



Childhood predictors of adult obesity: a systematic review

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OBJECTIVE: To identify factors in childhood which might influence the development of obesity in adulthood.

BACKGROUND: The prevalence of obesity is increasing in the UK and other developed countries, in adults and children. The adverse health consequences of adult obesity are well documented, but are less certain for childhood obesity. An association between fatness in adolescence and undesirable socio-economic consequences, such as lower educational attainment and income, has been observed, particularly for women. Childhood factors implicated in the development of adult obesity therefore have far-reaching implications for costs to the health-services and economy.

SEARCH STRATEGY: In order to identify relevant studies, electronic databases—Medline, Embase, CAB abstracts, PsycLit and Sport Discus—were searched from the start date of the database to Spring 1998. The general search structure for electronic databases was (childhood or synonyms) AND (fatness or synonyms) AND (longitudinal or synonyms). Further studies were identified by citations in retrieved papers and by consultation with experts.

INCLUSION CRITERIA: Longitudinal observational studies of healthy children which included measurement of a risk factor in childhood (< 18 y), and outcome measure at least 1 y later. Any measure of fatness, leanness or change in fatness or leanness was accepted. Measures of fat distribution were not included. Only studies with participants from an industrialized country were considered, and those concerning minority or special groups, e.g. Pima Indians or children born preterm, were excluded.

FINDINGS:

- Risk factors for obesity included parental fatness, social factors, birth weight, timing or rate of maturation, physical activity, dietary factors and other behavioural or psychological factors.
- Offspring of obese parent(s) were consistently seen to be at increased risk of fatness, although few studies have looked at this relationship over longer periods of childhood and into adulthood. The relative contributions of genes and inherited lifestyle factors to the parent–child fatness association remain largely unknown.
- No clear relationship is reported between socio-economic status (SES) in early life and childhood fatness. However, a strong consistent relationship is observed between low SES in early life and increased fatness in adulthood. Studies investigating SES were generally large but very few considered confounding by parental fatness. Women who change social class (social mobility) show the prevalence of obesity of the class they join, an association which is not present in men. The influence of other social factors such as family size, number of parents at home and child-care have been little researched.
- There is good evidence from large and reasonably long-term studies for an apparently clear relationship for increased fatness with higher birth weight, but in studies which attempted to address potential confounding by gestational age, parental fatness, or social group, the relationship was less consistent.
- The relationship between earlier maturation and greater subsequent fatness was investigated in predominantly smaller, but also a few large studies. Again, this relationship appeared to be consistent, but in general, the studies had not investigated whether there was confounding by other factors, including parental fatness, SES, earlier fatness in childhood, or dietary or activity behaviours.
- Studies investigating the role of diet or activity were generally small, and included diverse methods of risk factor measurement. There was almost no evidence for an influence of activity in infancy on later fatness, and inconsistent but suggestive evidence for a protective effect of activity in childhood on later fatness. No clear evidence for an effect of infant feeding on later fatness emerged, but follow-up to adulthood was rare, with only one study measuring fatness after 7 y. Studies investigating diet in childhood were limited and inconclusive. Again, confounding variables were seldom accounted for.
- A few, diverse studies investigated associations between behaviour or psychological factors and fatness, but mechanisms through which energy balance might be influenced were rarely addressed.

CONCLUSIONS AND RESEARCH PRIORITIES: The major research gap identified by the current review is the lack of long-term follow-up data spanning the childhood to adulthood period. This gap could in part be filled by: (i) follow-up of existing groups on whom good quality baseline data have already been collected; and (ii) further exploitation of existing longitudinal datasets.

Many of the risk factors investigated are related, and may operate on the same causal pathways. Inherent problems in defining and measuring these risk factors make controlling for confounding, and attempts to disentangle relationships more difficult. A given risk factor may modify the effect of another, and cumulative effects on the development of obesity are likely, both over time for specific risks, or at any particular time over a range of risk factors. An additional approach to addressing these issues may be to use large samples on whom more basic measures of risk factors have been collected.

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Specific issues that remain unresolved include:

- the mechanism by which SES in early life influences obesity in adulthood;
- whether the relationships between birth weight and maturation and later obesity persist after accounting for confounding factors;
- whether any relationships between dietary factors and activity and later fatness are due to a direct effect, or to tracking in dietary or activity behaviour;
- how psychological factors and behaviours influence energy balance, and therefore fatness.

A further neglected area of research is the identification of factors predicting the maintenance of a healthy relative weight, which may or may not be the opposite of predictors of obesity.

The challenge to future research remains to discern which are the important and modifiable factors, or clustering of factors, and effects over time, on the causal pathway to the development of obesity.

Keywords: systematic review; obesity; child; adult; predictors

Introduction

Objective

The aim of this systematic review was to identify factors in childhood which might influence the development of obesity in adulthood.

Background

The prevalence of obesity is increasing in adults¹ and children in the UK;² British children are getting fatter.³ A similar rise in prevalence of obesity, related to increasing affluence, is reported from many developed countries throughout the world.⁴ The association between adult obesity and adverse health outcomes, including diabetes, coronary heart disease, cancer and respiratory problems, is well documented,⁴ but there is limited evidence for an association between adolescent obesity and increased risks of adult morbidity⁵ and mortality.⁶ More immediate effects of developing obesity include psychosocial outcomes, with social isolation and peer problems more common in fatter children.⁷ Overweight adolescent women have lower educational attainment, lower incomes and are less likely to marry than those not overweight.⁸ If these relationships are indeed causal, then they imply far-reaching consequences for costs to the health services, and the economy. The limited data available suggest the direct medical costs might amount to 4–5% of total health care.⁹ In the USA, an annual cost of US\$4 billion has been attributed to loss of productivity due to obesity.⁹ The relationship between relative weight and morbidity and mortality may not be linear, but J-shaped, with an increased risk of adverse health outcomes reported at the lower extremes of fatness in many studies.⁴ The extent is unclear to which this relationship between underweight and an increased risk of adverse outcome reflects reverse causality, whereby smoking and pre-existing disease cause both underweight and increased morbidity and mortality.⁴

So far treatment of established obesity has been largely ineffective and therefore prevention is preferable.^{10–12} Existing data suggest that children may be more responsive to prevention than adults,¹³ and that risk factors identified in children may be more amen-

able to change. Despite the attractiveness of prevention strategies aimed at children, a cautious approach is required to avoid the possibility of unforeseen adverse consequences, particularly in respect of interruption of normal growth pattern in early childhood and distorted body image, excessive dieting and anorexia in adolescence. For instance, the recommendation for children over the age of 2 y in the USA that total fat intake amount to 30% of total calories or less¹⁴ has in some observational studies been associated with a greater risk of inadequate intake of several vitamins.¹⁵

The childhood period is also important for adult obesity because tracking of overweight, albeit moderate, is observed between childhood and adulthood. This topic has recently been reviewed.^{5,16} The figures vary according to the definition of obesity and length of follow-up, but fat children have a high risk of going on to become fat adults; for example in the 1958 British birth cohort, 38% of boys and 44% of girls above the 95th BMI centile at age 7 were obese at age 33.¹⁷ Even so, only a small proportion of fat adults were fat in childhood. It is likely that there are factors operating in early adulthood which promote obesity, but there may also be factors operating in childhood that promote adult obesity. It is still a matter of debate whether there are particular stages in childhood, during which physiological alterations increase the risk of later obesity. These stages are termed critical periods, and may include the prenatal period, the adiposity rebound (second rise in adiposity occurring at about 6 y), and puberty.¹⁸

In addition to the observed tracking of adiposity from childhood to adulthood, it has been suggested that lifestyle habits such as diet and activity levels may also track during childhood and into adulthood. There is some evidence that such tracking occurs,^{19–21} although relationships are modest. If lifestyle patterns established in childhood persist into adulthood, childhood becomes a target period for modification of lifestyle factors with a view to preventing obesity.

Obesity is a multi-factorial condition with wide-ranging causes including genetic, social, cultural and behavioural factors, all of which may interact. The current review takes a public health perspective, and therefore focuses on potentially modifiable factors.

There is an extensive literature concerning genetic factors and obesity, which at the gene level, is beyond the scope of this review. Moreover, as observed by Prentice,²² most obesities today must be caused by environmental and lifestyle factors in modern life, since the large increases in prevalence of obesity are occurring within a relatively constant gene pool. This does not mean that genetic effects are unimportant, but that the balance of genetic and environmental influences is changing. Genetic make-up undoubtedly contributes to obesity, but it is also highly likely that behaviours or lifestyle factors, which are potentially modifiable, contribute to the apparent heritability of obesity. The inheritance of phenotype parental fatness as a predictor of adult fatness, is therefore included in this review.

