadequate to calculate the precise distribution of requirements in a group of individuals for a nutrient; however, it has been assumed to be normally distributed (Figure 1). A notional mean requirement or Estimated Average Requirement (EAR) with the inter-individual variability in requirements is illustrated in Figure 1. The EAR is the best statistical approximation of the nutrient requirement for any one individual in the population. The Reference Nutrient Intake (RNI) is defined as two notional standard deviations above the (EAR). For any individual intake above this amount will almost certainly be adequate. The Lower Reference Nutrient Intake (LNRI) is defined as two notional standard deviations below the mean and represents the lowest intakes which will meet the needs of some individuals in the group. For most individuals intakes below this level are almost certainly inadequate for most individuals .
5. The RNI is equivalent to the 1969 Recommended Daily Intake – that is the amount sufficient or more than sufficient to meet the nutritional needs of practically all healthy persons in a population, and therefore exceeds the requirements of most . At higher levels of consumption there may be evidence of undesirable effects. The revised nomenclature, however, emphasizes the true nature of what are estimates of reference values rather than recommendations for intakes by individuals or groups.
6. Setting the RNI at a notional two standard deviations above the EAR might, in theory, be perceived as leaving up to 2.5 per cent of the population inadequately provided for, but this is unlikely to be so in practice. In any population choosing spontaneous diets it is likely that, while the distribution stays roughly the same, the individuals comprising the extremes will vary, so that consistent intakes at the extremes are unlikely .

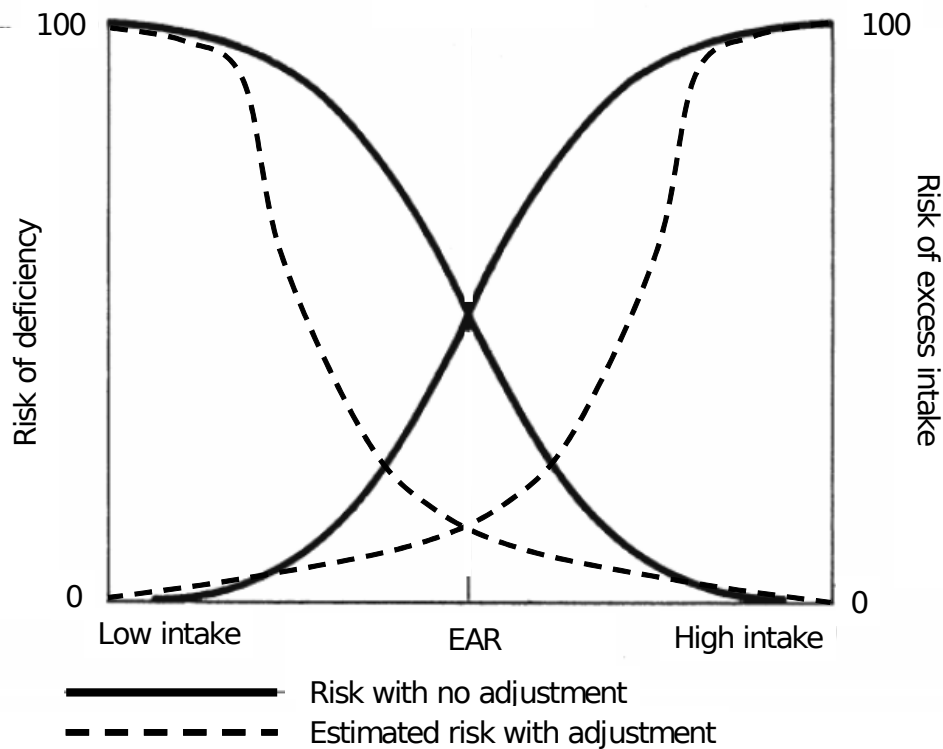
Figure 1. Assumed frequency distribution of individual nutrient requirements



Energy dietary reference values

7. Energy requirements are affected by age, gender, body size and composition, pregnancy, lactation and physical activity. These characteristics are used to define population groups for whom DRV are given.
8. RNIs for most specific nutrients, except energy, can be set at the upper end of the range of requirements to reduce the risk of deficiency in an individual because an intake moderately in excess of requirements has no adverse effects. For energy, however, this is not the case. Energy intakes that exceed requirements will produce a positive energy balance and could lead to obesity in the long term. An RNI for energy intakes would result in a high probability of energy excess and, hence, obesity. The EAR is the point at which the probability of excessive energy intake is equal to the probability of insufficient energy intake, in a random individual within the population (see Figure 2). If, as is likely, there is some correlation between intakes and requirement then the risk of both deficiency or excessive intake for an individual with an intake equal to the EAR is likely to be considerably less than 50%. Such a correlation between energy requirements and intakes is likely because of the physiological control of intakes through the appetite mechanism which acts to some extent to match intakes with expenditure. Expenditure can vary through body size and through different levels of physical activity. While the exact extent of the correlation between requirements and intake and consequent reduction in the risk of deficiency and excess intake at an intake equivalent to the EAR cannot be calculated with any certainty, the dotted lines in fig 2 show the likely adjusted risk curves.

Figure 2. Probability of excessive intake or deficiency in a random individual at a given energy intake level



9. Recommendations for energy, therefore, have always been set as the average of energy requirements for any population group. The EARs for energy, but not LRNIs or RNIs, have been calculated .

Food energy

10. Ingested food contains chemical energy – the maximum amount can be determined, by measuring the heat produced after complete combustion to carbon dioxide and water, in a bomb calorimeter. Not all combustible energy is available to human metabolism.
11. Incomplete digestion of food in the small intestine, in some cases accompanied by fermentation of unabsorbed carbohydrate in the colon, results in losses in gases (e.g. hydrogen and methane) and the faeces. Short-chain fatty acids are formed in the process, some of which are absorbed and available as energy. Most of the energy that is absorbed is available to human metabolism; however, due to the incomplete catabolism of protein some is lost as urea in the urine. A small amount of energy is also lost from the body surface. The energy that is available to human metabolism is termed metabolizable energy and is the value quoted in the UK food composition tables .
12. The body is able to capture some of the energy from food, through cytoplasmic glycolysis and mitochondrial respiration, resulting in the generation of an intermediary chemical form – the high energy pyrophosphate bond of adenosine triphosphate (ATP). Enzymatic hydrolysis of the high energy bond produces adenosine diphosphate (ADP), phosphate and releases

energy, which when coupled to various chemical reactions drives them to completion. ATP, therefore, acts as an energy source for cellular processes and is regenerated from ADP using the energy in food. Cells require chemical energy for three general types of tasks: to drive metabolic reactions that would not occur automatically; the transport of substances across cell membranes; and mechanical work, e.g. muscle contraction. Energy is also released as heat in these metabolic processes, which maintains body temperature.

13. Not all metabolizable energy is available for the production of ATP. Some energy is utilized during the metabolic processes associated with digestion, absorption and intermediary metabolism of food and can be measured as heat production; this is referred to as diet-induced thermogenesis, or the thermic effect of food, and varies with the type of food ingested. When the energy lost to microbial fermentation and diet-induced thermogenesis are subtracted from metabolizable energy, the result is an expression of the ATP-producing capacity of foods, and has been termed the net metabolizable energy .

Energy yields from substrates

14. The unit of energy in the International System of Units (SI) is the joule (J) and is the energy expended when 1 kg is moved 1 m by a force of 1 Newton. Nutritionists, physiologists and food scientists are concerned with large amounts of energy and the convenient units are the kiloJoule (kJ = 10^3 J) and the megaJoule (MJ = 10^6 J). The calorie is a derived unit and a thermochemical calorie is equivalent to 4.184 J (1 kcal = 4.184 kJ).
15. The macronutrients, carbohydrate, fat, protein and alcohol, provide the energy supplied by foods. The metabolizable energy value of a food or diet is calculated by applying conversion factors for each macronutrient (see Table 1) to the amount of substrates determined by chemical analysis or estimated from food composition tables. These conversion factors are estimates of the energy content of each macronutrient and have been rounded-up for practical purposes.

Table 1. Metabolizable energy conversion factors .

	kJ/g	kcal/g
Protein	17	4
Fat	37	9
Available carbohydrate expressed as monosaccharide	16	3.75
Alcohol	29	7

16. The amount (weight) of carbohydrate to yield a specific amount of energy differs depending on the molecular form of the carbohydrate, due to the water of hydration in different molecules. If expressed as monosaccharide equivalent, 100 g of most disaccharides and starch each contain of 105 g and 110 g of anhydrous glucose respectively. The actual available energy content per unit weight, therefore, is different for these different forms of carbohydrate (i.e. 15kJ,(3.68 kcal/g) 16kJ (3.87 kcal/g) & 17kJ (4.16 kcal/g) for glucose, disaccharide and starch respectively). The FAO, however, has recommended that when carbohydrate is expressed as monosaccharide equivalents, a conversion factor of 16 kJ/g (3.75 kcal/g) should be used and when determined by direct analysis, it is expressed as the weight of the carbohydrate with a conversion factor of 17 kJ/g (4.0 kcal/g), the latter value being a likely average of the different forms of carbohydrate in food.

17. Other carbohydrates may also provide energy. Non-starch polysaccharides that are fermented in the colon are an energy source. A conversion factor of 8 kJ/g (2 kcal/g) has been suggested, as have conversion factors for organic acids (13 kJ/g; 3 kcal/g), and polyols (10 kJ/g; 2.4 kcal/g). The UK food composition table energy values only include carbohydrate expressed as monosaccharide. The COMA Dietary Reference Values report noted that diets rich in non-starch polysaccharides were associated with a lower apparent digestibility of fat and protein and that this apparent loss in energy intake was similar to the net gain in energy intake from certain non-starch polysaccharides.

Energy balance and storage

18. Energy balance is the difference between metabolizable energy intake and total energy expenditure. A neutral energy balance means that energy intake is equal to energy expenditure; a positive energy balance is when energy intakes are in excess of energy expenditure; and a negative energy balance is when energy intakes are insufficient for energy expenditure.

19. Energy is stored in the body in the form of triglyceride and glycogen within adipose tissue, liver, and skeletal muscle. Triglyceride present within adipose tissue is the body's major fuel reserve. The amount of energy stored in the adipose tissue of a healthy adult of normal weight provides a large buffer capacity and is equivalent to over a month's energy requirements.

20. An individual in negative energy balance mobilizes stored energy from triglyceride and glycogen; energy is also mobilized from protein in muscle and viscera. An individual in positive energy balance stores excess food energy mainly as triglyceride and glycogen. Muscle and liver glycogen stores are small and have a limited capacity; whereas, the capacity of the body to store triglycerides in adipose tissue appears almost limitless.

21. Short-term, day to day energy imbalances are mostly accommodated by the deposition and mobilization of glycogen. Long-term energy imbalances are mostly accommodated by the deposition and mobilization of adipose tissue triglycerides, together with small amounts of lean tissue. Substantial positive and negative energy balances occur in the short term in free living individuals, so it is important to consider the overall energy balance over a prolonged period of time.

Obesity

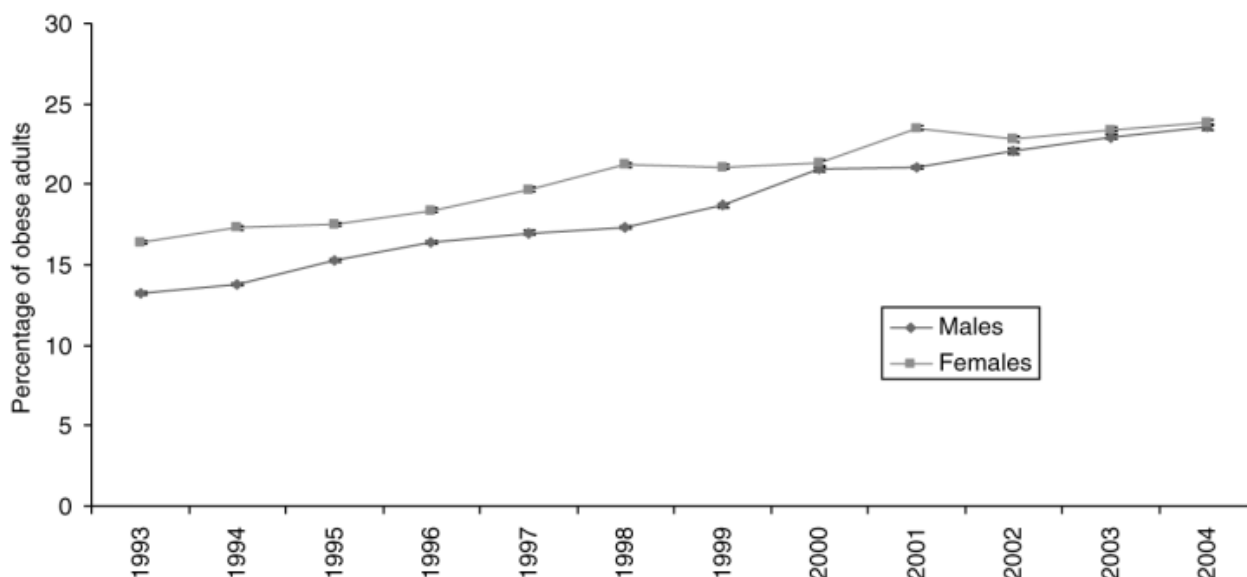
22. Problems relating to insufficient energy intakes are uncommon in the UK, and do not generally arise from insufficient food supplies, but from accompanying physical or psychological diseases. In contrast, there is a high prevalence of overweight and obesity in the population resulting from a chronic excess of dietary energy intake over energy expenditure. Overweight is defined as a Body Mass Index (BMI) in excess of 25.0 kg/m² and obesity is defined as a BMI in excess of 30 kg/m².

23. The prevalence of obesity in children aged under 11 in England increased from 9.9 % in 1995 to 13.7 % in 2003; 31.6% of boys and 30.7% of girls aged 2-15 years old were overweight or obese. If the current trends continue, 20% will be obese by 2010 – estimated

at over 1 million children.

24. The 2004 Health Survey for England estimates that 22% of men and 23.5% of women are obese; 67% of men and 57% of women are overweight. The prevalence of obesity has trebled since the 1980s, and almost 24 million adults are either overweight or obese. Obesity in both adults and children is more common among lower social groups (see Figure 1.).

Figure 1. Trends in obesity in England, 1993-2004 .



25. Obesity is an important risk factor for a number of diseases (see Table 2 for an overview) and is responsible for more than 9,000 premature deaths per year in England alone. The Health Select Committee has estimated the costs of obesity at £3.3 - £3.7 billion per year and of obesity plus overweight at £6.6 - £7.4 Billion. The National Audit Office estimates that one million fewer obese people in this country could lead to around 15,000 fewer people with coronary heart disease, 34,000 fewer people developing type 2 diabetes, and 99,000 fewer people with high blood pressure .
See

Table 2. Summary of associations observed in prospective studies between obesity and subsequent ill health .

Association for increased risk	Health 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)

	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 arising from maternal obesity

Components of energy requirements

26. The energy requirement of an individual has been defined by the FAO/WHO/UNU as ‘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 secretion of milk during lactation consistent with the good health of mother and child’ .
27. The energy requirements of an individual can be divided into a number of discrete components, which can be determined separately.

Basal and resting metabolism

28. A measure of an individual’s metabolism in a basal state is termed the basal metabolic rate (BMR) and is the minimal energy requirement needed to sustain life in a resting state, e.g. for the cellular and tissue functions required for the functioning of the heart, lungs, nervous system, liver, kidneys, sex organs, muscles and skin. BMR is the energy expenditure of an individual at mental and physical rest in a temperate neutral environment, in a post-absorptive state (at least 12 hours after eating) and without having performed heavy physical exercise on the preceding day. If the measure is conducted at least 6 hours after eating or performing a rigorous physical activity, the energy expenditure is termed resting metabolic rate (RMR), as all the conditions for BMR have not been met. BMR is usually the largest component and ranges from 45 to 70% of energy requirements depending on age and lifestyle. BMR includes a small component associated with arousal, as compared to sleeping metabolic rate.

Diet-induced thermogenesis

29. The metabolic processes associated with ingestion, digestion and absorption of food, and intermediary metabolism and deposition of nutrients increase heat production and oxygen consumption; this is referred to as diet-induced thermogenesis, the thermic effect of food or postprandial thermogenesis. This leads to an increase in energy expenditure for several hours after the ingestion of food and is assumed to be about 10% of the energy requirements.

Physical activity

30. This is the most variable component of energy requirements and the energy expended depends on the type and duration of activities, but, in most individuals, accounts for 20-40% of energy requirements. Due to differences in body size and skill there is a large inter-individual variation in the energy expended for a given activity. The energy cost of different physical activities are often expressed as multiples of BMR or RMR to account for differences in body size. The metabolic equivalent (MET) values give multiples of RMR (defined as multiples of an individual's resting oxygen uptake) and are usually expressed per hour per kg; the physical activity ratio (PAR) give multiples of BMR and are also usually expressed per hour.

Growth

31. During growth energy is deposited into new tissues and is required for the synthesis of growing tissues. The energy required for growth is highest in the first 3 months of life when it accounts for about 35% of energy requirements, by 12 months of age this has fallen to about 5% and by the second year is about 3%. Between 2 years of age and mid-adolescence 1-2% of energy requirements are required for growth, but by the late teens the amount is negligible.

Pregnancy and lactation

32. During pregnancy the energy requirements for placental and foetal growth are provided by the mother, as are the energy requirements for the growth of maternal tissues, e.g. uterus, breast and adipose tissue triglycerides. There are, therefore, increased energy costs from changes in maternal metabolism and a larger tissue mass, along with an increased energy cost of movement, particularly for weight bearing activities after 25 weeks.
33. During lactation energy is required for the energy content of secreted milk and the energy required producing the milk. Fat stores that accumulate during pregnancy provide part of this requirement.

Methodologies for estimating energy requirements

34. There are a variety of methods for estimating energy requirements and problems of interpretation are inherent in all methods. In the 1991 DRV report, measures of total energy expenditure in infants and children were used to validate the use of dietary intake data in the formation of energy requirements for these groups.

Measurement of energy intake

35. In the absence of sufficient information on energy expenditure, measurements of energy intake, reported in surveys of healthy well-nourished subjects, have been used to estimate energy requirements, e.g. in infants and preschool children. The reported energy intakes of weight-stable subjects (i.e. those in neutral energy balance, where energy intake equals energy expenditure) could, in principle, be used to predict energy requirements for weight maintenance. This method is dependent on the accuracy of the dietary data.

36. A variety of dietary assessment instruments have been developed to assess dietary intake, including weighed food records, diet histories, 24-hour recalls and food-frequency questionnaires, each with many variations to suit particular investigative situations .
37. By comparing subjects' reported energy intake to their measured free-living total energy expenditure (TEE), the accuracy of food intake reporting has been assessed . Studies examining the accuracy of reported energy intakes, where TEE has been measured, have consistently shown under-reporting of energy intakes . The majority of self-reported dietary intakes are systematically biased toward the underestimation of energy intakes .
38. Under-reporting tends to increase as children grow older ; for younger children, reporting is the responsibility of a parent or carer, and there is likely to be less access to unsupervised eating. Under-reporting is more pronounced among overweight and obese, than among lean, adults and children . Low socioeconomic status has also been shown to increase the tendency to underreport energy intakes , as has subject-specific bias to dietary assessment . There also exists a small element of over-reporting within study populations . These issues complicate the interpretation of self-reported food consumption data for the determination of estimated energy requirements.
39. An assessment of the under-reporting of energy intake by self-reported dietary methods in the NDNS for adults compared estimated under-reporting using individualized estimates of energy requirements with a population cut-off based on minimum energy needs . Reported physical activity was used to assign each subject's activity level, and then to calculate estimated energy requirements (EER) from published equations. By the individual method under-reporting was approximately 27 % of energy needs in men and 29 % in women, with 75 % of men and 77 % of women classified as under-reporters; by the population method 80 and 88 % were classified as under-reporters respectively. When subjects who reported their eating being affected by dieting or illness during dietary recording were excluded, UR was 25 % of energy needs in both sexes. UR was higher in overweight and obese men and women compared with their lean counterparts.
40. Determining the energy intake of breast-fed and formula-fed infants is also not straightforward . Test weighing in breast-fed infants can be time consuming, invasive in relation to the mother and infant, and is based on assumptions about the energy content of breast milk that may be inaccurate. The calculation of energy intake in formula-fed infants is easier if the child is being fed ready-to-feed formula, but if the formula is being made up by the mother or carer, large variations in energy density can be produced.

Measurement of energy expenditure

41. There are three components to TEE in humans: basal metabolic rate (BMR), diet-induced thermogenesis and the energy expenditure of activity (physical activity). There are three approaches used to measure energy expenditure. In indirect calorimetry, oxygen consumption and/or carbon dioxide production is measured and converted to energy expenditure using formulae. In direct calorimetry, the rate of heat loss from the subject to the calorimeter is measured. A number of non-calorimetric techniques have been used to predict the energy expenditure by extrapolation from physiological measurements, e.g. heart rate and accelerometry measures .

42. There are two variants on indirect calorimetry, closed-circuit spirometry and open-circuit spirometry. Open-circuit systems comprise components to collect and mix expired air, measure flow rate, analyse gas concentrations and pump air through the system; whereas, closed-circuit systems consist of a sealed respiratory gas circuit in which gaseous concentrations are measured over a short time. The closed circuit method was widely used during the first half of the 20th century, but has been shown to overestimate measures of BMR, relative to the open circuit method. Chamber-based systems are the most accurate open circuit method for the long-term measurement of specified activity patterns, but behaviour constraints mean they do not reflect real life.
43. The components of TEE can be measured separately using direct and indirect calorimetry, but not by using non-calorimetric methods. Although TEE can be measured by some calorimetric methods it is usually only in confined subjects or, in the case of open-circuit expiratory collection, measures may be imprecise. Non-calorimetric methods allow TEE to be estimated in free-living subjects. The use of the stable isotope technique, doubly labelled water (DLW), enables TEE to be measured accurately in free-living subjects – other non-calorimetric methods, e.g. heart rate monitoring, are less accurate. Calorimetric methods are less applicable to large scale studies over periods of several days. The use of DLW techniques provides an indirect measure of energy expenditure in free living individuals integrated over days and weeks.
44. In the doubly labelled water technique, the subject consumes water containing a known concentration of the stable isotopes of hydrogen (^2H) and oxygen (^{18}O), based on their body weight. The isotopes mix with normal hydrogen and oxygen in body water within about 5 hours. As energy is expended in the body, CO_2 and H_2O are released. Labelled hydrogen leaves the body as water ($^2\text{H}_2\text{O}$) in sweat, urine, and evaporation from the lung, while labelled oxygen leaves as both water (H_2^{18}O) and carbon dioxide (C^{18}O_2). The isotope concentration of a subject's urine or saliva is determined by isotope ratio mass spectrometry. The difference between the elimination rates of the two isotopes relative to the baseline levels reflects the rate at which CO_2 is produced from metabolism. Oxygen consumption, and energy expenditure, can be estimated based on an assumed (or measured) respiratory quotient value (ratio of CO_2 produced to the O_2 consumed), which is determined by substrate oxidation.
45. The technique is based on the assumptions about the amount of water lost from the body by evaporation and the extent of incorporation of ^2H and ^{18}O into body tissues, especially during growth. This technique, however, provides a direct measure of TEE and is the most accurate available measure in free-living subjects. The TEE is the energy expended during a time period and, therefore, 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. The TEE does include the energy required for tissue synthesis during a time period.

Calculation of energy requirements

46. As discussed above, estimates of energy requirements can be based on energy intake data. If sufficient data are available, however, energy requirements are based on measures of energy expenditure, as these provide a more reliable basis for estimating energy requirements than

energy intake data . Energy requirements have previously employed a factorial approach based on calorimetric measures of the components of TEE: BMR, diet-induced thermogenesis and the energy expended in specific physical activities. Diet-induced thermogenesis is normally included in any measurement or estimation of energy expenditure and is not assessed separately.

47. The compilation of calorimetric measures of BMR values and anthropometric data led to a series of predictive equations being developed for BMR based on body weight, age and gender . The BMR is predicted using these equations and used, in conjunction with an assessment of energy expended through physical activity, to obtain an estimate of energy requirements. The predicted BMR is multiplied by a factor that reflects the physical activity level (PAL) of the individual or group to give an estimate of energy requirements.
48. PAL is the ratio of TEE to BMR and is characterized by a description of lifestyle, e.g. non-active, moderately active, and very active. PAL is calculated from the duration of the various work and leisure activities, which are each assigned a specific energy cost (a PAR value; a multiple of BMR for a given activity), e.g. 4 x BMR for walking on the level at an average pace. The activities are multiplied by their corresponding PAR values to give total PAR hours of activity per day and the PAL. Tables of the energy costs of different physical activities given in PAR values have been compiled .
49. The main reason for using the BMR multiple approach to calculate energy requirements is that it is assumed to compensate for differences in body weight between individuals; however, energy expenditure in programmed work activities has been shown to be influenced by body weight and body fatness, suggesting that the assumed constancy of BMR multiples across a wide range of body weights might not be valid . The accuracy of the MET values to estimate the energy cost of physical activity, may also be affected by adiposity . Energy requirements derived from factorial approaches may, therefore, be biased with respect to the body fatness of subjects, as it assumes that physical activity energy expenditure is dependent entirely on BMR or RMR.
50. The dataset upon which the predictive equations for BMR were based was mostly obtained from results in West European and North American subjects, with almost half being Italian subjects in whom BMR was estimated using a closed circuit method in the 1930s and 1940s. Questions have been raised about the universal applicability of these equations . The predictive equations proposed in 1985 form the basis for the FAO/WHO/UNU energy requirements for adults and modified versions were used for the previous UK DRV for energy in children aged 3-18 years and adults .
51. The factorial approach only includes discrete conscious activities in the assessment of PAL and the energy costs of all activities may not be available, which may lead to underestimation. Measures of free-living TEE do include all activities, but do not give a direct measure of the energy expenditure of physical activity or BMR. It has been suggested that the factorial approach is not based on a sound physiological model of TEE, which should be the sum of its components and not multiples of one component .
52. A more recent approach to estimating energy requirements uses multiple regression techniques to develop prediction models of total energy expenditure as a function of measured predictor variables, such as body weight, age, height etc. This approach has been

developed by the compilation of measures of free-living TEE and their potential predictor variables .

53. The development of predictive equations from studies measuring TEE forms the basis of FAO/WHO/UNU energy requirements for infants, children and adolescents . For infants, DLW studies were used, and for children and adolescents DLW and heart rate monitoring studies were used to develop datasets on which the equations were derived. The USA Dietary Reference Intakes for energy were based on datasets of DLW measures of TEE (normal weight and overweight/obese datasets) for all age/gender groups; the TEE results were presented in units of PAL . The USA energy requirements are based on a dataset of DLW measures comprising individual TEE and ancillary data obtained directly from investigators of each study; whereas, the FAO/WHO/UNU dataset of TEE studies used mean values from each study, and weighted the results on the number of subjects, to derive the predictive equations.

Methodologies used to determine the Dietary Reference Values for energy

To be determined

Infants

Children and adolescents

Adults

Pregnancy and lactation

Factors affected energy expenditure

Alcohol

54. Alcohol oxidation starts instantly after intake, and alcohol is eventually completely eliminated by oxidation. Consumption of alcohol can activate the hepatic *de novo* lipogenesis pathway modestly, but acetate produced in the liver and released into plasma inhibits adipose tissue lipolysis, alters tissue fuel selection, and represents the major quantitative fate of ingested ethanol. The results from studies on the magnitude of diet-induced thermogenesis after alcohol consumption vary, with reported values ranging between 9% and 28%. One long-term study, however, reported similar metabolizable energy after alcohol and carbohydrate, so discrepancies exist.