The definition of obesity is not without problems, particularly in childhood, during growth and maturation. Many different methods are currently in use to estimate body fatness or relative weight, and for each method various cut-off levels are used to describe overweight or obesity. The pros and cons of each method have been discussed elsewhere^{5,23} and these details are beyond the scope of the current review. In adults, BMI is the standard obesity assessment,^{24,25} although it is important to recognize that is only an indicator of obesity. For a given BMI, there is a large range of percentage body fat and thus the sensitivity (proportion of truly obese subjects detected by the measure), may be low.²⁶

Body weight (or BMI) can vary considerably in adult life, for example, by 10 kg (3 kg/m²) over 18 y.²⁷ The use of an arbitrary dichotomous (or categorical) classification of obesity (or grades of) will inevitably result in substantial numbers of individuals entering or leaving the 'obese' group over time. This is a disadvantage of using a cut-off to define obesity. In children, the validity of several convenient methods for assessing fatness, (skinfolds, relative weight, BMI) has been compared with underwater weighing.²⁸ The sensitivity (as above) and specificity (proportion of truly non-fat subjects detected by the measure) varied for the different methods and cut-off points used to define obesity. Despite reservations that BMI does not identify obese subjects as well as other methods, for example skinfold measurements,²⁸ the recent consensus report on obesity evaluation and treatment in children²⁹ concluded that BMI was a straightforward and useful measure of adiposity. To encourage consistency in defining fatness, the International Task Force on Obesity is developing a provisional international reference population and BMI standards to classify overweight and obesity, using age and sex specific curves.^{30,31} There may however be a good case for surveys to collect skinfold or body circumference data as well.⁵

To investigate the influence of factors in childhood on obesity in adulthood, several sources of evidence may be drawn on: cross-sectional, longitudinal and secular trend data, and intervention studies in both

children and adults. The strongest evidence however, comes from longitudinal studies, and it is these that form the basis for the current review. Where possible, studies starting in childhood and following through into adulthood have been used, but those longitudinal studies examining the associations between exposure of interest and fatness later in childhood have also been included.

Methodology and results

Methodology

The best evidence for assessing the impact of childhood predictors on adult obesity is provided by longitudinal studies. This review therefore aimed to identify all such studies.

Criteria for considering studies for this review

Inclusion criteria. (1) Types of studies: longitudinal observational studies in healthy children, of at least 1 y duration, in which a baseline measurement of a proposed risk factor was made in childhood, defined as under 18 y of age were included.

(2) Population: only studies where participants were from a developed, industrialized country were considered.

(3) Predictors: predictors of obesity (diet, physical activity etc.) were not pre-defined so that pre-conceived ideas about what may or may not be a risk factor for obesity were not imposed on the search strategy, leading to a biased group of studies being identified in the literature. Therefore predictors were not searched for specifically in the search strategy (Appendix II). All predictors addressed in the literature, which were relevant from a public health perspective and potentially amenable to change, were included.

(4) Outcome: any measure of fatness, leanness or relative weight, or change in fatness, leanness or relative weight was included. Absolute change in weight was considered only if it occurred wholly within adulthood (≥ 18 y), since height is assumed to be constant.

Exclusion criteria. (1) Type of study (quality): studies were excluded if they were less than 1 y in duration, or if basic information, such as numbers of participants, the ages at which they were measured, or sufficient definition of risk factor or outcome was lacking, or not referenced to a previous publication.

(2) Population: studies concerning minority groups, e.g. Pima Indians, vegans, children born preterm or to diabetic mothers, or other special groups, were excluded.

(3) Predictors: predictors such as smoking by the child, contraception, pregnancy occurring under the

age of 18, which usually concern older children, were not included.

(4) Outcome: studies including only a measure of fat distribution rather than overall fatness were excluded.

Search strategy. The following sources are included in the search process:

| | |
|----------------------------|-----------------|
| Medline | 1966–April 1998 |
| EMBASE | 1980–May 1998 |
| CAB Abstracts | 1973–May 1998 |
| PSYCLIT | 1996–March 1998 |
| Sport Discus | 1975–March 1998 |
| conference abstracts | |
| consultation with experts. | |

The electronic search strategies incorporated three concepts: childhood, fatness and longitudinal. Appropriate synonyms, misspellings and truncations were included. In addition, potentially relevant longitudinal studies known about at the time of searching were included specifically within the strategy. Search strategies used for each database are included in Appendix II. A list of experts consulted is given in Appendix III. *Proceedings of the North American Association for the Study of Obesity Meeting*, Cancun, 9–13 November, 1997,³² and the *8th International Congress on Obesity*, Paris, 29 August–3 September, 1998³³ were hand-searched.

Additional existing reviews relevant to the broader subject area, secular trend and ecological studies were consulted in order to provide a context for the studies identified for the current review. These studies were not identified by a systematic process. Studies published too recently to be identified by the search strategy, but identified by the review group in the course of reading, or brought to our attention by experts consulted, were included.

Process of study selection. Titles and abstracts of studies identified by electronic searches were examined on screen. Those thought to possibly meet inclusion criteria were obtained, along with papers identified by consulting experts. Full copies of these papers were examined to determine whether they met the study inclusion criteria.

Methodological quality of included studies. Apart from determination of whether studies were at least 1 y in duration and included adequate descriptions of study populations and data, methodological quality was not formally assessed. Although there is a body of research around quality issues for trials,³⁴ there is very little relating to observational data. In particular, there is little empirical research on the magnitude of the effect of different aspects of study design on the validity of observational studies. Theoretically it

would be possible to develop a quality scoring system based on factors like participant selection, sample size, length of follow-up, proportion of follow-up, and methods of measurement of outcome (objective/subjective). However, further complications arise due to the heterogeneity in definition of outcome, statistical methods employed to analyse results, and adjustment for a variety of confounding variables. Formal assessment of quality was therefore not carried out, due to difficulties in developing quality criteria for such a heterogeneous group of studies, and time constraints in doing so. Instead, limitations of studies identified are discussed within each section of the review.

Data extraction. A data extraction form was designed to include information on the following:

- study design, prospective or retrospective;
- study setting, population characteristics, subject recruitment;
- numbers of participants, invited, eligible, enrolled;
- numbers of participants at each follow-up;
- ages at which baseline and follow-up measures were made;
- definition and measurement of predictors and outcome;
- method of analysis;
- main findings.

This information is included in the summary tables of studies, presented in Appendix I, Tables A1–11.

Analysis. Formal statistical combination of results, even within subject headings, was clearly inappropriate due to the marked heterogeneity of all aspects of identified studies. Although it would be useful to be able to estimate the magnitude of the effect of each predictor, effect size varies depending on the quality issues discussed above, the definition of predictor variables, and on whether adjustments have been made for covariates. Study results are therefore presented in tabular form, grouped by categories of risk factor.

Steering group and collaborators. An advisory group of experts in aspects of obesity, systematic reviews and/or statistics was formed and met in December 1997 for discussion. Subsequently, members were consulted individually about sections of the review within their area of expertise. Members of the steering group and collaborators are given in Appendix III.

Results

The electronic database search produced 22,359 hits:

| | |
|---------------|--------|
| Medline | 10,212 |
| Embase | 10,169 |
| CAB Abstracts | 661 |

| | |
|--------------|-----|
| PsycLIT | 535 |
| Sport Discus | 782 |

Numbers of studies identified are given within each section of the review. Several studies were published and identified after the search strategy was run and included in the review. Five non-English publications (Italian, Czech, French, Dutch, Japanese)^{35–39} were identified that potentially contained relevant information and were translated. Only one was included in the review.³⁹

Genetic — inheritance of phenotype

Introduction

It is widely acknowledged that genetic factors play a role in obesity, and that fatness is, to some extent ‘heritable’. This topic has formed the subject of several reviews in the last decade.⁴⁰ How much variation in fatness is explained by genetic factors, rather than environmental factors, is far more controversial. There is little agreement about the proportion of heritability due to genes and that due to inherited lifestyle habits.

Essentially three types of studies address the phenotypic inheritance of fatness: family, twin and adoption studies. Family studies may be further divided into those investigating parent–offspring fatness relationships and those investigating similarity between siblings, although obviously many family studies examine both. Studies of all types are largely cross-sectional, and therefore estimate heritability at a single time-point. For the current review, our primary focus was the longitudinal relationship between parental and offspring fatness, but since such studies are scarce, we also considered cross-sectional data from family, twin and adoption studies coincidentally identified by the search strategy.

Methodology

From the results of the search strategy, we identified studies reporting longitudinal data, where data concerning fatness of offspring were measured at multiple time-points, and parental fatness was assessed at least once. However, we also considered cross-sectional data from family, twin and adoption studies identified by the search strategy. It should be emphasized that these latter studies were not searched for systematically and thus we were not attempting to identify all such studies in the literature.

Results

Limitations of the data. The main problem in investigating the influence of parental fatness is that it represents the cumulative result, not only of genetic

make-up, but of lifestyle effects and behaviours which may also be inherited. It is important that this issue is acknowledged when, as seen in subsequent sections of the review, it might be desirable to adjust for a potential confounding effect of parental fatness.

Three studies were identified which reported multiple measures of fatness from childhood into adulthood,^{41–43} and a further five studies which reported multiple measures of fatness within childhood.^{44–48}

Findings. Longitudinal studies—childhood to adulthood: two of the three studies which followed through into adulthood included a single measure of parental fatness,^{41,42} the third included several measures over the study course.⁴³ The findings are summarized in Appendix I, Table A1; all studies observed that offspring fatness increased with parental fatness, and at all ages, from childhood into adulthood.

Data from Kaplowitz *et al* suggested that this relationship may be stronger between mothers and their offspring than fathers and offspring, and that the mother–offspring relationship strengthens over time.⁴¹ Whitaker *et al* found that parental obesity was a more important predictor of offspring obesity earlier in childhood (< 6 y), becoming less important with increasing age of the child.⁴³ Data from Lake *et al* showed that parental obesity influences tracking of the offspring’s own obesity, which is much stronger if both parents are obese.⁴²

Other types of studies: all other studies identified, family and sibling,^{39,44–63} twin^{64,65} and adoption studies,^{66–68} support the view that genetic factors contribute to fatness level, although the estimated effects vary between studies (Appendix I, Tables A2–4).

Summary

The presence of a genetic influence on obesity has been well researched, although the size of the genetic contribution and whether it differs during childhood, has not been extensively investigated. Existing studies, however, show consistency in that parent obesity is an important factor in predicting adult obesity of offspring, and that offspring of obese parents who themselves are fatter in childhood, may be at particular risk.

Impressions

There would seem to be little doubt that the development of fatness has a genetic component. The eight longitudinal studies we identified which met our inclusion criteria demonstrated a positive relationship between parent and offspring fatness, which was universally supported by the additional range of studies considered. What is far less clear, and is important from a public health perspective, is how much of the variation in fatness is due to the genetic component, and how much to the environment. Also

important is the influence of the environment on the genetic component, i.e. gene–environment interactions. For example, a genetic predisposition to select high fat foods, or to perform little physical activity will be dependent on the availability of such foods or the necessity of performing activity. The strong relationship between parental and offspring fatness is likely to represent both genetic factors and cumulative environmental influences including learnt lifestyle behaviours. Adoption and twin studies are more powerful tools than family studies in resolving genetic and environmental effects, although those we identified were cross-sectional and therefore did not meet our original inclusion criteria. As seen in this review and others,⁴⁰ these studies tend to give high estimates of heritability, and argue that little variation in fatness is due to the environment.⁶⁸ Each design however, has its disadvantages, and incorporates assumptions that may well be violated, with implications for the strength of the findings.⁴⁰ An integrated approach, incorporating the best aspects of each design, may add to current understanding of this nature–nurture debate.⁴⁰ These questions are largely beyond the scope of the current systematic review, and have been more comprehensively discussed elsewhere.⁴⁰

One of the major disadvantages of family studies, is that most include only one time-point and therefore parents and offspring are measured at different ages. If genetic and environmental influences vary with age or different genetic/environmental factors operate at different ages, observed relationships between parents and children will be weakened. The longitudinal studies with follow-up data in adulthood identified for this review partly address this issue. Although Kaplowitz *et al*⁴¹ found that mother–child correlations increased with age of the child, Lake *et al*⁴² found that mother–child correlations did not increase over 11 y, and Whitaker *et al*⁴³ observed a decrease in likelihood of a child of an obese mother being obese, with increasing age of the child. The study of Kaplowitz⁴¹ was comparatively small, and the outcome measure was based on skinfold thicknesses, whereas the other studies used BMI, which might contribute to these differences.

Findings from both studies of Lake *et al*⁴² and Whitaker *et al*⁴³ suggest that, in addition to parent obesity being a risk factor for adult obesity in their offspring, parental obesity is particularly important for the development of fatness in childhood. In the study of Whitaker *et al*, below 6 y, parent obesity is especially predictive of adult obesity in the offspring, but above 9 y, fatness of the child becomes more important.⁴³ The child of obese parent(s) is therefore at increased risk of becoming fat early in life, and once relatively fat, he/she is more likely to be so later in adulthood. Data from the British 1958 birth cohort⁴² show that obese children of obese parents are much more likely to be obese in adulthood than obese children of normal weight parents. As the prevalence of obesity in the population increases, more children

will show stronger tracking of fatness into adulthood, although the contributions of genetic and modifiable lifestyle factors to the tracking of fatness remain largely unknown.

Social factors

Introduction

One of the most striking facts about obesity is the negative relationship in adults between obesity and socio-economic status (SES) in the developed world, especially among women.⁶⁹ About 10 y ago, Sobal and Stunkard published a comprehensive review of SES and obesity,⁶⁹ which included 144 studies from all over the world, of men, women and/or children. Despite the fact that these studies used a range of measures of SES and fatness, and spanned a period of 45 y, a strong negative relationship between SES and fatness was obvious in women across the developed world, such that fatness increased from higher social groups (professional and management) to lower social groups (unskilled and manual). Of 54 studies of women, 85% demonstrated a negative relationship, 13% showed no relationship, and only 2% a positive relationship (greater fatness among higher social groups). Among men and children, however, the relationship was far less consistent, with studies more evenly spread over positive, negative and no relationships. In stark contrast, in developing countries, not a single study found a negative relationship between SES and fatness in women. Indeed, the vast majority reported a positive relationship, in both men and women, and in children.

UK data concerning SES and BMI among adults is collected regularly as part of the Health Survey for England. Both in the first survey in 1991,⁷⁰ and in 1996 (most recent published data for adults)⁷¹ a lower mean BMI was demonstrated in women from non-manual, rather than manual households, but the opposite was found in men. Both surveys demonstrated a clear social gradient in the prevalence of obesity in women, but no clear pattern in men.

Since 1995, children have been included in the Health Survey. Combined data from 1995–1997⁶³ show no pattern in social class and prevalence of overweight in males aged 2–15 y or 16–24 y, or in females aged 2–15 y. In females aged 16–24 y, prevalence of obesity was higher in manual than non-manual social classes. In addition, a recent study of social factors and fatness in children found no consistent influence of social class, parent education, number of parents, hours worked by mother outside the home, free school meals, or family size on fatness of children aged 5–11 y.⁷²

However, these data are cross-sectional, as were most other studies in Sobal and Stunkard's review, and are therefore unable to determine long-term

relationships between SES in childhood and adult obesity. Associations between low SES and greater fatness in studies of adults have been attributed to two main causal models: low SES may promote the development of fatness or, alternatively, greater fatness may lead to downward social mobility and thereby lower SES. There is some evidence from longitudinal studies to support both models, but the current systematic review examines evidence on the former, that low SES in childhood promotes the development of fatness later in life. Establishing the direction of association with SES is likely to provide clues on the aetiology of adult obesity, especially if relationships are investigated for several life stages, since this might suggest the likely timing of risk factors.

SES has not been the only social measure proposed to be related to the development of obesity, and other social factors reported in the literature, such as family structure and functioning, age of mother at birth of child, rearing area, and scholarly abilities in childhood were included in this review. Studies concerning these factors (except mother's age) had to fulfil our initial criteria and be of at least 1 y in duration.

Methodology

All longitudinal, observational studies identified by the search strategy, which reported an assessment of social group or other social measure during childhood (< 18 y), and fatness at least 1 y later, were included in this section of the review. Some cross-sectional studies were also included, for reasons described below.

Results

Limitations of the data. SES is recognized to be a crude summary measure of a range of circumstances, making it difficult to determine what it is about social group that relates to obesity. Social class cannot itself 'cause' obesity, but might influence energy balance via characteristics of socio-economic groups related to material circumstances, behaviour or knowledge. Consideration of some of these measures might help disentangle and inform on the association between SES and obesity.

Since the prevalence of overweight in women is greater in lower social groups, maternal, if not also paternal fatness is likely to be a confounding variable in the relationship between SES and subsequent fatness of offspring. Only two studies included a measure of parent relative weight in analyses.^{73,74}

Certain problems were encountered with identifying studies for this section of the review. In several longitudinal studies it was difficult to elicit when SES had been assessed. Both those studies in which it was clearly stated, and those in which it seemed highly likely that SES was measured at baseline, were included. Further questions arose concerning the

stability of parental occupation, income and education over time, and the importance of these factors being assessed at least 1 y before offspring fatness. We considered parent education likely to be reasonably constant, and so whether it was measured at least a year prior to offspring fatness unimportant. We therefore considered additional studies identified by our search, which measured parent education level and fatness in offspring in child- or adulthood, cross-sectionally. It should be emphasized that these studies were not searched for systematically, but were coincidentally identified by the strategy, and will form (probably a small) subset of all published cross-sectional studies investigating this relationship.

Thirty papers and one abstract were identified from 21 studies investigating the effect of social factors in childhood on subsequent fatness (Tables 1 and 2, and Appendix I, Table A5). Twelve studies (18 papers and one abstract)^{75–93} spanned the period from childhood to adulthood; one paper and one abstract were from the 1946 UK birth cohort study,^{77,78} two papers were from the Tecumseh Community Health study,^{80,89} three papers were from the same study by Lissau *et al.*,^{82–84} and four papers were from the 1958 UK birth cohort study.^{90–93} Nine studies (12 papers)^{61,73,74,94–102} spanned a period within childhood; two papers were from the same study by Agram *et al.*,^{73,94} and three papers were from the same study by Kramer *et al.*^{74,96,97} Papers from the 1958 UK birth cohort^{90–93} and the Tecumseh Community Health study,⁸⁹

Table 1 Childhood to adulthood—twelve studies

| Relationship | Numbers of studies | | |
|--------------|--------------------------|--------------------------------------|---------------------------|
| | Women | Men | Women + men combined |
| Negative | 4 ^{77,81,89,93} | 8 ^{76,77,81,85,86,88,89,93} | 4 ^{78,79,82,87a} |
| None | 1 ⁷⁵ | 1 ⁷⁵ | 0 |
| Positive | 0 | 0 | 0 |
| Total | 5 | 9 | 4 |

Negative relationship: lower risk of fatness associated with higher social group. Positive relationship: higher risk of fatness associated with higher social group.^aThe study of Lissau *et al.*⁸² is included here, but this study reported negative or no relationship depending on SES variable used, and therefore the SES-fatness relationship could also be classified as none.

Table 2 Within childhood—nine studies^a

| Relationship | Numbers of studies | | |
|--------------|---------------------|------------------|------------------------|
| | Girls | Boys | Girls + boys combined |
| Negative | 3 ^{98–100} | 1 ⁹⁸ | 3 ^{61,73,101} |
| None | 0 | 1 ¹⁰⁰ | 2 ^{74,95} |
| Positive | 0 | 1 ⁹⁹ | 0 |
| Total | 3 | 3 | 5 |

Negative relationship: lower risk of fatness associated with higher social group. Positive relationship: higher risk of fatness associated with higher social group.^aStudies of Garn *et al.*⁸⁹ and Lasker *et al.*⁹⁰ (interim reports of childhood to adulthood studies) not included in table. See text for relationships.

reported follow-up measurement at several time-points, in childhood as well as adulthood. Where more than one paper from a study was identified, only the most relevant, or where more than one paper is relevant, only the most recent paper is cited when the study is referred to. Studies were generally large, with over half of sample sizes more than 1500.