[DN: should the effect of other macronutrients on DIT be included? Also should it be included in the DIT or food energy sections, e.g. as net metabolizable energy, rather than here?]

Body composition and size

55. Body size and weight exert marked effects on energy expenditure. The metabolically active tissue mass of the body, termed fat-free mass (FFM), comprises the organs (e.g. digestive tract, kidney, lungs, heart, liver, brain), which together constitute about 5% of body weight and contribute around 60% to the energy expended by FFM, and muscle, which constitutes about 35% of body weight and is responsible for the remaining 40%.

56. FFM is the principal determinant of BMR and RMR and the inter-individual variation in BMR and RMR. Fat mass (FM), which is less metabolically active than FFM, has been observed to account for a small amount of the inter-individual variation in BMR and RMR in most studies, but not all.

57. Metabolically, FFM is a heterogeneous compartment. Organ size, as determined by magnetic resonance imaging, explains a greater proportion of the variation in RMR than total FFM. Resting energy expenditure per kg FFM is, therefore, not constant; resting energy expenditure per kg FFM decreases with increasing body weight because of a disproportional increase of muscle mass. Variation in the composition of FFM may account for a small amount of the inter-individual variation in BMR and RMR.

58. It has been suggested that, on a population basis and up to a moderate level of fatness (BMI < 30 kg/m²), the relative proportions of FFM and of FM are unlikely to influence energy metabolism at rest or while physically active in ways other than through their impact on body weight. In adults with higher percentages of body fat composition, an effect on the mechanical efficiency of movement can increase the energy expenditure associated with certain types of activity. The energy expenditures for weight-bearing activities have been observed to be affected by body mass. Physical activity-related energy expenditure (PAEE) and its relation to body mass and composition depend on whether the activity is weight-bearing or not; different activities have different weight-bearing impacts on PAEE.

59. Body mass and body composition have been shown to impact on PAEE and TEE in children and adults.

60. Gains in body weight and percent fat mass are the consequence of a positive energy balance over time. 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, both FM and FFM . This observed increase in energy expenditure was not in direct proportion to body weight since, when expressed per kg, both TEE and PAEE declined significantly with increasing BMI .

Physical activity

61. Physical activity is defined as any bodily movement produced by the contraction of skeletal muscles resulting in energy expenditure . Physical activity in relation to energy balance is discussed in Chapters 7 and 9. PAEE is the most variable component of TEE. Analysis of energy expenditure measures has established the extremes of human daily energy expenditure: from a TEE of about 1.2 x BMR (PAL value of 1.2) in non-ambulant subjects to a TEE of about 4.5 x BMR (PAL value of 4.5) in elite endurance athletes . Within the general population the upper limit for PAL values is thought to be 2.5

62. Occupational, leisure and home activities are major determinants of PAEE . The role of planned exercise in raising TEE depends on the intensity and duration of the physical 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 decrease in another. Planned exercise has been observed to raise TEE in children and adults , but some studies, especially where the exercise was of a relatively low intensity, have not observed planned exercise to raise TEE despite improving physical fitness .

63. Many studies of human subjects indicate a short-term elevation in RMR in response to single exercise events (generally termed the excess post-exercise O₂ consumption; EPOC). This EPOC appears to have two phases, one lasting < 2 h and a smaller much more prolonged effect lasting up to 48 h .

Gender

64. Variation in energy expenditure among individuals is primarily determined by differences in FFM. Women have a lower percentage FFM than men, i.e. they have a higher percentage FM, so absolute energy expenditure is lower in women. After adjustment for body size and composition, BMR and TEE have been observed, in most studies, to be lower in girls and women; however, some studies have observed no differences in energy expenditure between sexes after adjustment for FFM in children and adults.
65. In premenopausal 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, although not all. 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. 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, but results from studies investigating the effect of contraceptive drug use on energy expenditure have been equivocal.

Age

66. BMR and RMR decline with older age and this is mainly attributable to the progressive loss of FFM observed with aging; however, a small decline in BMR with age, independent of any age-related changes in body composition, has been observed.
67. When the decline in body cell mass (the total mass of cells comprising the metabolically active tissue of the body) with age was measured, using the total body potassium technique, it was observed to fully account for the age-related decrease in RMR, whereas other measures of FFM did not. This suggests that elderly subjects may not have slower organ metabolic rates compared to younger subjects; however, one study applying an RMR-prediction model based on seven organ/tissue components to calculation of RMR in older men and women, reported that factors other than organ atrophy contributed to the lower metabolic rate of older persons (i.e. lower organ metabolic rates). In another study in healthy subjects, the age-related decline in resting energy expenditure was not associated with decreasing organ metabolic rate and was fully accounted for by both a reduction in FFM and proportional changes in its metabolically active components.
68. It is unclear, therefore, whether changes in the energy expenditure 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, decreased muscle protein turnover, and changes in mitochondrial membrane protein permeability. A difficulty encountered in studies of the effects of aging on the decline in BMR and RMR 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.
69. Physical activity energy expenditure has been observed to decline with aging, but the results from studies investigating an effect of aging on diet-induced thermogenesis have been

inconsistent .

Genetics

70. Genetic inheritance potentially influences all factors affecting inter-individual variation in energy expenditure, e.g. body size and composition, differences in BMR independent of body composition, ethnicity. A familial influence on RMR, independent of FFM, age, and sex, has been reported .
71. Studies have investigated the association between different genotypes and variation in energy expenditure. Several have investigated the role of the Trp64Arg polymorphism in the β_3 -adrenoceptor gene (*ADRB3*). Although one study observed a lower RMR, adjusted for lean body mass and age, in subjects with the *ADRB3* Trp64Arg genotype relative to Trp64Trp homozygotes , most have reported no association between the *ADRB3* Trp64Arg genotype and RMR , post-prandial energy expenditure and TEE .
72. A polymorphism that leads to a three-amino acid deletion in the α_{2b} -adrenoceptor was associated with a lower BMR in subjects homozygous for the short allele , but in a subsequent study no association was observed .
73. A polymorphism in the β_1 -adrenoceptor (Gly389Arg *ADRB1*) was not observed to be associated with RMR and no independent contribution of the Gly16Arg polymorphism of the β_2 -adrenoceptor gene (*ADRB2*) to the variation in thermogenic response to a high-carbohydrate meal was demonstrated either .
74. Other studies have investigated the role of genetic variation in the leptin receptor gene (*LEPR*) on energy expenditure. The *LEPR* Gln223Arg polymorphism was observed to be associated with TEE and PAL in one study – homozygotes for Arg223Arg had a lower TEE and PAL – but other studies failed to observe an association . RMR has been associated with the *LEPR* Lys656Asn polymorphism, with the Asn656Asn homozygotes having a higher RMR compared to the other two genotypes , but no association was observed in another study .
75. Several studies have investigated the association between the mitochondrial uncoupling protein gene (*UCP*) variants and energy expenditure. A polymorphism in *UCP1* gene (-3826A>G) was identified, but no association with RMR was observed . Other studies have also failed to observe an association between this polymorphism and energy expenditure , however, in subjects with both the *UCP1* -3826A>G and the *ADRB3* Trp64Arg genotypes a lower BMR, adjusted for FFM, age and sex, was observed relative to the subjects without these polymorphisms. A lower thermic effect of food after the high fat, but not a high carbohydrate, meal was observed in subjects with the *UCP1* -3826A>G genotype relative to the other genotypes .
76. A study of the linkage relationships between 3 microsatellite markers that encompass the *UCP2* gene location on 11q13 with RMR, concluded that they were linked to RMR . One study identified three polymorphisms, informative for association studies, in the *UCP2*-*UCP3* gene cluster . Heterozygotes for two *UCP2* gene variants (Ala55Val and a 45 base pair insertion/deletion in the 3'-untranslated region of exon 8) had higher sleeping metabolic rates, adjusted for fat-free mass, fat mass and sex, than homozygotes; the *UCP3* variant (C>T

silent polymorphism, Tyr99Tyr) was not associated with metabolic rate.

77. In subsequent studies, the *UCP2* Ala55Val polymorphism was not associated with BMR or RMR ; however, a lower sedentary TEE, adjusted for fat-free mass, fat mass, and spontaneous physical activity, was observed in the Val55Val homozygotes relative to the other genotypes . In a study of energy expenditure during bicycling, subjects with the Val55Val *UCP2* genotype had higher exercise efficiency across the three different work levels than subjects with the Ala55Ala genotype .
78. The 45-base pair deletion/insertion in 3'-untranslated region of *UCP2* was not observed to be associated with RMR in other studies , but was associated with sedentary TEE, adjusted for age, sex, family membership, FFM and FM , as was the *UCP2* variant -866G>A in the 5' upstream region.
79. RMR was observed to be lower in subjects with the *CC* genotype of the *UCP3* -55C>T polymorphism in one study , but another study found no association with this genotype ; however, a C>T silent polymorphism (Tyr210Tyr) in exon 5 of *UCP3* was observed to be associated with RMR in African American, but not white, women; *CC* homozygotes had a lower RMR, adjusted for FFM, FM, and smoking status, than those with the *CT* or *TT* genotype. This may reflect linkage disequilibrium for a functional variant elsewhere in the *UCP2* and *UCP3* gene regions in African Americans but not in whites.
80. Genetic variants of other genes have been investigated with regard to a role in energy expenditure. Several have found associations. A higher RMR was observed in women, but not men, who were heterozygotes for the Asn363Ser polymorphism in the glucocorticoid receptor gene (*GCCR*) relative to homozygotes . Subjects heterozygous for the interleukin-6 (*IL6*) gene -174C>G promoter polymorphism were found to have a lower RMR than homozygous subjects . The melanocortin-4 receptor (*MC4R*) Val103Ile genotype was associated with a higher RMR, adjusted for age, sex, and BMI, compared to the subjects with the Val103Val genotype .
81. Other studies have reported no association between RMR and genetic variants of the sodium-potassium ATPase genes (*ATP1A1*, *ATP1BL1*) or the Ala54Thr substitution in the intestinal fatty acid binding protein 2 (*FABP2*) gene .
82. There have, as yet, been no clearly established relationships between specific gene variants and energy expenditure, although potentially genetic inheritance is an important factor.

Ethnicity

83. Differences in body composition and FFM composition have been observed between different ethnic groups, e.g. between whites and blacks and whites and South Asians . RMR, BMR and sleeping metabolic rate, adjusted for differences in FFM and FM, have been reported to be lower in black subjects than white subjects in most studies , but not all . In one study a lower RMR and TEE was observed in African Americans after adjustment for FFM, but not trunk lean tissue ; equally, another study found a lower RMR, adjusted for FFM and FM, in African American subjects, but these differences were no longer evident after adjustment for regional FFM distribution . A racial difference in RMR, after adjustment for trunk lean tissue, was observed in one study, although this was decreased

from the association observed with adjustment for FFM . The racial differences in RMR, apparent after adjustment for FFM, sex and age, were no longer significant in studies that adjusted for the mass of specific high-metabolic-rate organs .

84. A lower TEE, adjusted for differences in FFM and FM, in African American subjects relative to whites has been reported , but other studies have observed no association with TEE . As mentioned above, one study that observed a lower and TEE in African Americans relative to whites, after adjustment for FFM, found this became non significant after adjustment for trunk lean tissue .
85. Differences have been observed between the BMR, adjusted for body weight, of South Asians and whites, but these became insignificant when BMR was adjusted for FFM and FM . Differences in body composition may, therefore, be mainly responsible for the reported differences in energy expenditure between ethnic groups.
86. Because of the lack of available evidence, it is not possible to make specific allowances for different ethnicities in the energy requirements.

Endocrine state

87. As discussed above sex hormones may affect energy expenditure. Other hormones have also been implicated in energy balance.
88. Thyroid status is a major determinant of metabolic rate. Hyperthyroidism increases while hypothyroidism decreases RMR . Physiological variations in plasma concentration of the thyroid hormone, tri-iodothyronine (T_3), have been associated with between- and within-subject variations in BMR or RMR, independently of FFM, in some studies , but not all . Serum concentration of thyroid hormones, RMR and TEE have been observed to decrease in some studies during energy restriction and weight loss and, during weight regain, increases in T_3 have been associated with increases in RMR, independently of FFM, in some studies , but not all .
89. Plasma noradrenaline concentration has been observed to be associated with RMR, adjusted for FFM , but not all studies have found this association .
90. 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 . Plasma leptin concentration is related to adiposity, but other factors also affect the inter-individual variability, e.g. women have higher plasma leptin concentrations, adjusted for FM, than men .
91. Some studies have found positive associations between plasma leptin concentrations and RMR, adjusted for FFM and FM , others have found negative associations or no significant association . Some of these differences may reflect problems accounting for the confounding effects of FM on plasma leptin concentrations . Plasma leptin concentrations were positively associated with TEE, adjusted for FM, in one study , but no association was found in other studies .
92. Plasma leptin concentration has been observed to decrease with acute starvation,

disproportionately to changes in FM and increase with subsequent weight gain .

93. Longitudinal studies of leptin secretion during nutritional rehabilitation in anorexia nervosa patients have not observed serum leptin concentration to be associated with RMR, adjusted for FFM, during weight gain .
94. Metabolic stress and fever have also been observed to increase BMR; this is discussed in more detail in Chapter 6 (the energy requirements for illness).