Findings. (i) *Childhood to adulthood:* a total of 12 studies^{75–93} measured the influence of social factors in childhood on fatness in adulthood, with a duration of between 10 and ~55 y (Table 1 and Appendix I, Table A5). All studies examined the influence of SES, based on parent(s) occupation,^{76,79,82,86–88,93} parent(s) education,^{80,82,85} a composite of the two,^{77,81} or family income.^{75,89}

Of five studies of women, four found a negative relationship,^{77,81,89,93} and one found no relationship⁷⁵ between childhood SES and subsequent fatness. Nine studies included men: eight observed a negative relationship^{76,77,81,85,86,88,89,93} and one no relationship.⁷⁵ Four studies analysed data from men and women combined: three showed a negative relationship,^{78,79,87} the other showed either a negative or no relationship depending on the SES variable in question.⁸² Three studies looked at social mobility (between class of origin and own class) and adult obesity in men, all finding no relationship.^{76,77,81} Two studies included women, and both reported that the prevalence of overweight was lower in the upwardly mobile than in the downwardly mobile groups.^{77,81}

Additional social factors were investigated in several European studies which showed scholarly difficulties/special education,⁸³ parental support, poor housing, child's hygiene,⁸⁴ single parent families⁸⁵ and geographical rearing area^{85,87} to be related to later fatness, although some of these factors were no longer predictors after adjustment for other covariates. The influence of family size on fatness seems to be inconsistent, with greater fatness associated with both increasing and decreasing number of children in the family.^{85,86} Two studies found no consistent influence of birth order on later fatness.^{79,86}

(ii) *Within childhood:* follow-up measurements of 11 studies fell within childhood: nine studies based wholly within childhood^{61,73,74,95,98–102} and two child-

to adulthood studies with follow-up measurements in childhood.^{89,90} Once again all studies assessed SES, based on either parent(s)'s occupation,^{61,90,98,100} parent(s)'s education,^{73,80,98,99,101} a combination of the two,^{74,95,98} or family income.¹⁰² Study duration was 2–17 y.

Of the nine childhood studies, (Table 2 and Appendix I, Table A5) six studies combined data from boys and girls^{61,73,74,95,101,102} and three observed a negative relationship between SES and fatness at 6–17 y.^{61,73,101} Two studies found no association between SES and fatness,^{74,95} and in one study the relationship was not explicit.¹⁰² One study observed a negative relationship in boys and girls separately,⁹⁸ and a further study found a negative relationship in girls but not boys.¹⁰⁰ The ninth study found a negative relationship in girls, although many were missing from follow-up (Table A5), and that the proportion of overweight and severely overweight boys combined, was *less* in those whose parents had lower levels of schooling compared with the *whole study population*.⁹⁹ This finding is probably due to the particular comparison made, since prevalence of overweight and severe overweight was lower in not only the lowest, but also the highest of three parental education groups, than in the middle group.

In the two studies which included parental fatness in analysis, the negative relationships between parent education and childhood BMI persisted after adjustment for parental BMI,⁷³ whereas in the other study, SES was not a significant predictor of child obesity in a multivariate model.⁷⁴

The two studies with several repeat follow-up measurements^{89,90} found no, or even tending towards positive, relationships between SES and fatness in younger children, which became negative relationships in older children, strengthening with age into adulthood. Within childhood, one study looked at social mobility,⁹⁰ but found no influence on BMI at age 7, 11 or 16 y in boys and girls combined. A British study found no influence of the state of the home or care of children in the first 3 y of life on obesity at 12 y, and that more obese than non-obese children, had mothers over 35 y at birth.⁶¹

(iii) *Cross-sectional studies:* seven studies (eight papers)^{8,103–109} investigated the cross-sectional rela-

Table 3 Seven cross-sectional studies

| Relationship | Numbers of studies | | | | | | | |
|--------------|----------------------|----------------------|--------------------|------------------|------------------|-----------------|-----------------------|----------------------|
| | Girls | | | Boys | | | Girls + boys combined | |
| | Black | White | Mixed race | Black | White | Mixed race | Black | White |
| Negative | | 2 ^{106,109} | 2 ^{8,107} | 1 ¹⁰⁹ | | 0 ^a | | 2 ^{103,108} |
| None | 2 ^{106,109} | | | | 1 ¹⁰⁹ | 1 ^{8b} | | 1 ¹⁰⁴ |
| Positive | | | | | | | | |
| U-shaped | | | | | | | 1 ¹⁰⁴ | |

Negative relationship: lower risk of fatness associated with higher social group. Positive relationship: higher risk of fatness associated with higher social group. ^aThis becomes one study if Khoury *et al*¹⁰⁷ are included in this group. ^bThis becomes two studies if Khoury *et al*¹⁰⁷ are included in this group. Khoury *et al*¹⁰⁷ found either a negative or no relationships depending on outcome variable used.

tionship between parental education level and fatness in their offspring (Table 3 and Appendix I, Table A6); two papers were from the NGHS of NHLBI study.^{105,106} Parental education level was assessed while their offspring were children in five studies,^{103–108} when their offspring were adults in one study,¹⁰⁹ and during a transitional child-to-adult period in one study.⁸ Table 3 shows that some studies present data from White and Black participants separately, since studies in the USA have demonstrated ethnic differences in the prevalence of, and relationships between, risk factors and obesity.¹¹⁰ Among studies which analysed girls/women separately, two studies^{8,107} found a negative relationship between SES and fatness, and two studies which also analysed by race found a negative relationship in White girls but not in Black girls.^{106,109} A study of 16–24-y-old males found no effect of SES on fatness,⁸ whereas a study in boys found a negative relationship with BMI, but none with triceps skinfold.¹⁰⁷ Three studies report results for girls and boys combined, one found a U-shaped relationship in Blacks and no relation in Whites,¹⁰⁴ the other two studies found negative relationships.^{103,108}

Summary

The longitudinal studies identified for the present review show that both in males and females, there is a remarkably consistent negative relationship between SES in childhood and fatness in adulthood. This negative relationship is not quite so consistent when fatness is measured in childhood, or cross-sectionally. Social mobility, the change between social class of origin and own social class later in life, appears to influence obesity only in women, in whom prevalence of overweight is less in the upwardly than the downwardly mobile. The influence of social factors other than social class, such as family size, number of parents at home and child-care, has not been well researched.

Impressions

The longitudinal evidence identified for the current review therefore contrasts with that from cross-sectional studies in showing a strong relationship with SES among both sexes and not, as the latter show, an effect that is largely confined to women.⁶⁹ Of a total of 21 longitudinal studies, 18 found a negative relationship between some measure of SES and later fatness. Three studies found no relationship between SES and fatness in childhood, but only one study found no relationship between SES and fatness in adulthood.⁷⁵ In the latter study of Arnesen *et al*,⁷⁵ SES was self-reported retrospectively as whether economic conditions in childhood were very difficult, difficult, good or very good, rather than based on a more objective measure, which might contribute to the lack of relationship. In the present review, the consistency of the negative relationship was mirrored by those cross-sectional studies of parent education level and offspring fatness identified by the search strategy.

These cross-sectional studies were included in the review on the basis that adult 'social class'/occupation/income might change quite considerably over a number of years, whereas education is likely to be more constant. We recognize however, that this assumption may differ across countries, and is possibly dependent on factors such as access and cost of education. For example, the USA has a history of payment for further education and a larger proportion of mature students than the UK, where until recently further education has been free, and largely taken up on finishing school. If however, it is assumed that parent education is reasonably constant, it becomes irrelevant whether it is measured at least a year prior to offspring fatness, and therefore cross-sectional studies coincidentally identified by the search strategy and concerning this relationship were included.

Studies investigating social mobility showed no influence of social mobility on fatness in men, whereas women show the prevalence of obesity of the social group they join. Further evidence for social mobility effects in women but not men is available in two large additional studies.^{8,111} Data from the British 1958 birth cohort demonstrate that, at 23 y, women who had been obese at 16 y had significantly fewer years of schooling and lower earnings than their non-obese peers.¹¹¹ These relationships were not seen in men. Similar findings have been reported from the USA.⁸ Thus in women, a cycle is created where lower SES leads to greater fatness, which in turn tends to decrease SES. In men, the former part of the cycle appears to be weaker and the latter not to exist. Negative attitudes to obesity are more prevalent in girls than boys.¹¹² Dornbusch *et al*¹¹³ found that girls were dissatisfied with their weight because it represented fat, but boys were satisfied because, to them, weight represented muscularity. In the same study, the desire to be thin among girls increased with social class, whereas it differed little with social class for boys. It has been suggested that girls are highly aware of the value society places on attractiveness in women, and see thinness and attractiveness as synonymous.¹¹⁴ Furthermore, insecurity in adolescent girls may increase their compliance with societies' standards. Thus, as Sobal and Stunkard summarize,⁶⁹ the circumstances and values of life in the SES into which one is born may transmit pressures for thinness to adolescent girls during their socialization.

Studies to date therefore demonstrate a consistent pattern regarding social mobility and fatness in women. Even so, social mobility cannot account for SES differences in obesity, since these differences have now been shown in several studies to pre-date the mobility. Importantly, strong associations have been found between fatness and social class of origin. Thus, in developed countries, the longitudinal relationship between childhood SES and child or adult fatness, and the cross-sectional relationship in women⁶⁹ would appear to be robust, whereas cross-sectional relationships in men and children, and even

longitudinal relationships in young children seem to be less so. Interestingly, this pattern does not seem to depend on the measure of SES, and holds across several decades, during which social patterns have changed considerably. Although education, occupation and income are related, it could be argued that each represents quite different aspects of SES. Over the last 50 y, the proportion of the UK population completing school has increased greatly, as has the percentage of those entering further education. At the same time, as reported in a recent government publication,¹¹⁵ the nature and organization of paid work has undergone major transformations. The economy has shifted from heavy industry and manufacturing towards service sector employment. Women's participation in the workforce has increased markedly, part-time working for both men and women has increased and full-time working decreased. A greater proportion of the population is now outside the labour force, due to unemployment, early retirement and education. Meanwhile, the average household disposable income has grown, yet has not been evenly distributed across the population. The gap between high and low incomes has increased and the proportion of households living on low incomes doubled between 1961 and 1990. These trends are not unique to Britain: other developed countries also show persisting or widening socio-economic inequalities.¹¹⁵ Other social factors also show changing patterns. Households have been getting smaller, with the number of UK households increasing by 44% between 1961 and 1995, while the size of the population increased by only 12%. The number of children per family has decreased, and the average age of the mother at first birth has increased. The percentage of households headed by lone parents with dependent children has risen from 2% in 1961 to 7% in 1995–1996.

It is intriguing therefore, that while social patterns have been shifting in the developed world, a consistent relationship between SES and fatness has emerged, although exactly what it is about SES that promotes the development of greater fatness remains largely unknown. Being born into a particular social class cannot itself 'cause' obesity, but characteristics of socio-economic groups related to material circumstances and behaviour or knowledge, which ultimately influence energy balance, might. Attitudes towards obesity in developed societies however, may provide some explanation of the relationship to SES. Obesity is far more severely stigmatized in women than in men, both socially¹¹⁶ and economically.

Intra-uterine growth

Introduction

Birth weight is a crude indicator of growth *in utero*, and an association between birth weight and adult

obesity might therefore implicate foetal environment in the development of obesity in adult life. There are many factors which operate to determine the development of the foetus, and birth weight has been observed to be related to, amongst other factors, socio-economic status,¹¹⁵ gestational age,¹¹⁷ maternal smoking,¹¹⁸ gestational diabetes¹¹⁹ and famine.¹²⁰ Such factors could potentially confound the observed relationship between birth weight and later fatness. There have been several recent suggestions for mechanisms which may contribute to a birth weight–later fatness relationship. Higher birth weights and greater levels of obesity in offspring of mothers with gestational diabetes suggest that development of the hypothalamus/pituitary/adrenal axis, in particular cortisol and insulin secretions, may alter foetal programming and development. Metabolic responses of adipocytes, muscles and liver tissue, and efficiency of macronutrient oxidation, may also be determined, to some extent, *in utero*.

Secular increases in birth weight in developed countries, including the UK,¹²¹ suggest that if greater weight at birth is positively correlated with fatness in adulthood, this may partly underlie the trend in increasing obesity.

Methodology

All longitudinal, observational studies identified by the search strategy, which reported birth weight and a measure of fatness at least 1 y later, were included in this section of the review. Both studies reporting measured birth weights, and birth weights reported by parents/guardians were included.

Results

Limitations of the data. The relationship between birth weight and later fatness was investigated in 19 study populations, reported in 20 publications (Table 4 and Appendix I, Table A7). Eight studies (seven papers) spanned from childhood into adulthood;^{77,79,85,122–125} one paper by Curhan *et al*¹²³ included data from the Nurses Health Studies I and II, the remainder falling within the childhood period (< 18 y).^{47,54,57,63,74,96,97,99,126–130} Three papers were from the same study by Kramer *et al*^{74,96,97} Unfortunately, although gestational age is a major determinant of birth weight, only three studies^{47,85,125} attempted to deal with gestational age in the analyses. Five studies reported on parental fatness^{47,54,57,74,123} (Nurses Health Study I in the paper by Curhan *et al*¹²³). Two studies made some attempt to investigate the effect of parental fatness on the birth weight–later fatness relationship.^{57,123} Guilleme *et al*⁵⁴ observed that maternal (but not paternal) BMI was positively related to birth weight but did not make an adjustment in analyses. O'Callaghan *et al*⁴⁷ did not include parental fatness and birth weight in the same multi-

Table 4 Relationship between birth weight and subsequent fatness

| Author | Age at outcome | Outcome measure | Relationship |
|---|-----------------|---|---|
| Outcome in adulthood | | | |
| Allison <i>et al</i> ¹²² | 28–52 y | BMI | Positive |
| Braddon <i>et al</i> ⁷⁷ | 36 y | BMI | Positive (males) None (females) |
| Charney <i>et al</i> ⁷⁹ | 20–30 y | Overweight based on weight for height and age | Positive |
| Curhan <i>et al</i> ^{123a} | 58 y (NHS I) | BMI | Positive (females) |
| Curhan <i>et al</i> ^{123a} | 36.5 y (NHS II) | BMI | Positive (females) |
| Hulman <i>et al</i> ¹²⁴ | 28 y | Obesity based on BMI | None (sexes combined) |
| Rasmussen and Johansson ⁸⁵ | 18.2 y | Overweight based on BMI | Positive (for higher birth weights only) |
| Sorensen <i>et al</i> ¹²⁵ | 18–26 y | BMI | Positive (males) |
| Outcome in childhood | | | |
| Barker <i>et al</i> ¹²⁶ | 14–16 y | BMI Subscapular skinfold Triceps skinfold | Positive ($P=0.08$) Negative (females) None |
| Fomon <i>et al</i> ¹²⁷ | 8 y | BMI | Positive in formula fed males None in formula fed females None in breast fed males or females |
| Guillaume <i>et al</i> ⁵⁴ | 6–13 y | BMI | Positive (sexes combined) |
| Harland <i>et al</i> ¹²⁸ | 12–16 y | BMI | None (sexes combined) |
| Kramer <i>et al</i> ^{74,96,97} | 2 y | BMI Sum triceps + subscapular + suprailiac skinfolds | Positive at 1 and 2 y Positive at 1 and 2 y (sexes combined) |
| Maffeis <i>et al</i> ⁵⁷ | 12 y | Obesity based on BMI | Positive (males and females) |
| O'Callaghan <i>et al</i> ⁴⁷ | 5 y | Obesity based on BMI | Positive (sexes combined) |
| Seidman <i>et al</i> ⁹⁹ | 17 y | Overweight based on BMI | Positive (males) |
| Taylor <i>et al</i> ¹²⁹ | 8–11 y | BMI | Positive (males) |
| Zive <i>et al</i> ¹³⁰ | 4 y | BMI Sum triceps + subscapular skinfolds | Positive Positive |
| Health Survey for England ⁶³ | 2–15 y | BMI | Positive |

^aTwo studies reported in one publication.

variate model, and Kramer *et al*⁷⁴ reported a positive relationship between birth weight and later fatness despite adjusting for parental fatness. Five studies considered social group,^{47,74,99,125,126} but only four of these included birth weight and social group in the same multivariate model.^{74,99,125,126}

Findings. (i) *Childhood to adulthood:* seven out of eight studies investigating the influence of birth weight on adult fatness used BMI as a measure of fatness,^{77,85,122–125} the eighth using weight for height and age.⁷⁹ Five studies observed a positive relationship, with adult BMI increasing with increasing birth weight,^{85,122,123,125} and one study found no association.¹²⁴ This last study is the smallest, including only 137 subjects, while four of the other five are large studies including between 4062 and 92,940 subjects. The study of Braddon *et al*,⁷⁷ with a total sample size of 3249 men and women combined, found a positive relationship in men, but not in women.

(ii) *Within childhood:* thirteen publications from 11 studies examined the relationship between birth weight and fatness later in childhood, all of which included BMI as an outcome measure.^{47,54,57,63,74,96,97,99,126–130} Nine of 11 studies^{47,54,57,63,74,99,126,129,130} found positive relationships between birth weight and BMI, although in the smallest of these studies ($n=216$) the relationship was of borderline significance.¹²⁶ Six of the studies reporting significant positive relationships are relatively large, reporting results from between 1028 and 20,7467 subjects.^{47,54,57,63,99,129} Both positive and no

significant relationships between birth weight and BMI were seen in one study,¹²⁷ depending on gender and infant feeding method. This was a relatively small study (432 subjects with follow-up data) and the data were analysed in subgroups by mode of infant feeding and gender. The other study¹²⁸ reporting no significant relationship between birth weight and BMI was again a small study (188 subjects). Three studies examined associations between birth weight and skinfold measurements (in addition to BMI), and results were inconsistent; a negative relationship was observed with subscapular skinfold,¹²⁶ and positive relationships with triceps¹²⁶ or sum of two¹³⁰ or three skinfolds.⁷⁴

Summary

On the whole, the association between birth weight and later BMI, as reported in the longitudinal studies identified for this review, would appear to be consistent and positive. Study sample sizes were generally large, and interestingly, those studies observing a negative, or no relationship, were those with the smallest sample sizes either in total,^{124,126,128} or due to analyses by sub-group.¹²⁷ Despite the apparent consistency in the larger studies, in those which attempted to take account of potential confounders, the relationship was less clear.

Impressions

Birth weight is strongly related to gestational age,¹¹⁷ yet only two studies attempted to account for this in analyses. Sorensen *et al*¹²⁵ included an adjustment for

gestational age, and O'Callaghan *et al*¹⁴⁷ adjusted for size for gestational age. Both studies reported positive relationships between birth weight and adult BMI after these adjustments, suggesting that the relationship may be independent of gestational age.