Pharmacological agents

95. Administration of pharmacological doses of leptin to overweight and normal subjects was observed to have no effect on RMR and did not reverse fasting-induced decreases in the thyroid hormones, cortisol, growth hormone, insulin-like growth factor-1 and RMR ; subsequently, it was suggested that leptin predominantly influences the human energy balance through appetite, but does not appear to be involved in regulating energy expenditure .
96. In subjects undergoing dietary energy restriction, low-dose leptin administration (restoration of circulating concentrations to levels that were present prior to weight reduction) was observed to have no effect on RMR, but the observed reduction in TEE after weight loss was attenuated in subjects receiving leptin . Low-dose leptin administration was also observed to reduce the decline in plasma T₃ concentration observed with dietary energy restriction and weight reduction . The effect observed on TEE in response to energy restriction was due predominantly to a reduction in the energy expended in physical activity, which may reflect an increased work efficiency of skeletal muscle in response to energy restriction . Low-dose leptin administration has been observed to reverse the decline in TEE and the increased work efficiency of muscle in response to energy restriction . The role of leptin on energy expenditure is, therefore, unclear.
97. Smoking has been shown to increase energy expenditure to a small extent; sympathoadrenal activation by nicotine is thought to be primarily responsible for this effect . Caffeine has also been shown to increase energy expenditure to a small extent and to have an additive thermogenic effect to nicotine . Administration of alcohol was not observed to have an acute effect on energy expenditure in one study , but chronic alcoholics have been observed to have a higher RMR, adjusted for FFM, than healthy social drinking controls and abstinence from alcohol reduced the alcoholics' RMR .
98. Administration of glucocorticoids , adrenaline , amphetamines and novel anti-obesity drugs have all been shown to increase energy expenditure. Growth hormone administration may also increase energy expenditure , but this may be partly explained by increased FFM . β -blockers (β -adrenergic antagonists) have been shown to reduce RMR .

Environment

99. In cold-exposed adult humans, decreases in body temperature are delayed by reducing heat loss via peripheral vasoconstriction and by increasing energy expenditure by shivering thermogenesis and increased muscular activity . The maintenance of indoor temperatures to within 20-25°C and the use of clothes to control body heat loss, however, mean that ambient

temperature remains relatively constant for most people in the UK and shivering thermogenesis is not a significant contributor to energy expenditure.

100. Most studies in adult subjects in the UK and Holland suggest that short-term variation (1-3 days) in ambient temperatures of between 16-28°C is inversely associated with energy expenditure. During the studies subjects are asked to execute the same daily activities protocol at different ambient temperatures.
101. The short-term exposure to mild cold (20-22°C relative to 26-28°C) has been shown to produce a small increase in BMR or sleeping metabolic rate in some studies, although most observe no significant increase. The short-term exposure to 16°C relative to 22-24°C was shown to produce a small increase in sleeping metabolic rate in one study, but not in others.
102. An increase in sedentary TEE has been observed after the short-term exposure to mild cold: both for 16°C relative to 22-24°C and for 20-22°C relative to 26-28°C. Skin and core temperatures were also observed to decrease in response to mild cold. One study, however, in the UK, observed an increased sedentary TEE at both 20°C and 30°C relative to 23°C and 26°C.
103. Overall, these studies suggest that the variation in energy expenditure due to differences in environmental temperature is about 2-5 per cent TEE.
104. Two studies in the USA have observed higher sedentary and free-living TEE and sleeping metabolic rate, adjusted for fat-free mass, in spring than in autumn. A study in Dutch adults reported seasonal changes in sleeping metabolic rate that could not be explained by changes in body composition, thyroid activity, or plasma leptin concentration. Sleeping metabolic rate was at its highest in the winter and lowest in the summer. No changes in TEE between seasons were reported.
105. It is not possible to make specific allowances for changes in ambient temperature, e.g. seasonal variation, in the energy requirements.

References

Physical Activity

Background

106. '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. Physical activity is a complex and multi-dimensional behaviour taking place in a variety of domains: in transportation, domestic life, occupation and recreation. The dimensions of a specific physical activity are defined as its volume, frequency, intensity, time and type. Exercise is a subset of physical activity that is planned, structured, and repetitive and has as a final or an intermediate objective the improvement or maintenance of physical fitness. Physical fitness is a set of attributes that are either health- or skill-related. The degree to which people have these attributes can be measured with specific tests.

Physical activity and chronic disease risk

107. Prospective studies in which occupational and leisure physical activity were assessed by questionnaire established that physical inactivity was an independent risk factor for coronary heart disease; people who were regularly active had up to half the risk of those who are sedentary.

108. Increased self-reported physical activity has also been associated with a reduced risk for type 2 diabetes, stroke, lung cancer, colorectal cancer, postmenopausal breast cancer and endometrial cancer.

109. The observational studies that have reported these associations have relied on self-reported physical activity, which does not provide accurate estimates of absolute amounts of activity and cannot, therefore, determine whether higher levels of activity-induced energy expenditure confer survival advantages.

110. Some prospective studies have used measures of cardiorespiratory fitness as a proxy for physical activity and have generally shown stronger associations with health outcomes than did studies with self-reported physical activity as the exposure. Cardiorespiratory fitness measures, however, do not describe the amounts and types of physical activity or the amount of energy expenditure associated with decreased disease risk; they may also be influenced by other environmental and genetic factors.

111. A prospective study in adults with a family history of type 2 diabetes (n=393), observed that an increase in objectively measured PAEE (heart rate monitoring) between baseline to follow-up (5.6 years) was associated with reduced metabolic risk factor concentrations (blood pressure, fasting triglycerides, HDL cholesterol, insulin, and glucose) independently of aerobic fitness. Both the non-exercise and exercise components of PAEE may be important determinants of longevity. The use of semiquantitative food frequency questionnaires, however, to assess non-exercise physical activity is limited.

112. In a prospective study of healthy older adults (n=302; aged 70 to 79 years) TEE was determined using the DLW technique and RMR by indirect calorimetry. Over an average of

6.15 years of follow-up, participants in the upper tertile of PAEE (PAL >1.78) had a reduced risk of all-cause mortality (HR 0.43, 95% CI 0.21-0.88; $P_{\text{trend}} = 0.02$) than those in the lowest tertile (PAL <1.57). Objectively measured free-living PAEE was strongly associated with lower risk of all-cause mortality in healthy older adults. The intensity and type of physical activity was not objectively measured, but physical activity questionnaires suggested that the proportion of individuals who reported high-intensity exercise and walking for exercise (both in terms of duration and intensity) was similar across tertiles of free-living activity energy expenditure. It was concluded that simply expending energy through any activity may influence survival in older adults, seemingly contradicting reports that exercise needs to be performed at a specific intensity to produce health benefits .

113. Randomized controlled trials have shown that exercise training programs reduce blood pressure , beneficially affect dyslipidemia and, in high-risk populations (e.g. with impaired glucose tolerance) reduce insulin resistance and prevent or postpone the onset of type 2 diabetes . Exercise-based cardiac rehabilitation programmes have been shown to be effective in reducing the risk of premature mortality in patients with coronary heart disease .

114. Overall, adults with higher levels of physical activity tend to have lower mortality risk than their unfit and inactive peers within the same body mass index group. Accumulating evidence suggests that higher levels of physical activity attenuate the health risks of obesity . Prospective evidence also suggests that body weight may have a greater influence on diabetes risk, and physical activity plays a greater role in cardiovascular disease risk .

115. In the UK, adults are recommended to have at least 30 minutes a day of moderate intensity physical activity (similar to brisk walking) for general health. It is also recommended that, for many people, it is likely that 45-60 minutes of moderate intensity physical activity a day would be needed to prevent obesity . For children and young people, a total of at least 60 minutes each day of at least moderate intensity physical activity is recommended, and at least twice a week this should include activities to improve bone health (activities that produce high physical stresses on the bones), muscle strength and flexibility.

Proportion of UK population reporting achieving physical activity recommendations

Adults

116. The Health Survey for England (HSE) reports on physical activity data collected since 1997 . In 2004, 6,692 completed questionnaires were obtained from respondents, 3,256 men and 3,436 women; 37% of men and 25% of women reported achieving the physical activity recommendations for adults.

Table 1. Proportion of adults aged 16 and over achieving the physical activity target, 1997, 1998, 2003, 2004, by age and sex in the HSE

	Age							Total
	16-24	25-34	35-44	45-54	55-64	65-74	75+	
	%	%	%	%	%	%	%	%
Men								
1997	49	41	37	32	23	12	7	32

1998	53	45	41	34	30	14	6	34
2003 (unweighted)*	53	44	41	37	32	17	8	35
2004 (unweighted)*	56	46	41	37	32	19	9	35
2003 (weighted)	52	44	41	38	32	17	8	36
2004 (weighted)	56	46	41	37	32	18	8	37
Women								
1997	26	26	29	24	19	8	5	21
1998	28	28	28	25	18	9	3	21
2003 (unweighted)*	30	29	30	30	23	13	3	24
2004 (unweighted)*	31	31	33	29	19	14	4	24
2003 (weighted)	30	29	30	31	23	13	3	24
2004 (weighted)	32	30	32	30	20	14	4	25
Base								
Men								
1997	492	739	740	694	535	455	243	3898
1998	875	1338	1305	1289	987	837	562	7193
2003 (unweighted)	744	1024	1260	1098	1097	807	551	6581
2004 (unweighted)	291	446	535	439	508	378	276	2873
2003 (weighted)	1044	1272	1412	1180	1037	731	501	7177
2004 (weighted)	485	556	647	530	477	329	231	3256
Women								
1997	560	916	833	806	585	545	439	4684
1998	1006	1630	1573	1484	1148	967	907	8715
2003 (unweighted)	886	1279	1615	1278	1304	948	900	8210
2004 (unweighted)	364	550	746	626	621	482	429	3818
2003 (weighted)	1029	1279	1437	1199	1071	813	782	7611
2004 (weighted)	472	563	653	541	491	364	353	3436

* Data from 2003 onwards have been weighted for non-response. Unweighted data for 2003 onwards are provided for consistency with previous years which are also unweighted.

For data comparability across HSE years, only activity sessions that lasted at least 30 minutes were included.

117. The HSE reports on trends in physical activity from 1997, 1998, 2003 and 2004. The questionnaires used in the 2003 and 2004 survey were shorter than the surveys in 1997 and 1998, therefore, results from these two years have been recalculated to allow for comparisons to be made. For both men and women, the proportion reporting achieving physical activity recommendations has increased overall, from 32% in 1997 to 35% in 2004 for men, and from 21% to 24% for women. This perhaps presents a paradox given the rising prevalence of obesity.

118. For both men and women in 2004, the proportion reporting meeting the guidelines decreased steadily with age, from 56% of those aged 16 to 24, to 9% for those aged 75 and over among men. The proportion of women reporting meeting the guidelines remained stable for women aged 16 to 54 (29-33%) and decreased thereafter to 4% among women 75 and over. Men aged 16-24 are the group most likely to report meeting physical activity targets and are also the least likely group to be obese.

119. In the 1991 to 1994 HSE, physical activity was examined as occasions, rather than days (as in 1997 to 2004), that respondents took part in heavy domestic (heavy housework, heavy

DIY/gardening), walking, and sports (see the Temporal trends in physical activity section below).

120. The Allied Dunbar National Fitness Survey in 1990 assessed reported physical activity patterns and fitness levels of adults in England, achieving a sample size of over 4,000 people. Similar to the HSE, the survey measured levels of participation in sport and active recreation, physical activity in housework, DIY and gardening in the previous four weeks. Activities were classified as either light, moderate or of vigorous intensity. A six point activity level scale was then devised using information about duration, frequency and intensity. Physical activity targets were defined for different age groups based on varying levels of intensity and activity, which lasted 20 minutes or more. The main findings from the survey were that 7 out of 10 men and 8 out of 10 women reported falling below their age appropriate activity level necessary to achieve a health benefit.
121. The NDNS of adults aged 19-64 years, 2000/1, was the first of the NDNS series to collect data on physical activity. The HSE used a seven-day recall method to assess physical activity, compared with the NDNS, which used a seven-day diary; also, activities lasting less than 15 minutes were excluded in the HSE, while activities lasting less than 10 minutes were excluded in the NDNS.
122. Complete seven-day physical activity diaries were obtained from 1,658 respondents, 741 men and 917 women in the NDNS. Overall, 36% of men and 26% of women reported spending 30 minutes or more per day in activities of at least moderate intensity on five or more days, which is very similar to the HSE results. For men, the proportion who reported achieving the physical activity recommendation decreased with age, whereas, for women, there were no significant age differences (see Table 2).

Table 2. The proportion of adults aged 19-64 achieving the physical activity target, 2000/1, by age and sex in the NDNS

	Age				Total
	19-24	25-34	35-49	50-64	
	%	%	%	%	%
Men	49	46	34	24	36
Women	29	30	25	22	26
Base					
Men	104	211	243	243	801
Women	100	202	305	249	857

Children and adolescents

123. The HSE 2002, shows 70% of boys and 61% of girls report meeting current physical activity guidelines for children (achieving 60 minutes or more of at least moderate intensity physical activity a day). The HSE 2002 was the most recent data available with a large enough sample size for children to carry out a detailed analysis.
124. Information from the HSE 2002 is presented on the reported participation in out of school physical activity and the physical activity levels of children aged 2 to 15. Data on the

physical activity that children do as part of the school curriculum is available from the School Sport Survey .