Although the positive relationship between birth weight and subsequent fatness appears consistent, the picture is less clear when other confounding variables, such as parental fatness and socio-economic status (SES) are taken into account. Results from two studies suggest that maternal fatness influences the relationship.^{57,123} One study reported on the variation in the relationship between birth weight and later BMI with reported mother's figure at age 50 y.¹²³ Only for mothers with a medium figure (rather than thin or heavy), was there an increased likelihood of being in the highest vs the lowest BMI quintile with increasing birth weight. A second study reported⁵⁷ that within the 1st and 2nd quartiles of maternal BMI, prevalence of obesity increased with birth weight > 3500 g, but this relationship did not hold in the 3rd and 4th quartiles of maternal BMI. Since only two studies investigated the influence of parental fatness and findings were inconsistent, this issue remains unresolved.

The relationship between birth weight and fatness might also be confounded by SES, and the relationships between the three variables are not all what might be expected. Babies born to parents of lower social groups tend to have lower birth weights.¹¹⁵ Adult fatness, particularly in women, is more common among those of lower social group of origin. One might therefore expect lower birth weight to be associated with greater subsequent fatness, which the studies identified for this review show is generally not the case. SES would be expected to act as a confounding variable, decreasing the likelihood of observing a positive relationship between birth weight and adult fatness. Unfortunately none of the studies which considered SES, reported on the relationship between birth weight and fatness, before and after adjusting for SES.^{47,74,99,125,126,131} Four studies reported adjusting for SES.^{74,99,125,126} In these four studies, birth weight remained a positive predictor of BMI after adjustment for SES.

In the study of Fomon *et al*,¹²⁷ the presence of a relationship varied according to infant feeding method, suggesting that the birth weight–later fatness association may be moderated by other factors which also potentially influence later fatness, although sample sizes in this study were relatively small.

The influence of birth weight on BMI does not seem to be uniform across the range of birth weight. In those studies which reported later BMI by birth weight, or the effect of birth weight relative to a reference group (mid-range of the birth weight distribution), a J-shaped relationship was observed. Lower birth weights therefore were also observed to be associated with an increased risk of greater BMI later in life, although the effect was generally not as great as that of high birth weights.^{47,99,123,124}

Unfortunately in the studies identified, it was not possible to determine whether this observation was confounded by socio-economic characteristics.

Allison *et al* reported results from a twin study, in which they too, found birth weight correlated with adult BMI. They went on to attempt to investigate whether this relationship persisted after controlling for genetic factors. They argued that if intra-pair differences in birth weight in monozygotic twins were related to differences in adult relative weight, then genetic factors could not be solely responsible for the relationship, and the intra-uterine environment must be important. They did not find that birth weight differences were related to adult differences in relative weight, and therefore suggested that, after allowing for genetic factors, the intra-uterine environment, summarized by birth weight, was not a major contributor to adult fatness. Studies of infants whose mothers smoked during pregnancy,^{118,132} or were exposed to famine¹²⁰ seem to refute this argument. Birth weight is lower in babies born to mothers who smoke,¹¹⁸ although 'catch up' is evident by the first year,¹¹⁸ and fatness at 5 y is greater than in children of non-smoking mothers.¹³² Clearly this relationship may be confounded by social class which was not accounted for in these studies. Data from the Dutch famine study¹²⁰ suggest that exposure to famine early in pregnancy leads to greater prevalence of obesity in offspring in adulthood, whereas exposure late in pregnancy reduces the risk of obesity. However, famine exposure resulted in an excess of premature deliveries, infants of very low birth weight, perinatal deaths and malformations of the nervous system, making it difficult to generalize the role of intra-uterine environment to healthy populations.

There is good evidence for a relationship between birth weight and adult fatness, as shown by generally large and reasonably long-term studies. Despite this consistency, in studies which attempted to take account of potential confounders such as maternal fatness^{57,123} the relationship between birth weight and later fatness was inconsistent. It is unclear the extent to which the observed relationship is due to a direct effect of the intra-uterine environment, or to genetic or other factors.

Maturation

Introduction

Maturity has long been noted to be associated with variation in body size; those more advanced in biological maturation tend to be taller, heavier and fatter, than average and later maturing individuals of the same chronological age.^{133,134} Improvement in environmental conditions, primarily nutrition and exposure to infectious disease, are major causes of earlier maturation. Menarche has been the most commonly

used marker of maturation, and in developed countries there has been a trend towards earlier menarche over the last hundred years or so, with age of menarche at about 15 y in 1860 in the south of the UK, falling to below 13 y by 1970.¹³⁵ At the same time, increases have occurred in height and weight. However, over the last two decades, there has been a marked slowing of the earlier maturation trend in Europe, North America and Japan, with stabilization in most better-off countries.¹³⁵ Most datasets have shown that girls from better off families reach menarche earlier than those from worse-off, and urban girls earlier than rural.¹³⁵ However, within the last 20 y, upper social classes in developed countries have been found to show a slightly later age of menarche,^{136,137} indicating that the relationship between socio-economic status (SES) and menarche might have changed. The later age of menarche in higher social groups has been attributed to the improvement in social conditions, removing the primary causes for late maturation in lower social groups, and to a lower weight for height in the higher social groups, first observed to be associated with age of menarche at least 20 y ago.¹³⁸ In women, both too little and excess body fat have been associated with disrupted reproductive function, leading to the hypothesis that there might be a minimum or threshold weight-for-height for menarche and maintenance of regular ovulatory menstrual cycles to occur.¹³⁸

Additional factors relate to age of menarche and include mother's age of menarche (positive relationship), percentage dietary energy from fat (accelerates menarche)¹³⁹ physical training (delays menarche)¹³⁹ and eating disorders (delays menarche).

The suggested relationships between maturation and body size have not been confined to girls, and in both boys and girls, skeletal maturity and peak height velocity have been used as markers of biological maturation.

In addition to the measures of maturation described above which occur during puberty, the timing of an earlier period during growth, termed the 'adiposity rebound' has been reported to be associated with fatness.¹⁸ Body mass index and skinfold measurements increase during the first year of life, subsequently decrease and then increase again at about 6 y of age. This second rise in childhood adiposity has been defined as the adiposity rebound.

Methodology

All longitudinal, observational studies identified by the search strategy, which reported a measure of time, or rate, of maturation during childhood (< 18 y), and fatness at least 1 y later, were included in this section of the review.

Results

Limitations of the data. Twenty-one papers describing results from 18 study populations investigating the

relationship between maturation and later fatness were identified (Table 5 and Appendix I, Table A8). Three papers were from the 1958 UK birth cohort,^{17,140,141} two papers were from the Zurich Growth Study,^{142,143} two papers from the same study by Rolland-Cachera *et al*,^{144,145} and two papers from the Amsterdam Growth and Health Study.^{146,147} In addition, two papers were from the Fels study and may have included some of the same individuals.^{148,149} One paper by Garn *et al* described two studies,¹⁵⁰ and another by Wellens *et al* described three cohorts.¹⁵¹ Sixteen of 18 studies spanned from childhood into adulthood. One of these 16 studies did not measure maturation as such, but reported on the pattern of velocity of change in BMI,^{142,143} which best fit into this section of the review. Follow-up measurements in two studies fell within childhood (< 18 y).^{152,153} The majority of studies were small, with sample sizes of 100–300, although three studies were larger, with over 1000 individuals.^{17,154,155} The childhood to adulthood period however, was well documented, with 16 out of 18 study populations reporting outcome measures in adulthood. Factors that might confound the relationship between maturation and fatness include parental fatness, socio-economic status (SES), earlier fatness in childhood, diet and activity.

Findings. (i) *Childhood to adulthood:* fifteen studies examined the relationship between time or rate of maturation and adult fatness, 13 of which included BMI as an outcome measure. Ten studies observed a negative relationship between maturation and adult BMI.^{17,144,146,149–151,154–157} where the earlier or more rapid the rate of maturation, the greater BMI in adulthood. Three studies found no relationship between timing of maturation and adult BMI.^{151,158} One study used skinfold measurements to define fatness¹⁵⁰ finding a negative relationship between maturation and each of several skinfolds.¹⁵⁰ Three studies included skinfold measurements in addition to BMI, reporting negative associations with some, and none with others.^{146,147,151,158} One study reported that more rapid maturation was related to greater subsequent percentage body fat¹⁴⁸ and another that earlier maturation was related to greater weight-for-height.¹⁵⁹ Where the relationship between maturation and later fatness was not statistically significant, it was negative in direction in all but one study, in which the direction of the non-significant relationship was not given.¹⁴⁹ An additional study investigated the pattern of change in velocity of BMI and fat and lean areas of the arm, noting that the peaks and troughs in velocity, especially at puberty, were greater in those with higher BMI in early adulthood.^{142,143}

(ii) *Within childhood:* one study investigated the association between age at menarche and BMI at 14 y, and observed a negative relationship,¹⁵² the other looked at gynaecologic age and change in skinfold

Table 5 Relationships between maturation in childhood and subsequent fatness

| Study | Age at outcome | Measure of maturity | Outcome measure | Relationship |
|--|----------------|---|--|---|
| Outcome in adulthood | | | | |
| Beunen <i>et al</i> ¹⁵⁸ | 30 y | PHV _{age} | BMI Triceps Subscapular Suprailiac Medial calf skinfolds | None None Negative Negative None/negative (depending on test) |
| Burke <i>et al</i> ¹⁵⁴ | 18–30 y | Age at menarche | BMI | Negative |
| Garn <i>et al</i> ^{150a} (NCP) | 20–35 y | Age at menarche | BMI | Negative |
| Garn <i>et al</i> ^{150a} (Tecumseh) | 20–35 y | Age at menarche | Triceps, subscapular, iliac, abdominal skinfolds | Negative for all skinfolds |
| Gasser ¹⁴² | 17–25 y | Pattern of velocity of change in BMI throughout childhood | BMI velocity Sum triceps + biceps, sum subscapular + suprailiac skinfolds | Cannot classify as other studies: patterning of velocity differed, refer to more detailed table (Appendix I). |
| Gasser <i>et al</i> ¹⁴³ | 17–25 y | Pattern of velocity of change in fat and lean areas of upper arm throughout childhood | Fat area of upper arm Lean area of upper arm | Cannot classify as other studies: patterning of velocity differed, refer to more detailed table (Appendix I). |
| Guo <i>et al</i> ¹⁴⁸ | 23 y | Rate of maturation | % body fat | Negative |
| Siervogel <i>et al</i> ¹⁴⁹ | 18 y | Age at max. BMI Age at max. velocity BMI Age at min. BMI | BMI | Positive None Negative |
| Miller <i>et al</i> ¹⁵⁹ | 22 y | Age at menarche | Weight-for-height | Negative |
| Power <i>et al</i> ¹⁷ | 33 y | Tanner stage | BMI | Negative/none (females/males) |
| Freeman <i>et al</i> ¹⁴⁰ | 23 y | Axillary hair (males) Age at menarche Comparison of relative height at 7 and 16 y | BMI | Negative Negative Negative |
| Stark <i>et al</i> ¹⁴¹ | 16 y | Age at menarche | Relative weight | Negative |
| Prokopec and Bellisle ¹⁵⁶ | 18 y | Adiposity rebound | BMI | Negative |
| Rolland-Cachera <i>et al</i> ¹⁴⁴ | 21 y | Adiposity rebound | BMI | Boys: negative Girls: negative/none depending on comparison |
| Rolland-Cachera <i>et al</i> ¹⁴⁵ | 16 y | Adiposity rebound | BMI | Negative |
| St. George <i>et al</i> ¹⁵⁷ | 18 y | Age at menarche | BMI | Negative |
| Sherman <i>et al</i> ¹⁵⁵ | 19–73 y | Age at menarche | BMI | Negative |
| van Lenthe <i>et al</i> ¹⁴⁶ | 27 y | Skeletal age | BMI | Skeletal age: negative boys and girls |
| | | PHV _{age} (boys only) Age at menarche (girls) | Sum skinfolds (biceps, triceps, subscapular, suprailiac) | PHV _{age} : none Age at menarche: negative (all apply to BMI and skinfolds) |
| Post and Kemper ¹⁴⁷ | 21 y | Skeletal age | Sum skinfolds (as above) | Negative (boys and girls) |
| Wellens <i>et al</i> ^{151b} | 19.2 y | Age of menarche | BMI, triceps skinfold | None |
| Wellens <i>et al</i> ^{151b} | 18.8 y | Age of menarche | BMI, triceps skinfold | None |
| Wellens <i>et al</i> ^{151b} | 18.3 y | Age of menarche | BMI, triceps skinfold | Negative |
| Outcome in childhood | | | | |
| Hediger <i>et al</i> ¹⁵³ | 15–19 y | Gynaecologic age | Δ triceps skinfold Δ subscapular skinfold | None None |
| Knishkowy <i>et al</i> ¹⁵² | 14 y | Age at menarche | BMI | Negative |

^aTwo studies reported in one publication. ^bData from three cohorts reported in one publication. Negative relationship—earlier/rapid maturation associated with greater fatness. Relationship: negative if reported as such with no statistical test mentioned, or significance at $P < 0.05$ stated; none if relationship reported as not significant. Adiposity rebound = second rise in adiposity, usually occurs at ~6 y on BMI and skinfold charts. PHV_{age} = age at peak height velocity.

measures, finding no relationship.¹⁵³ In this latter study, although the relationship was positive in direction (not significant), gynaecologic age was a predictor in a complex model which included many

variables, one of which was also a measure of maturation. Two interim papers of childhood to adulthood studies, also reported negative relationships between timing of maturation and fatness at 16 y.^{141,145}

Summary

The relationship between early or rapid maturation and greater subsequent fatness appears to be remarkably consistent, but potentially confounding factors were seldom accounted for. Social group was reported in two studies,¹⁵⁰ dietary intake in another,¹⁴⁷ and two studies included earlier fatness as a covariate.^{17,153}

Impressions

The relationship between maturation in childhood and adult fatness is consistent with early or more rapid maturers demonstrating greater fatness in later life. This relationship holds true in both males and females, and for a variety of markers of maturation: adiposity rebound, occurring at about 6 y of age, and peak height velocity, pubertal stages or age at menarche, all measured during adolescence. Few studies adjusted for possible confounding factors in their analyses. In one of the studies reported by Garn *et al* (NCP) ¹⁵⁰ the observed negative relationship persisted if the sample was restricted on the basis of education, and in the other (Tecumseh),¹⁵⁰ a negative relationship was observed in a group of subjects of similar SES. Post and Kemper¹⁴⁷ looked at differences in diet and activity between late and early maturers and observed that activity levels, energy intake and protein intake were higher between 13 and 21 y in both boys and girls who were late maturers. Whether the effect of maturation timing on later fatness is independent of diet and activity is not reported, but the potential correlations between diet, activity and maturation are demonstrated.

It is evident, from several studies,^{17,140,141,144,156,157,160} that a relationship between fatness *earlier* in childhood and maturation also exists. In general, this relationship would appear to be negative, such that greater fatness early in life is associated with earlier maturation, but may well depend on how early in childhood fatness is measured. Data from the 1958 UK birth cohort, show a negative relationship between fatness from 7 y onwards and timing of maturation.^{17,140,141} Data from the Fels¹⁶⁰ and Dunedin¹⁵⁷ studies show similar associations, although one study found the relationship at 6 y was not statistically significant.¹⁵² French data, which are more detailed in early childhood, also demonstrate this negative relationship between early fatness and timing of maturation, but only above 3–4 y of age.¹⁴⁴ Below this, the relationship seems to be less consistent, with greater¹⁴² or lower^{144,156} early fatness/birth weight in the early compared to the later maturers, or no difference.^{17,144} Although the relationship between fatness in early childhood and age of maturation is unclear, that between fatness at 5 y until puberty and age of maturation appears consistent. Since fatness tends to track, the relationship between maturation and later fatness may be largely due to the association between earlier and later fatness. One study attempted to address this,¹⁷ finding that once an adjustment was

made for BMI at 11 y, the relationship between puberty and BMI at 33 y was markedly reduced. In another study which included initial fatness in a multivariate analysis, no relationship was seen between maturation and later fatness.¹⁵³

There is good evidence for a consistent association between early maturation and greater fatness in adulthood, which suggests that the generally accepted ideal of faster and greater growth in childhood may not always be appropriate. However, there is also evidence for a relationship between greater pre-pubertal fatness and earlier maturation, which would seem to largely account for the relationship between early maturation and greater fatness. Other lifestyle factors such as diet and activity might contribute to this relationship which were hardly investigated by the studies we identified.

Physical activity

Introduction

Physical activity and obesity are linked by the energy balance equation, in which obesity is the result of accumulated excess energy, and physical activity is a component of energy expenditure. Energy expended during activity varies enormously, from close to basal metabolic rate (BMR) while at rest in bed, to more than 10-fold the BMR during vigorous exercise. This variation would tend to implicate a sedentary lifestyle in the development of obesity, but the relationships between activity or energy expenditure and fatness, both within and across populations, are not as clear as might be expected. As discussed by Ferro-Luzzi and Martino,¹⁶¹ some surveys have found increased physical activity to be associated with lower fatness, whereas others have not. They also point out that individuals in developing countries show the lowest body mass index (BMI), athletes are not very different, and those from developed countries have a BMI somewhat higher. However, although energy expenditure/BMR (essentially energy expenditure adjusted for body size) was highest in the athletes, there was no difference between subjects from developed and developing countries. This apparent contradiction might be due to engagement in very low cost activity among those in developing countries after completion of their essential work and domestic activities. Evidently, differences in energy intake will also contribute to the differences in BMI between developing and developed countries. As the relationship between diet and fatness may be confounded by energy expenditure, so that between energy expenditure and fatness may be confounded by dietary intake.

While the prevalence of obesity has been increasing in the developed world, it is believed that levels of activity have been declining. Although activity patterns have changed considerably, activity and energy

expenditure are not synonymous, and direct evidence of decreasing rates of energy expenditure are lacking. Evidence to support changing patterns of activity is available from surveys of secular changes in how much time people devote to various activities; time spent in paid work by men, and in domestic activities by women, has decreased, while leisure time has increased.¹⁶¹ Most manual jobs have become mechanized, and data from the Allied Dunbar Health and Fitness Survey (1990)¹⁶² show that only 20% of men and 10% of women are now employed in active occupations, which almost certainly differs from the situation, say, 50 y ago. The same survey reports that, of young adults aged 16–24, about a third take no regular activity (an average of three or more times per week).

Data on changing levels of activity in children is particularly poor, but further crude evidence is gained from use of transport data or by using markers of inactivity or time spent in passive activity such as watching television or reading. Trend data collected by the Department of Transport demonstrates that, in the UK between 1985 and 1992, walking and cycling by children decreased substantially.¹⁶³ During this period, the average distance walked in a year by a child aged 0–14 y declined by 20% and that cycled fell by 26%. In contrast, the average distance that children travelled in a car in a year increased by 40%. Current figures for television viewing show that boys and girls aged 4–15 y currently watch 17–18 h of television per week,¹⁶⁴ and secular trends in television viewing show that adults watch twice as much television today compared with the 1960s.