125. The structure of the physical activity questionnaire was considerably different between 1997 and 2002. To make 2002 data more comparable with 1997 data, only activities that lasted at least 15 minutes were included in the trend analysis. In addition, all walking and housework and gardening sessions in 2002 were capped at 15 minutes to make the data more comparable with 1997.
126. There were no significant differences in the proportions of boys and girls aged 2 to 10 and 11 to 15 reporting meeting the physical activity target of at least 60 minutes of activity each day between 1997 and 2002. Differences do emerge, however, when considering the proportion of those who did report at least 30 minutes of activity each day of the week. For example, the proportion of boys aged 2 to 10 who reported between 30 and 59 minutes activity each day of the week increased from 9% in 1997 to 20% in 2002. For girls aged 2 to 10 there was an increase from 10% to 21% over the same period.
127. Self-report methods, such as questionnaires, are impractical in children because they are likely to be inaccurate . Objective measures overcome some of the problems of measuring physical activity in children; as a result, accelerometry-based physical activity monitors have become increasingly popular as a means of assessing physical activity in children.
128. Physical activity has been objectively measured, using accelerometry, in 11 to 12 year olds (n=5,595) from the southwest of England . The median time spent in moderate to vigorous physical activity in this study is 20 minutes per day (boys, 25 minutes per day; girls, 16 minutes per day) and there was substantial intra-individual variation in the measures physical activity of these children . Overall, only 5.1% of boys and 0.5% of girls achieved the current recommended level of activity, which is much lower than the HSE estimate. These figures were also lower than those reported for European and North American children , but these differences may be caused by the use of different cut-points of accelerometer counts to define the lower threshold of moderate-intensity activity.
129. Using accelerometers, it was also found that children from a deprived inner city school were active at recommended levels and had similar levels of activity to children in other studies from more affluent populations . Overall, accelerometry studies , although dependent on the threshold used to define moderate physical activity, show much lower levels of physical activity in children than suggested by questionnaire data.

UK population PAL values

130. In the absence of any reliable information and based on the assumption that much of the population had an inactive lifestyle, the previous DRV for food energy report used a PAL value of 1.4 to calculate the adult EAR . Children aged 10-18 years were assumed to have slightly higher PAL values of 1.56 for boys and 1.48 for girls; this was based on provisional information on the time use and energy cost of activities in the school day. For younger children insufficient information was available.
131. It has been suggested that the energy costs of activity used in the report to calculate energy expenditure underestimate those achieved during active leisure in individuals who take

regular exercise and that PAL values for active subjects tend to be higher than those assumed in the report .

Adults

132.It was not possible to derive PAL values from the HSE because the survey did not record the amount of reported time spent on all types of activity. PAL values for adults aged 19-64 years were derived from the seven-day physical activity questionnaire in the NDNS (see Table 3); BMR was calculated using the modified Schofield equations.

Table 3. Distribution of PAL values for adults aged 19-64 years derived from seven-day physical activity questionnaire in the NDNS

	Age				Total
	19-24	25-34	35-49	50-64	
Men					
Mean PAL	1.93	1.94	1.86	1.77	1.85
	%	%	%	%	%
1.3	0.00	0.00	0.00	0.00	0.00
1.4	0.00	0.00	0.71	1.85	0.86
1.5	0.00	2.78	1.41	1.39	1.58
1.6	20.37	10.42	13.78	21.30	15.93
1.7	22.22	28.47	34.28	36.57	32.86
1.8	9.26	12.50	9.89	11.57	10.90
1.9	5.56	6.25	4.24	5.09	5.02
2.0	5.56	9.72	11.66	4.17	8.46
2.1	12.96	4.86	5.65	6.02	6.17
2.2	5.56	5.56	4.59	5.09	5.02
2.3	1.85	0.69	1.77	0.93	1.29
2.4	1.85	3.47	1.77	0.46	1.72
2.5	3.70	2.08	3.18	0.93	2.30
>2.5	11.11	13.19	7.07	4.63	7.89
No of subjects	54	144	283	216	697
Women					
Mean PAL	1.72	1.71	1.72	1.66	1.70
	%	%	%	%	%
1.3	0.00	0.53	0.00	0.00	0.12
1.4	0.00	0.00	0.00	0.00	0.00
1.5	1.52	1.60	1.17	3.08	1.87
1.6	28.79	26.20	28.45	33.85	29.63
1.7	33.33	33.69	33.14	40.00	35.36
1.8	13.64	16.04	12.32	9.62	12.41
1.9	6.06	6.95	7.33	6.15	6.79
2.0	6.06	6.95	9.68	5.00	7.38
2.1	6.06	3.74	2.64	1.54	2.81
2.2	0.00	1.07	1.76	0.38	1.05
2.3	1.52	1.60	0.59	0.00	0.70
2.4	1.52	0.00	1.47	0.00	0.70
2.5	0.00	0.00	0.29	0.38	0.23
>2.5	1.52	1.60	1.17	0.00	0.94
No of subjects	66	187	341	260	854

Percentages may not add to 100 due to rounding

Table 4. Distribution of PAL values by BMI category for women aged 19-64 years derived from seven-day physical activity questionnaire in the NDNS

	Percentages				Total
	Underweight	Normal	Overweight	Obese	
Mean PAL	1.74	1.70	1.71	1.68	1.70
1.3	0	0	0	0	0
1.4	0	0	0	0	0
1.5	2	1	3	2	2
1.6	28	29	26	37	30
1.7	34	34	35	39	35
1.8	11	13	16	7	12
1.9	8	9	6	5	7
2.0	6	8	7	7	7
2.1	4	3	3	1	3
2.2	2	1	1	1	1
2.3	0	1	1	0	1
2.4	4	0	1	1	1
2.5	0	0	1	0	0
>2.5	2	1	1	1	1
No of subjects	53	338	287	176	854

Percentages may not add to 100 due to rounding

Table 5. Distribution of PAL values by BMI category for men aged 19-64 years derived from seven-day physical activity questionnaire in the NDNS

	Percentages				Total
	Underweight	Normal	Overweight	Obese	
Mean PAL	1.76	1.88	1.83	1.87	1.85
1.3	0	0	0	0	0
1.4	0	0	1	2	1
1.5	10	1	2	1	2
1.6	25	14	16	17	16
1.7	20	32	36	30	33
1.8	15	11	11	10	11
1.9	10	5	6	3	5
2.0	15	7	8	11	8
2.1	0	6	5	8	6
2.2	0	8	3	5	5
2.3	0	2	1	1	1
2.4	0	3	1	1	2
2.5	0	3	2	3	2

>2.5	5	7	8	8	8
No of subjects	20	204	305	168	697

Percentages may not add to 100 due to rounding

133. Participants recorded time spent sleeping, at work and participating in listed leisure, sports and other activities. The total time spent in sleep, light, moderate and vigorous intensity activities was multiplied by a metabolic equivalent (MET) value to give total MET hours of activity per day. The MET values used were: sleep 1.0; light 2.0; moderate 3.5; vigorous 6.0. These were summed to give a mean MET score for the week from which PAL values were derived.

134. An unpublished validation study for the seven-day physical activity questionnaire was conducted prior to the main adult survey in a sample of 66 adults. This study included a DLW assessment of energy expenditure as well as dietary and activity records. PAL values for each subject were derived from both seven-day activity records and from energy expenditure derived from DLW measurements (see Tables 6 & 7); BMR was calculated using the modified Schofield equations.

Table 6. Distribution of PAL values for adults aged 19-64 years derived from validation study energy expenditure data [DN: awaiting graphs with numbers rather than %]

	19-64 years		
	Men	Women	Men & Women
Mean PAL	1.88	1.74	1.79
	%	%	
1.3	0.00	0.00	0.00
1.4	4.35	6.98	6.06
1.5	0.00	11.63	7.58
1.6	13.04	9.30	10.61
1.7	13.04	16.28	15.15
1.8	17.39	16.28	16.67
1.9	13.04	13.95	13.64
2.0	13.04	11.63	12.12
2.1	4.35	11.63	9.09
2.2	0.00	2.33	1.52
2.3	8.70	0.00	3.03
2.4	4.35	0.00	1.52
2.5	8.70	0.00	3.03
>2.5	0.00	0.00	0.00
No of subjects	23	43	66

Percentages may not add to 100 due to rounding

PAL = TEE / BMR (calculated)

Table 7. Distribution of PAL values for adults aged 19-64 years derived from validation study activity records

	19-64 years		
	Men	Women	Men & Women
Mean PAL	1.88	1.74	1.79
	%	%	
1.3	0.00	0.00	0.00
1.4	0.00	0.00	0.00
1.5	0.00	5.13	3.33
1.6	23.81	30.77	28.33
1.7	38.10	43.59	41.67
1.8	9.52	10.26	10.00
1.9	9.52	5.13	6.67
2.0	9.52	5.13	6.67
2.1	0.00	0.00	0.00
2.2	0.00	0.00	0.00
2.3	4.76	0.00	1.67
2.4	0.00	0.00	0.00
2.5	0.00	0.00	0.00
>2.5	4.76	0.00	1.67
No of subjects	23	43	66

Percentages may not add to 100 due to rounding

PAL = TEE / BMR (calculated)

135. The mean PAL values derived by the two methods were similar at 1.8-1.9 for men and 1.7 for women; however, the correlation between the individual PAL values derived from DLW measurements and from activity records, for the 60 individuals who took part in all components of the validation study, was weak and did not reach statistical significance.

136. Considering that only 36% of men and 26% of women report meeting the 30 min physical activity recommendation, the NDNS PAL values based on the activity records seem high, although mean PAL values are in agreement with those derived from the validation study.

137. The PAL values obtained from the NDNS, an average of 1.85 for men and 1.70 for women, are for fairly active people. Moderate intensity activity of approximately 45 to 60 minutes per day, which has been recommended as required to prevent the transition to overweight or obesity, which has been suggested to correspond to an approximate PAL value of 1.7.

138. The discrepancy between the PAL values and the percentage meeting the physical activity recommendation could be due to relatively light physical activities that were recorded and contributed to PAL, but not to achieving the physical activity recommendation. The NDNS methodology is thought to overestimate activity in people with low activity levels.

139. It has been suggested that many physical activity questionnaires have an arbitrary grading in their classification of relative activities and do not necessarily reflect the true total energy expenditure, which results in an overestimation.

140. The NDNS data did not show any consistent differences between PAL values of different weight categories: underweight, ideal, overweight and obese (see Tables 4 & 5).

Children and adolescents

141.No survey data of PAL values for children and adolescents are available.

Temporal trends in physical activity in the UK

142.In the UK, there is no dedicated surveillance system for population-level physical activity to enable the elucidation of patterns and trends in physical activity at the national level. The information that is available on population-level physical activity trends mostly stems from ecological proxy domain-specific measures . For example, there has been as a massive change in the proportion of the workforce employed in manufacturing, farming and other physically-demanding occupations; car ownership has increased; and the distance children and adults walk or cycle per year has declined . It could be that the apparent temporal changes in activity in domestic life, work and travel are compensated for by an increase in recreational activity, but this information is not available for the UK.

143.In 2005, the three main activities of men and women in Great Britain were sleeping, working in their main job and watching television and videos/DVDs or listening to music . The *Time Use Survey* observed that people were less likely to spend time on housework in 2005 than in 2000, but, generally, the pattern of time use was very similar between 2005 and 2000.

144.The HSE has been collecting multiple-domain physical activity data, since 1991. An analysis of the HSE physical activity data was performed to estimate temporal trends of participation in physical activity between 1991 to 2004 , a period when the prevalence of obesity in adults living in England rose by over 65% . The study found a consistent upward trend for regular participation and time spent in sports, but a decline in occupational physical activity between 1991/2 and 2004. The decrease in reported high physical activity levels at work was more pronounced from 1998 and onwards. Increases in time spent in sports were evident in all age groups, but particularly pronounced among the middle and late life age groups (aged 35 to 49 and older).

145.The data were deemed insufficient, however, to provide a definitive answer as to whether overall physical activity has changed between 1991 and 2004. Fluctuations in time spent at moderate to vigorous physical activity and the percentage of men and women meeting the physical activity recommendations were more likely a function of changes in the questions included in different surveys and the imposition of certain analytical assumptions these changes forced, rather than a reflection of true changes in the physical activity levels of the population . It was concluded that there is an urgent need for a carefully designed UK physical activity surveillance system that includes objective measures of physical activity.

146.The Health Behaviour in School-aged Children study (a WHO cross-national survey) of 11-, 13- and 15-year-old schoolchildren has been carried out in Scotland and Wales since 1986 at four-yearly intervals . In Scotland and Wales, there was an increase in the percentage of schoolchildren who reported undertaking vigorous physically active at least four times a week between 1985/86 and 1997/98, followed by a decline in 2001/02. The proportions reporting watching 4 hours of television daily changed little between 1985/86 and 1997/98 as did the Children in Wales were most likely to report higher levels of TV watching across all four surveys.

147.Increased sedentary behaviours, e.g. increases in the average time spent watching television,

have been suggested as a factor influencing the increase in obesity . 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 physical activity levels; equally, computer use could have replaced time spent in other sedentary activities, such as listening to the radio or music, watching television and reading, and not impacted on overall activity levels.

148.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 . 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 .

149.Several prospective studies since the meta-analysis have also observed positive associations between television viewing in children and subsequent weight gain . 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 .

150.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 .

151.A prospective study of adolescents in the US, observed that changes in television viewing were not associated with changes in leisure-time moderate/vigorous physical activity . It was suggested that that television viewing and leisure-time physical activity were separate constructs that could each affect weight gain, but were not functional opposites. Television viewing may be related to overweight through television's effects on dietary intake; television advertising may promote consumption of unhealthy foods and people tend to snack while watching television .

Physical activity and body fatness

152.The energy expended during physical activity would appear to be an important factor in maintaining a neutral energy balance. PAEE is the most variable component of TEE and is amenable to modification. The rising prevalence of obesity has been attributed in part to population-level changes in physical activity .

153.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 . 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 .

154.The focus of this section is on the role of physical activity in the primary prevention of weight gain and obesity.

Prospective studies of self-reported physical activity and weight gain

155.The prospective studies relating physical activity and weight change were systematically reviewed in 2000 both in adults and children ; this was subsequently updated by a systematic review of prospective cohort studies and trials published since 2000, which examined the association between physical activity and weight change in adults and children .

Adults

156.The Fogelholm and Kukkonen-Harjula (2000) systematic review included 16 prospective studies investigating the relationship between self-reported physical activity and weight change. The mean duration of the follow-up was approximately 7 years, with a range from 2 to 21 years. The range of participants was 210 to 79,236. All studies used a retrospective questionnaire to assess the habitual level of physical activity. The studies differed in the physical activity data that was collected, i.e. whether baseline, follow-up or change in physical activity compared to weight change.

157.Overall, two studies reported a consistent inverse association between baseline physical activity and subsequent weight gain ; whereas, three did not and one found a positive association . One study observed a positive association for men and a negative association for women between baseline physical activity and subsequent weight gain . Jogging or running, but not other less strenuous activities, were inversely associated with weight gain in one study .

158.There were seven studies that found an increase in physical activity between baseline and follow-up was associated with less weight gain and two did not . Four studies found that a large volume of physical activity at follow-up was associated with less weight gain , while one did not . In one study the cumulative duration of increased physical activity was observed not to be associated with weight change .

159.It was concluded that there was inconsistent evidence of a predictive effect 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 .

160.A follow-up systematic review included twelve prospective studies investigating the relationship between self-reported physical activity and weight change. Most studies included a large range of subjects (539 to 50,277) and a range of follow-up from 3 to 11 years. Nine studies reported a negative association between baseline physical activity and subsequent weight gain and two found no association . One study reported an inverse association suggesting higher baseline levels of BMI predicted physical inactivity . 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 had no studies with less than 500 participants, whereas the studies included in the previous review included five studies with less than 500 participants and, therefore, less power to

detect small differences. Improvements in study design could also be factor, as could publication bias in determining why the follow-up systematic review produced more consistent results.

161. It was 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 because of issues of confounding, measurement error and reverse causality, i.e. obesity may lead to physical inactivity .
162. A study subsequent to the systematic review (n=11,115; followed-up for 12 years) observed leisure-time physical activity to be inversely associated, and physical activity at work positively associated, with BMI, especially in women .

Children and adolescents

163. The Molnar and Livingstone systematic review identified two prospective studies that investigated the influence of self-reported physical activity on the change in relative body mass index. In a cohort of 146 preschool children, followed-up over a 3-year period, increases in children's leisure activity at follow-up were associated with decreases in subsequent weight gain . In a cohort of 112 prepubertal children, followed up for 4 years, the amount of physical activity at baseline was not associated with BMI variance at follow-up.
164. The Wareham *et al* (2005) systematic review identified a further 11 studies with a follow-up ranging from 1 to 23 years; five of the studies had a follow-up of 2 years or less. The studies included between 59 and 11,887 children, with four including more than 1000 children. Most studies included children less than 10 years old; the range was 3 to 14 years. All studies, except one , 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 . The other six studies found an inverse association between higher levels of physical activity and weight gain or a positive association with sedentary activities .
165. Overall, the results were mixed and it was concluded that, as in the adult studies, the measures of association tended to be small . Another review of prospective studies also concluded that the results were mixed and that the associations identified were generally of small magnitude .
166. The National Heart Lung and Blood Institute's Growth and Health Study of 1,152 black and 1,135 white girls followed up from ages 9 or 10 to 18 or 19 years reported physical activity levels of girls during adolescence significantly affected changes in BMI and adiposity . It was concluded preventing the decline in activity observed during adolescence might be an important method to reduce obesity.
167. In a study of 208 children followed-up for a median of five years, physical activity was inversely related to fat mass development in boys and but not in girls . A prospective comparison (5 year follow-up) of children (n=2,727) from different schools in the UK, observed that boys in schools providing 3 weekly physical education sessions had smaller increases in fat mass than boys in schools providing one or two sessions. Differences in girls were in the same direction, but not significant; There were no differences in BMI changes or the percentage of students classified as obese between schools of higher and lower frequency

of physical education .

The tracking of physical activity from youth to adulthood and risk of weight gain

168. Most obese children remain obese as adults a progression that is referred to as ‘tracking’ of overweight. The prevention of obesity has focused on childhood as a target period. Several studies have examined whether adolescent physical activity affects subsequent weight gain through to adulthood.
169. Some prospective studies do provide evidence that a decline in physical activity between adolescence and adulthood may increase risk of weight gain and obesity, but overall these associations were generally weak and inconsistent

Summary

170. 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 were mixed and the associations that were identified were generally of a small magnitude.
171. It is likely that imprecise measurement of activity exposures weakens the observed relationships . 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 of limited value in determining fat and lean tissue mass across the normal range in adults and children .

Prospective studies of objectively measured physical activity and weight gain

172. Children are less likely than adults to make an accurate self-reported physical activity assessment and in children of younger age groups it is virtually impossible to obtain valid self-reported physical activity data .
173. Physical activity is not easy to assess because it is a complex multi-dimensional behaviour and takes place in a variety of different domains, i.e. in transportation, domestic life, occupation and recreation . In addition, physical activity assessment needs to consider intensity, frequency, duration and the type of activity undertaken .
174. 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 total energy expenditure . Although subjective measures of physical activity have proved sufficient to demonstrate inverse associations with many health outcome, they have infrequently separated physical activity into its different dimensions and they have not allowed the estimation of dose-response effects . The use of objective measures of physical activity is required to elucidate the dose-response relationship between physical activity and health, as well as, for determining current levels of physical activity, monitoring compliance with physical activity guidelines and determining the effectiveness of intervention programmes designed to improve physical activity.

Objective measures of physical activity

175. As noted above cardiorespiratory fitness had been used as a surrogate for physical activity, but this does not define the amounts and types of physical activity or the amount of energy expenditure. In children, furthermore, there is only a weak association between cardiorespiratory fitness and the level of physical activity .
176. The DLW method measures TEE over several days and in conjunction with measures, or estimates, of BMR/RMR can be used to measure PAEE indirectly. The DLW method, however, does not give day-to-day information nor does it give information on the forms, frequency and intensity of physical activity undertaken .
177. 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 the 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. The HRM method is limited in its ability to differentiate between modest increases in 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 . PAEE can be estimated in groups using HRM and accelerometry, but the DLW method provides a more accurate assessment.

Adults

178. No relevant studies were identified in the Fogelholm and Kukkonen-Harjula (2000) systematic review, but the Wareham *et al* (2005) systematic review identified two studies. In Pima Indians (n=92) PAEE, as determined by DLW technique, was not associated with change in body weight during follow-up after 4 years . In a population cohort of UK adults (n=739), the association between PAEE, as assessed using HRM, and change in body composition (FM and FFM) over a 4 year follow-up was investigated . PAEE predicted change in FM in younger adults, but not older adults, although the effect size was small. Younger adults on average gained weight during the study. In older adults, who were on average weight stable, PAEE predicted gain in body weight (an increase in FM and FFM).
179. A later study of obese (n=13) and normal weight (n=15) young adults followed-up for 4 years assessed RMR, substrate oxidation, movement economy (ml O₂ kg⁻¹ min⁻¹), aerobic fitness, TEE and PAEE by DLW, and physical activity by accelerometry in relation to change in body weight and FM. Energy expenditure estimates and subcomponents of physical activity (i.e. time spent at different intensity levels) were not significantly associated with change in body weight or FM, but change in overall physical activity (accelerometry) was inversely associated with change in body weight and FM. *Post hoc* analyses suggested that this association was explained by changes in the normal weight group only suggesting that the observed association may differ depending on obesity status.

Children and adolescents

180. The Molnar and Livingstone systematic review identified five prospective studies that investigated the influence of objectively measured physical activity on the change in relative body mass index.

181. A study of 32 active and 32 non-active boys followed longitudinally from 13 to 18 years of age used a physical fitness test battery to assess physical activity found no association between physical activity and growth in somatic dimensions, including adiposity (skinfolds). The Framingham Children's Study (106 children aged 3-5 years followed-up till first grade) found children with low levels of physical activity (assessed using electronic motion sensor) gained substantially more subcutaneous fat than did more active children .
182. Three studies had used DLW techniques. A study of infants of 6 lean and 12 overweight mothers observed reduced TEE, and particularly PAEE, at 3 months was associated with weight gain during the first year of life in infants born to overweight mothers . A subsequent study in infants (n=33) born to non-obese mothers, found no association between TEE at 3 months with indices of body fatness at 9 and 24 months of age .
183. A study of 75 children aged 3.5–7.0 years and followed-up over 4 years found no association between TEE or PAEE and change in percentage FM, adjusted for fat-free mass . It has been suggested that measurement error and adjustment of data could have obscured any differences that may have existed in this study .
184. The Wareham *et al* (2005) systematic review identified five subsequent studies. The children included in these studies were mostly younger than 10 years and the duration of follow-up ranged from 2 to 8 years.
185. One study in 103 children assessed physical activity using accelerometry and observed children in the highest tertile of average daily activity from ages 4 to 11 years had consistently smaller gains in BMI, triceps, and sum of five skinfolds throughout childhood .
186. The other studies used DLW techniques. A study in 47 girls observed an inverse association between PAEE adjusted for FFM and percentage FM at a follow-up of 1.6 years, but not at 2.7 years . One study in 88 girls observed a small positive relationship between TEE, but none for PAEE, adjusted for baseline weight and percentage FM at a follow-up for 2 years . Another study in 95 children followed-up for 3-5 years observed no association between RMR, TEE or PAEE, adjusting for initial body composition, and change in adiposity, but aerobic fitness was inversely associated with adiposity . A study in 26 infants (9-12 months) followed up at 2 years of age observed that TEE, adjusted for FFM, was not related to change in FM . It was concluded that overall the results from these studies was mixed.
187. Studies in overweight Pima Indian children and adults were not included in the systematic review. These studies measured TEE and PAEE using DLW techniques and observed no relationship with changes in body weight.
188. Several studies have been published since the review. A study in 196 pre-menarcheal girls followed up over 4 years found no association of PAEE (or other EE variables determined by DLW techniques) with relative weight and percentage FM during adolescence .
189. A study in a triethnic cohort of 3-4 year old children followed for 3 years, observed physical activity (as assessed by heart rate monitoring) to be positively associated with BMI in year 1, and negatively associated in years 2 and 3, with a stronger negative relationship in year 3 than 2 .

190. A study in 879 children and adolescents (aged 5-18) followed-up after 1 year, assessed physical activity using accelerometry . The percentage of time in sedentary activity was positively associated with weight gain and the percentage of time in light activity was negatively associated. The percentage of time in moderate and vigorous activity was not associated with weight gain.

Summary

191. Overall, the results from prospective studies using objective measures of physical activity in children, adolescences and adults were mixed and the associations identified were generally of small magnitude .

192. 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 . 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 underestimated, resulting in associations being biased towards null . The use of DLW measures of PAEE to identify how much physical activity 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 heart rate monitoring and accelerometry is also required .

193. The potential impact of exercise intensity on change in BW and FM remains unclear 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 to prevent weight gain

194. Interventions aimed at weight reduction or at preventing weight regain are not included. A systematic review was published in 2000 that identified nine interventions (eleven publications) of the primary prevention of weight gain . Interventions lasted from 6 weeks to 36 months. Four interventions took place in the community and five were school based .

195. 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.

196. This systematic review was subsequently updated with a further seventeen trials in 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, but weight stability in the intervention group, one found a weight reduction in the intervention group and the other, decreases in both groups. Two trials observed no effect on weight gain.

197. A total of eleven trials were identified in children aimed at preventing unhealthy weight gain by increasing physical activity or reducing sedentary behaviour. 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, with two of them reporting effects in boys only. The other eight trials reported no significant effects on body weight or composition at follow-up.

198. It was concluded that there were still relatively few trials aimed at the primary prevention of weight gain and that there was still insufficient evidence on which to base conclusions about which of the approaches were effective.

199. A cluster randomised controlled single blinded trial over 12 months in 545 preschool children (average age 4.2 years) using nursery and home-based intervention was found not to reduce body mass index at follow-up.

Summary

200. There were relatively few trials aimed at the primary prevention of weight gain and, overall, findings were inconsistent.

Conclusions

201. Survey data suggest that only about a third of the adult UK population meet the general physical activity recommendation to have at least 30 minutes a day of moderate intensity physical activity, which, results from surveys in children using objectively measured physical activity suggest, is likely to be an overestimation.

202. 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 and children. Error is introduced on both sides of the relationship reducing the ability to detect any change. The application of more precise methods for the measurement of physical activity and body fatness – e.g. accelerometers and DXA – is required to define their interrelationship.

203. 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 were mixed and the associations that were identified were generally of a small magnitude. Prospective studies also suggest television watching may be a predictor for weight gain, but again the associations were weak. Evidence from studies employing objective measures of physical activity, especially DLW-derived measures of PAEE, and trials of the primary prevention of weight gain was

inconsistent.

204. The issue of how much physical activity is required to prevent unhealthy weight gain is very complicated, and available data is insufficient for a definitive conclusion. As weight gain is dependent on energy intake, consideration of energy balance and flux is required, rather than energy intake and expenditure in isolation. This is the topic of Chapter 9.

References

Dietary determinants of weight gain

Background

205. Body weight is only gained when energy intake exceeds energy expenditure for a prolonged period. Studies of basic physiology under standardized conditions, and controlled intervention studies involving manipulations of the components of energy balance, clearly demonstrate weight loss with underfeeding and weight gain with overfeeding. It is important to consider energy intake in the context of an individual's energy expenditure. Weight gain does not necessarily result from a high absolute energy intake, but from an intake which exceeds energy needs, even as a small fraction of energy flux. The coupling between energy intake and expenditure is, therefore, central to energy balance. Energy flux, the absolute level of energy intake and expenditure under conditions of energy balance, will be considered further in Chapter 9.

Appetite control and energy balance

206. In order to maintain a stable body weight over a long period of time, energy intake must be continually balanced with energy expenditure. In most adults, adiposity and body weight are remarkably constant despite huge variations in daily food intake and energy expended. A powerful and complex physiological system exists to balance energy intake and expenditure, composed of both afferent signals and efferent effectors. This system consists of multiple pathways which incorporate significant redundancy in order to maintain the drive to eat. In the circulation, there are both hormones which act acutely to initiate or terminate a meal, e.g. the gut peptides cholecystokinin and ghrelin, and hormones that reflect body adiposity and energy balance, e.g. insulin and the adipose tissue hormones leptin and adiponectin. These signals are integrated by peripheral nerves and brain centres, such as the hypothalamus and brain stem. The integrated signals regulate central neuropeptides, which modulate feeding and energy expenditure. This energy homeostasis, in most cases, regulates body weight tightly.

207. The manipulation of energy intake has been shown to result in compensatory changes in energy expenditure. In response to a negative energy balance an adaptive suppression of thermogenesis occurs in the resting compartment of energy expenditure (i.e. in BMR, thermic response to food or in sleeping metabolic rate) and/or in the non-resting components of energy expenditure (walking, bicycle exercise and spontaneous physical activity), which has also been observed in the long-term maintenance of a lower body weight, and after weight recovery. There is considerable inter-individual variation in this response and, in most studies, this corresponds to mean values of 5–15% of energy expenditure in either the resting or non-resting compartments.

208. A state of negative energy balance appears to be defended more strongly than one of positive energy balance. Overfeeding produces much less compensatory change in energy expenditure than food restriction. Overall, adaptive changes in energy expenditure are small and changes in body weight and composition are the primary mechanisms by which the body accommodates energy excess or inadequacy. Alterations in energy balance must be accommodated by adjustments in the net storage of the major energy-yielding macronutrients: carbohydrate, protein, and fat.

209. The body's regulatory systems, therefore, are developed to avoid depletion of body energy stores, whereas the systems that prevent excessive accumulation of fat in response to excess energy intake are more easily suppressed. Psychological, social, and cultural factors are important underlying influences on dietary patterns and physical activity energy expenditure.
210. While brain mechanisms control appetite, a number of sensory and environmental factors contribute to overstimulation of the sensory systems, producing sensory reward signals that are stronger than can always be controlled easily by satiety signals. Factors that contribute to over stimulating the brain's food reward systems relative to satiety signals include food palatability and appearance, sensory-specific satiety, food variety, food availability and the effects of visual stimulation and advertising.

Substrate utilization and energy balance

211. The fuel selection in tissues is regulated by endocrine and other sensor cells that monitor circulating concentrations of fuels and adjust supply and demand accordingly. The principal fuels in man are glucose (also glycogen, lactate, pyruvate and alanine) and fatty acids (also adipocyte, blood plasma and muscle triacylglycerol, and ketone bodies), although amino acids also make a significant contribution. Glucose is essential for cells that are obligatory anaerobes and for some brain cells. The metabolic relationship between plasma glucose and plasma fatty acid concentrations is reciprocal and not dependent. An increase in plasma glucose concentration (e.g. after carbohydrate ingestion) promotes glucose oxidation and glucose and lipid storage, and inhibits fatty acid oxidation. An increase in plasma non-esterified fatty acid concentration (e.g. from increased adipose tissue lipolysis) promotes fatty acid oxidation and storage, inhibits glucose oxidation and may promote glucose storage if glycogen stores are incomplete.
212. The fuel mix on an average Western diet is approximately 50% carbohydrate (half used in brain), 33% fat and 17% protein in the fed state, changing to 12, 70 and 18% respectively after overnight fast (80% use of protein and carbohydrate by brain) and to 0, 95 and 5% (all brain) respectively after 40 days of starvation. In prolonged starvation glucose oxidation is essentially replaced completely by oxidation of lipid fuels in non-brain tissues; in the brain glucose oxidation is replaced by ketone body oxidation to approximately 90%. Glucose oxidation is also decreased by a low-carbohydrate high-fat diet, while exercise increases oxygen consumption and glucose oxidation; the contribution of glucose decreases with duration of exercise (>1 hour). These changes are effected without a fall in plasma glucose below concentrations critical for consciousness. The major hormones known to be involved in coordination are insulin, glucagon, catecholamines, growth hormone and corticosteroids. Insulin activity is an important determinant of substrate partitioning, that is, by stimulating dietary fatty acid uptake in adipose tissue, inhibiting endogenous adipose tissue lipolysis, suppressing skeletal muscle fat oxidation, stimulating glucose uptake, oxidation and storage.
213. There is a large variation in the amplitude of the circulating concentrations for different fuels with dietary variation (e.g. high-carbohydrate diet, high-fat diet, starvation) or exercise. Glucose shows an amplitude of only about 3-fold; lactate, pyruvate, alanine, triacylglycerol about 10-fold; non-esterified fatty acids about 15-fold; and ketone bodies about 100-fold.

214. The energy balance equation can be reformulated in terms of separate balance equations for the individual energy-yielding macronutrients (see Figure 1.)

Figure 1. The oxidative hierarchy for the major energy supplying macronutrients. Protein and carbohydrate have a similar position in the hierarchy .

Nutrient stores	=	Energy Intake	–	Energy expenditure	Oxidative autoregulation
None	equals	Alcohol	minus	Alcohol	‘Perfect’
Glycogen		Carbohydrate		Carbohydrate	Excellent
Body protein		Protein		Protein	Good
Adipose tissue		Fat		Fat	Poor

215. The observations on the oxidative hierarchy of macronutrient regulation were made under a variety of conditions, including subjects in positive, negative or neutral energy balance or fed *ad libitum* . In the oxidative hierarchy, the macronutrients that are least easily stored assume priority in the metabolic fuel mixture being combusted . Alcohol in the plasma dominates oxidative pathways because it cannot be stored and must be detoxified . Ingestion of alcohol elicits a rapid rise in alcohol combustion that is maintained until all alcohol is cleared. Carbohydrate oxidation also shows tight autoregulatory linkage with carbohydrate intake because the capacity for glycogen storage is limited and *de novo* lipogenesis from carbohydrate, although influenced by the macronutrient composition of the diet, is likely to be quantitatively unimportant in typical diets with regard to the total fat balance .

216. Protein oxidation, and hence nitrogen excretion, is likewise linked to protein intake . The relative positions of protein and carbohydrate in this hierarchy remain a matter of debate . In contrast with the other macronutrients, there appears to be virtually no autoregulatory linkage between fat intake and fat oxidation . Fat oxidation is suppressed by high intakes of the other macronutrients, even in the presence of large quantities of dietary fat, and tends to decline under conditions of energy excess . Body fat can accumulate indirectly in response to overfeeding, even with high-carbohydrate diets, due to declining fat oxidation rather than to *de novo* lipogenesis .

217. During underfeeding fat is the major oxidative fuel. There is no evidence of fat-driven autoregulation, rather, fat oxidation reflects the difference between the rates of carbohydrate and protein oxidation and TEE. Replacement of carbohydrate with protein during energy restriction have been observed to increase fat oxidation and decrease carbohydrate oxidation postprandially and, in the longer term, improve FFM retention during weight loss .

218. The need to maintain carbohydrate balance has been suggested as contributing to the regulation of food intake and thereby to the control of body weight . Studies investigating the effect of extreme manipulations of carbohydrate intake, and hence glycogen stores, on modulating *ad libitum* food intake have, however, failed to show any significant influence , indicating that imbalances in glycogen stores are re-established not by adjustments of energy intake, but by adjustments of macronutrient oxidation rates .

219. The distribution of food energy between the macronutrients differs between cultures and countries, reflecting a wide range of carbohydrate-protein-fat ratios at which a neutral energy balance is maintained. The substrate oxidation pattern can shift to closely reflect the diet macronutrient composition. In healthy populations, subjects have been shown to switch their fuel use based upon substrate availability, e.g. increase fat oxidation in response to a high fat diet. It takes several days for substrate utilization to adapt to a change in diet macronutrient composition and modification of glycogen stores has been implicated in this adaptation.
220. In short-term studies, a hierarchy has been observed in the satiating efficacies of the macronutrients protein, carbohydrate and fat, with protein as the most satiating and fat the least. It has been proposed that the satiating efficiency of a macronutrient may be related to its position in the oxidative hierarchy during the postprandial state – with the position of alcohol still being unclear.
221. Studies manipulating the macronutrient composition of test meals to determine the relation between the macronutrient oxidation hierarchy and subsequent *ad libitum* energy intake have produced mixed results. Several have observed effects on satiety measures, but no significant effect on *ad libitum* energy intake, despite measurable differences in postprandial substrate oxidation; whereas, others have observed that increasing the protein content of the test meal reduced subsequent *ad libitum* energy intake relative to manipulations with the other macronutrients.
222. Two longer term studies have investigated the effect of increasing the diet protein content on energy intake. One study observed that an increase in dietary protein intake from 15% to 30% of energy at a constant carbohydrate intake produced a decrease in *ad libitum* energy intake. Another study observed that an increase in dietary protein intake from 12% to 25% of energy at a low-fat intake produced both a decrease in *ad libitum* energy intake and a greater magnitude of spontaneous weight loss than a low-fat diet alone after 6 months. These differences persisted, but were attenuated after consumption of the study diets for 12 months.
223. Because the short term regulation of fat balance has a lower priority than that of carbohydrates, protein and alcohol it has been suggested that fat intake could increase the risk of excess energy intake and the promotion of fat storage. Longer term studies investigating an effect of dietary carbohydrate and fat content on energy intake have shown that a low-fat, high-carbohydrate diet causes a decrease in *ad libitum* energy intake accompanied by modest spontaneous weight loss.
224. Short term and longer term studies investigating the effects of meals with contrasting glycaemic carbohydrate content have failed to demonstrate any differential effect on fuel partitioning when either substrate oxidation or body composition measurements were performed. Although single meal studies suggest glycaemic index may affect later *ad libitum* energy intake, longer term studies show no effect on *ad libitum* energy intake. The postprandial insulin response may be an important satiety signal, but glycaemic index-induced serum insulin differences appear insufficient to modify fuel oxidation and energy intake.
225. Overall, therefore, it is unclear whether substrate oxidation predicts satiety in subjects who

have adapted to a change in diet macronutrient composition, rather than in an acute situation .

226.Spontaneous long term weight changes have been observed to be accompanied by small metabolic adaptations in both energy expenditure and fat oxidation, e.g. an increase in energy expenditure and fat oxidation in response to weight gain, although this response was highly variable among individuals . A reduced ability to adapt to a greater fat oxidation rate in response to a high fat diet has been observed in formerly obese individuals , leading to the suggestion that inter-individual differences in the capacity to adapt fat oxidation to fat intake may translate into differences in weight gain over time, when subjects are exposed to a high-fat diet .

227.Results from prospective studies investigating whether differences in fuel oxidation predispose to weight gain, however, are inconsistent. Some studies have observed no relation , while others implicate differences in fat oxidation and fat balance in weight gain .

Methodological constraints

228.The evidence from prospective studies determining dietary intake and weight or of body mass index (BMI) has largely yielded weak and inconsistent results. In part, this may be a true reflection of the multi-factorial nature of weight gain and obesity, but it also relates to methodological difficulties inherent in these studies.

229.Body weight and composition is the integrated product of previous dietary and physical activity habits, and so nutrients, foods or broader dietary habits measured on a small number of occasions may not be related to the longer-term development of weight change. Many dietary factors are highly correlated and physical activity or other lifestyle factors are also important covariates .

230.A major limitation in most studies is the reliability of the data on dietary exposures. For example, the degree of error associated with the food frequency questionnaires is considerably larger than that associated with quantitative 7 day diaries of all food and drink consumed . An association between dietary saturated fatty acid intake and disease outcome observed using quantitative 7 day diaries was shown to be obscured when food frequency questionnaires were used .

231.In most dietary surveys, energy intake is under-reported, relative to estimated energy needs, by an average of 20-25% . The under-reporting of energy intake is more pronounced among overweight and obese, than among lean, adults and children . The nutritional composition of the 'energy gap' is unclear and statistical adjustments tend to be made based on energy alone. In one study, measurement of biomarkers of sugar and vitamin C consumption in obese and lean individuals suggested that those who were obese consumed more sugar and less vitamin C than their thinner counterparts, but this was not apparent from food frequency questionnaire data .

232.Despite the limitations of observational studies, there is a paucity of controlled dietary intervention studies to test aetiological hypotheses. Because of the nature of such interventions it is rarely possible to design a double-blind trial and the process of informed consent and baseline screening may act as an intervention in itself by raising awareness of the diet or health issue and prompting behaviour change. Dietary change is difficult to

sustain as traditional dietary practices are well established and strongly habituated. Numbers of dropouts from trials are frequently high and the extent of compliance is often difficult to measure. In terms of prevention, the mean weight change is small (especially superimposed on the background population weight increase) and there is large inter-individual variability which has implications for sample size and power. Accordingly, there are few large-scale, long-term, well-controlled dietary intervention studies .

233.Overall, these methodological constraints are likely to predispose studies towards negative findings and attenuate any observed associations. This is reflected in the relatively few observational studies that have demonstrated the basic physiological principles dictating habitual energy intake must exceed expenditure for weight gain to occur .

The National Diet and Nutrition Survey reported energy intakes

234.Surveys in the UK, both of dietary and household purchase data, indicate energy intakes by the population below the prevailing EAR. The National Diet and Nutrition Survey (NDNS) series consistently show average energy intakes below the EAR in all population groups (see Table 1). The NDNS and other surveys, however, also show that the number of people classified as overweight or obese is increasing.

Table 1. EARs used in NDNS analyses for 4-18 years, 19-64 years and 65 years and over.

Age group	Men (MJ/day)	Women: (MJ/day)
4 – 6 years:	7.16	6.46
7 – 10 years:	8.24	7.28
11 – 14 years:	9.27	7.72
15 – 18 years:	11.51	8.83
19 to 50 years:	10.60	8.10
51 to 59 years:	10.60	8.00
60 to 64 years:	9.93	7.99
65-74 years:	9.71	7.96
75+ years	8.77	7.61

Energy intake as a percentage of EAR was calculated for each respondent using the EAR appropriate for sex and age.

***Energy intakes per kilogram body weight was used to calculate Intake as % of EAR on a group basis for 1½-4½ year age group

Children & adolescents aged 4-18 years

235.Mean energy intakes were below the EARs at 80-90% of the EAR. In girls aged 15-18 years mean energy intake was 77% of the EAR.

Adults aged 19-64 years

236.Mean energy intakes for adults in 2000/01 fell below Estimated Average Requirements (EARs) for men and women in all age groups, and were 82-94% of EAR. However, the increasing prevalence of obesity suggests that energy intakes are generally in excess of

requirements rather than inadequate.

237.Data from the two surveys of adults in 1986/87 and 2000/01 showed that the prevalence of obesity or overweight had increased from 45% of men and 36% of women who were obese or overweight in 1986/87 to 66% of men and 53% of women in 2000/01. This trend is confirmed by data from the Health Survey for England. Mean energy intakes in the 1986/87 survey were also below EARs. Comparing the 1986/87 and 2000/01 surveys, mean energy intake had fallen slightly in men but was unchanged in women.

Adults aged 65 years and over

238.Mean energy intakes were below EARs. In the free-living group mean energy intake was 85% of EAR in men and 76% in women and for men was lowest in the 85+ age group. In the institution group mean intake was over 90% of the EAR.

239.In Table 2 energy intake (EI) is presented as a percentage of the EAR for all NDNS age/sex groups. Standard EAR values were used for each age/sex group as published in the UK Dietary Reference Values . EAR values for each age/sex group were derived from BMR calculated from the modified Schofield equations using mean body weight values for each age/sex group. PAL values for adults were taken as 1.4.

Table 2. Mean daily reported energy intake (MJ) as a percentage of EAR using standard EAR values

Gender and age of respondent	Mean energy intake (MJ)	Mean intake as % of EAR**	Number of subjects
Males and females 1.5-2.5 years***	4.39	90%	576
Males and females 2.5-3.5 years***	4.88	84%	606
Male aged (years)			
3.5-4.5***	5.36	82%	250
4 – 6	6.39	89%	184
7 – 10	7.47	91%	256
11 – 14	8.28	89%	237
15 –18	9.60	83%	179
19-24	9.44	89%	108
25-34	9.82	93%	219
35-49	9.93	94%	253
50-64	9.55	92%	253
65+ Free-living	8.02	85%	540
65+ Living in an institution	8.14	91%	93
Female aged (years)			
3.5-4.5***	4.98	82%	243
4 – 6	5.87	91%	171
7 – 10	6.72	92%	226
11 – 14	7.03	89%	238
15 –18	6.82	77%	210

19-24	7.00	86%	104
25-34	6.61	82%	210
35-49	6.96	86%	318
50-64	6.91	87%	259
65+ Free-living	5.98	76%	735
65+ Living in an institution	6.94	90%	319

Energy intake as a percentage of EAR was calculated for each respondent using the EAR appropriate for sex and age.

*** Energy intakes per kilogram body weight was used to calculate Intake as % of EAR on a group basis for 1½-4½ year age group

240. Table 3 presents energy intake as a percentage of EAR for the 19-64 age group only (NDNS adults 2000/01). The EAR was calculated for each subject using individual body weight data to estimate BMR and PAL values derived from the seven-day physical activity records.

Table 3. Average daily reported energy intake (MJ) as a percentage of the EAR using EAR values based on individual BMR and PAL

Gender and age of respondent	Mean energy intake (MJ)	Intake as % EAR**	Number of subjects
Male aged (years)			
19-24	9.60	66%	54
25-34	9.92	68%	144
35-49	9.99	72%	283
50-64	9.57	73%	216
All 19-64	9.82	71%	697
Female aged (years)			
19-24	7.13	69%	66
25-34	6.61	67%	187
35-49	7.05	71%	341
50-64	7.07	73%	260
All 19-64	6.97	71%	854

** EAR derived for each individual using BMR – estimated using modified Schofield equations and actual body weight measured during the survey period. PAL calculated for each individual using physical activity records.

Comparison of average daily energy and macronutrient intakes between the NDNS for adults 1986/7 and 2000/1

241. Comparisons between the 1986/7 and 2000/1 NDNS for adults data show that, overall, men and women in the 2000/1 survey reported a significantly higher proportion of their energy intake from protein and carbohydrate and a significantly lower proportion from fat than in 1986/7; men, but not women, also had significantly lower energy intakes in 2000/1 than 1986/7. There was no significant difference between the proportion of energy intake from alcohol between the two surveys (see Table 4).

Table 4. Comparison of average daily energy and macronutrient intakes between the NDNS for adults 1986/7 and 2000/1

	1986/7	2000/1
Men		
Total energy intake (MJ)	10.30	9.72
% energy from carbohydrate	41.6	44.7
% energy from protein	14.1	15.4
% energy from fat	37.6	33.5
% energy from alcohol	6.9	6.5
Women		
Total energy intake (MJ)	7.05	6.87
% energy from carbohydrate	43.0	46.7
% energy from protein	15.2	15.9
% energy from fat	39.2	33.5
% energy from alcohol	2.8	3.9

Diet composition and weight gain

242. The role of diet composition in the development of weight gain and obesity is controversial. As discussed above, macronutrient balance has been implicated in excess energy intake through effects on fuel metabolism and satiety responses, but any long term effects on weight gain need to be established.

Macronutrient composition

243. The relative impact of the different macronutrients on energy balance and risk of weight gain has been the focus of much research, with particular emphasis on the proportion of energy from fat in promoting weight gain. In the UK, however, survey data suggests there has been a decline in the percentage of energy intake from fat during the last 2 decades, which has corresponded with an increase in the prevalence of overweight and obesity. Evidence from observational studies of a specific role for the proportion of energy from dietary fat are inconsistent. A meta-analysis of *ad libitum* low-fat high-carbohydrate dietary intervention studies (16 trials; 2-12 months duration; mean fat reduction of 10.2% total energy) observed that a reduction in dietary fat caused spontaneous weight loss (mean weight loss of 3.2 kg as compared to control groups).

244. In a subsequent review, however, it was noted that in randomized trials lasting for a year or more, fat consumption within the range of 18% to 40% of energy appeared to have little effect on body fatness. The weighted mean difference was a weight reduction of 0.25 kg overall and a weight increase of 1.8 kg (i.e., less weight loss on the low-fat diets) for trials with a control group that was regarded of a comparable intensity intervention.

245. The Women's Health Initiative randomized intervention trial of 48,835 postmenopausal women in the United States, found a reduction in energy from fat (mean fat reduction of 9% total energy) and an increase in energy from carbohydrate resulted in no weight gain over 7.5

years in the intervention group, relative to the control group (weight difference of -1.9 kg, after 1 year and -0.4 kg after 7.5 years relative to control group). Intake of fibre, fruits and vegetables, total grains, and whole grains were also increased from baseline in the intervention group while remaining stable in the control group.

246. Lack of compliance in these studies makes it difficult to assess the magnitude of effect of any intervention, but it has been argued that any long-term effects on body weight are likely to be small and that there is no strong evidence that lowering the proportion of energy as dietary fat would prevent weight gain.
247. The proportion of carbohydrate in the diet tends to vary reciprocally with fat and it is difficult to segregate the impact of the total amount of carbohydrate in the diet from the total amount of fat. While some studies show an inverse association with weight gain, many others show no association, particularly in children.
248. Short-term intervention studies have shown that the energy density (kJ/g) of foods significantly affects energy intake in both men and women. These studies suggest that high energy-dense diets can undermine normal appetite regulation, a process that has been termed 'passive overconsumption'. This inability to recognize the energy density of food and compensate appropriately to maintain energy balance could lead to unhealthy weight gain, and overweight and obesity. Dietary fat is the main determinant of the energy density of foods, but some reduced-fat, and, therefore, higher carbohydrate, foods are highly energy dense. High carbohydrate foods do not necessarily have a low energy density and although it is easier to overeat on high fat than low fat foods, it may not be a case of simply replacing fat with carbohydrate in the diet as a means to protect against overconsumption.
249. Two prospective studies, in children and adolescents, have assessed the energy density of the diet in relation to weight gain, but their results have been inconsistent. So-called 'fast foods' typically have an energy density very much greater than 'household' food and 'fast food' consumption has been associated with weight gain in prospective studies.
250. As well as the total amount of fat or carbohydrate in the diet, studies have examined the role of fat and carbohydrate composition on weight gain. The evidence relating sugar intake *per se* to weight change is inconsistent. Two 6 month trials of low-fat high-carbohydrate *ad libitum* diets, which compared complex with simple carbohydrates on weight gain, have observed no significant effect of carbohydrate composition. By contrast, there is accumulating evidence to suggest that simple sugars in drinks do not possess the same satiating powers as sugars in solid food (the fluid medium rather than energy form or nutrient composition is thought responsible) and that a high consumption of sugar-sweetened beverages may be a risk factor for weight gain and obesity. Surveillance studies show a temporal association between *per capita* intake of sugar-sweetened beverages and increasing obesity rates. A meta-analysis of studies (10 prospective; 5 experimental) investigating the role of sugar-sweetened beverage consumption in weight gain concluded that in well-powered prospective cohort studies with long periods of follow-up (6 studies), a positive association between greater intakes of sugar-sweetened beverages and weight gain and obesity in both children and adults was observed; the other cohort studies (4 studies) observed no significant association. One subsequent study in children also observed no association, while another study in adults observed increased soft drink consumption to be associated with weight gain, obesity and developing metabolic syndrome. The meta-

analysis concluded that findings from short-term feeding trials in adults lend support to an induction of positive energy balance and weight gain by intake of sugar-sweetened beverages, but there were only a few studies and longer term trials were needed .

251. Several prospective studies have observed an inverse association between wholegrain and fibre/non-starch polysaccharide (NSP) intake and weight gain ; although the definitions used for dietary fibre/NSP and wholegrain vary between studies. Conversely, intake of refined-grain foods was positively related to weight gain in one study , but not another .

252. A diet rich in wholegrain and NSP may have beneficial effects on weight control through promoting satiety and decreasing the digestibility of the energy-containing nutrients . A review of 11 intervention studies (duration more than 4 weeks) of ad libitum NSP intake and weight change found that consumption of an additional 14 g/day NSP for >2 days was associated with a 10% decrease in energy intake and body weight loss of 1.9 kg over 3.8 months .

253. Results from prospective studies that have investigated whether a diet with a high glycemic index or glycemic load contributes to the risk of weight gain have been inconsistent . Intervention studies investigating this have also produced inconsistent results .

254. Different types of fatty acids have different metabolic effects and it is possible that this extends to differences in risk of weight gain , e.g. animal work suggests that saturated fatty acids may be preferentially stored and high *trans* fatty acid intake may enhance the intra-abdominal deposition of fat. Several prospective studies have observed *trans* fatty acid intake to be positively associated with weight gain and postpartum weight retention . In one study, the percent of calories from fat had only a weak positive association with weight gain, while percentage of calories from animal, saturated, and *trans* fatty acids had stronger associations . Single-meal studies do not support a differential effect on satiety of different fatty acids .

255. Evidence from prospective studies relating the proportion of protein in the diet with weight change is inconsistent . One intervention study that increased dietary protein intake from 12% to 25% of energy at a constant low-fat intake produced a greater magnitude of spontaneous weight loss than from a low-fat diet alone, after 6 months . These differences persisted, but were attenuated after consumption of the study diets for 12 months ; the evidence suggested that the high protein diet reduced visceral fat more than expected from fat loss.

256. Short term studies have consistently shown that alcohol having an acute stimulatory effect on appetite; however, results from prospective studies investigating alcohol consumption in relation to weight gain have been inconsistent . This is a complex area to study given the specific under-reporting of alcohol intake, confounding by other variables and ethical differences in conducting intervention studies .

Specific foods

257. A variety of specific foods have been investigated with regard to energy balance. Increased fruit and vegetable consumption has been associated with reduced weight gain in several prospective studies . Frequent nut consumption, a high-fat, energy-dense food, has been

shown not to lead to weight gain in well-controlled nut-feeding trials and prospective studies suggest frequent nut consumption is associated with reduced risk of weight gain ; residual confounding with other dietary and lifestyle differences may be a factor affecting interpretation of observational studies. Nuts are also relatively rich sources of protein and NSP and effects on satiety and faecal fat loss may be factors influencing the observed lack of weight gain .

258. Several large epidemiological studies have shown an inverse relationship between calcium intake and body weight or BMI ; however, a meta-analysis of 13 trials of calcium supplementation or increased provision of dairy products conducted for 12 or more weeks observed no statistically significant association with a reduction in body weight .

Dietary patterns

259. The investigation of dietary patterns and eating behaviour, rather than specific nutrients or foods, has become a focus of research. The Mediterranean or 'prudent' diet pattern characterized by high intakes of vegetables, fruits, legumes, nuts and olive oil, together with a reduced intake of meat and meat products and full-fat dairy products has been associated with reduced weight gain .

260. Short-term studies suggest habituation to large portion sizes may increase energy intake , but longer term interventions or prospective studies are lacking.

261. While two prospective studies have reported consumption of breakfast to be modestly associated with reduced weight gain, as compared with skipping breakfast , another study found no consistent association .

Conclusions

262. Methodological constraints are a severe limitation in defining the role of diet composition on the regulation of body weight. Overall, evidence suggests that traditional dietary patterns such as the Mediterranean diet, rich in fruit, vegetables and unrefined carbohydrate may be associated with decreased risk, while diets rich in energy-dense, nutrient-poor foods, typical of 'fast food', may increase risk of weight gain.

263. With regard to the regulation of body weight, it is important to consider diet in conjunction with physical activity. Consideration of energy balance and flux is required, rather than just energy intake and expenditure in isolation. This is the topic of Chapter 9.

References