Physical activity could be considered a behaviour, but since it is of particular interest in relation to obesity and has been more widely addressed in the literature, it is discussed separately in this section.

Physical activity is a complex exposure and certain aspects of activity may be more important in prevention of obesity than others. Classically, physical activity is composed of four elements, as defined by the FITT principle, frequency, intensity, time and type. However, these elements relate more to adult fitness training, and it may be inappropriate to use them to describe children's activity, which is highly sporadic and spontaneous in nature. It might be more appropriate to quantify children's activity in terms of amount or volume, in line with the shift in belief regarding the type and amount of activity required to confer a benefit to health.¹⁶⁵ Over the last decade there has been a move away from an emphasis on exercise training, to promotion of a general increase in moderate activity, as reflected in the UK's recent Health Education Authority 'More People, More Active, More Often' campaign.

Even so, the problems of measuring physical activity, and defining the dimension under assessment remain. In epidemiological studies, for reasons of practicality and cost, activity is frequently measured by questionnaire. Questionnaires themselves vary

widely, but tend to focus on one or a few dimensions of activity and will be most effective where activity is easily recalled. Thus questionnaires may be appropriate for measuring vigorous activity which is likely to be structured, but are unlikely to give a reliable estimate of moderate activity, which may be incorporated into daily living activities. This is particularly true in children, for whom much activity may be play. Measures of inactivity such as television watching may be reasonably estimated by questionnaire, but further work is required to explain the relationships between television viewing and obesity. Television viewing may be a marker of decreased play or fidgeting activity, decreased vigorous activity and/or increased energy intake. Objective methods of measuring activity include doubly-labelled water, whole body calorimetry, movement sensors and heart rate monitoring, each with its advantages and disadvantages. Doubly-labelled water is regarded as the gold standard method for estimating total energy expenditure in free-living individuals, but is very expensive and cannot measure patterns of activity. Whole body calorimetry is an alternative gold standard method for estimating total energy expenditure, but cannot measure the individual in the free-living state and is therefore of limited use in large scale epidemiological studies. Heart rate monitoring has the advantage of being an assessment of both energy expenditure and activity patterns, and with the advent of small and unobtrusive monitors, is an acceptable method in the free-living situation. Heart rate monitoring compares well with calorimetry and doubly-labelled water. Movement sensors vary quite considerably,¹⁶⁶ but have been used successfully to obtain activity data from children.¹⁶⁷ Even if time and cost were no object, since the dimension of physical activity most important for protection against obesity is still unclear, it is difficult to specify a measurement method of choice at present.

Methodology

To be included, the study must report a measurement of physical activity or inactivity (e.g. television watching) or energy expenditure in childhood (< 18 y), and a follow-up measurement of fatness at least 1 y later.

Results

Studies identified fell clearly into two groups, those where the baseline measurement was within the first year of life, or 'pre-walking', and those where baseline measurement is after the first year, or 'post-walking'. Only in the post-walking group were outcomes measured in adulthood.

Nineteen publications from 16 studies were identified, describing relationships between activity, inactivity or energy expenditure and later fatness. Five studies (five papers and one abstract) assessed infant activity;^{47,95,168–171} one paper¹⁶⁸ and the abstract¹⁶⁹

were from the same study. Twelve studies (14 papers) measured childhood activity;^{20,44–46,167,170,172–179} three papers from the Amsterdam Growth and Health Study were identified.^{172–174} One study measured both infant and childhood activity¹⁷⁰ and is included in both groups.

1. Pre-walking activity

Limitations of the data. Five studies investigated the influence of infant activity on fatness later in childhood,^{47,95,168–171} with outcome measurements confined to the earlier years of childhood, up to 8 y (Table 6 and Appendix I, Table A9). Sample sizes were generally small, except for one very large study.⁴⁷ The measurement of activity in the latter study however, was very crude, with the mother asked to report on a questionnaire the frequency of ‘overactivity’ of the child. No definition or specific explanation of ‘overactivity’ was given. Activity and energy expenditure in infancy might be associated with behaviour and temperament (see behaviour section), relationships on which there is very little information.

Findings. A variety of outcome measures were used by the five studies. No relationships between infant activity and BMI, percentage body fat, subscapular skinfold, or sum of triceps and subscapular skinfolds, were found between 2 and 8 y. The only exception was the study of Berkowitz *et al*⁹⁵ where, although there was no association between frequency or intensity of infant activity and BMI at 6.5 y, there was a negative relationship between intensity (but not frequency) of infant activity and triceps skinfold.

Summary. There is very little evidence for an influence of infant activity or energy expenditure on later fatness. Studies however, were few, all but one were small in sample size, and reported on childhood fatness only up to 8 y.

2. Post-walking activity

Limitations of the data. Two studies looked at the influence of childhood activity on adult fatness, one in a sample of 181 individuals,^{172–174} while the other compared consistently active and inactive subjects from a larger sample ($n = 961$)²⁰ (Table 7 and Appendix I, Table A9). The remaining 10 studies reported childhood activity at 1–12 y with study duration of 2–8 y. Sample sizes were generally small, only three studies with more than 250 participants at follow-up.^{175,176,178} One of these, however, was a very large study, of just over 2000 children.¹⁷⁶ Potential confounders of the relationship between activity and fatness include parental fatness, socio-economic status (SES) and dietary factors, which were considered by a few studies. Overall, interpretation is complicated by the wide variety of methods employed to assess activity, and the different dimensions of activity under investigation.

Findings. (i) *Childhood to adulthood:* The Amsterdam Growth and Health Study^{172–174} and the Cardiovascular Risk in Young Finns Study²⁰ investigated the influence of childhood activity on adult fatness. In the former study, using two slightly different methods of analysis, lower total activity was associated with higher skinfold thicknesses at 27 and 29 y. In the latter study,²⁰ those active at each of three measurements during the study had lower subscapular skinfolds than those consistently sedentary, but no group differences in BMI were found.

(ii) *Within childhood:* Ten studies examined the effect of activity, inactivity or energy expenditure, on fatness at ages between 6 and 17 y.^{44–46,167,170,175–179}

Activity: six studies addressed various dimensions of activity; both negative and no relationships were found with later fatness.^{45,46,167,170,175,177} Results were not consistent even within studies, differing according to dimension of activity,⁴⁵ gender,¹⁷⁰ or type of analysis.¹⁶⁷

Inactivity: inactivity, as measured by time spent watching television, was investigated in three

Table 6 Relationship between pre-walking physical activity and subsequent fatness

| Study | Age at outcome | Description of activity | Outcome | Relationship |
|--|----------------|---|-------------------------------------|--|
| Berkowitz <i>et al</i> ⁹⁵ | 6.5 y | Neonatal activity; frequency and intensity | Log BMI | None (frequency/intensity) |
| | | | Triceps skinfold | None (frequency) Negative (intensity) |
| Davies <i>et al</i> ^{168a} | 2 y | Total energy expenditure | BMI | None |
| Davies <i>et al</i> ^{169a} | 6.7 y | As above | Sum triceps + subscapular skinfolds | None |
| Ku <i>et al</i> ¹⁷⁰ | 8 y | Total activity | Fat mass (adjusted for FFM) | None |
| O’Callaghan <i>et al</i> ⁴⁷ | 5 y | ‘Overactivity’ | % body fat (UWW) | None |
| Wells <i>et al</i> ¹⁷¹ | 2–3.5 y | Total energy expenditure Resting energy Expenditure | Obesity based on BMI | None |
| | | | BMI | None |
| | | | % body fat (deuterium dilution) | None |
| | | | Triceps skinfold | None |
| | | | Subscapular skinfold | None |

^aOne paper¹⁶⁸ and one abstract¹⁶⁹ from same study. FFM = fat free mass, UWW = underwater weighing.

Table 7 Relationship between post-walking physical activity and subsequent fatness

| <i>Study</i> | <i>Age at outcome</i> | <i>Description of activity</i> | <i>Outcome</i> | <i>Relationship</i> |
|--|-----------------------|--|---|--|
| Raitakari et al²⁰ | 18–24 y | Leisure time activity (consistently active vs consistently sedentary) | Subscapular skinfold | Negative |
| Twisk et al^{172a} | 29 y | Total activity—METs/week | BMI | None |
| | | | Sum skinfolds representing | Negative |
| | | | ≥ 20% body fat males, | |
| | | | ≥ 30% females | |
| Twisk et al^{173a} | 27 y | As above | Sum four skinfolds | Negative |
| van Lenthe et al^{174a} | 27 y | As above | Subscapular skinfold | None/positive (males)^b |
| | | | | Negative/none females^b |
| Beunen <i>et al</i> ¹⁷⁵ | 17 y | Non-compulsory sport (active vs inactive) | Triceps, subscapular, suprailiac, calf skinfolds (separately) | None |
| Dietz and Gortmaker ¹⁷⁶ | 12–17 y | TV watching hours/day | Obesity based on triceps skinfold | Positive ^c |
| Goran <i>et al</i> ⁴⁴ | 8.3 y | Total energy expenditure | Rate of change of fat mass adjusted for fat-free mass | Positive (total EE) |
| | | Resting energy expenditure | | None (resting EE) |
| Klesges <i>et al</i> ⁴⁵ | 6.4 y | Increase in leisure activity | Change in BMI | Negative (leisure) |
| | | Structured activity | | None (structured) |
| | | Aerobic activity | | Negative (aerobic) |
| Ku <i>et al</i> ¹⁷⁰ | 8 y | Total activity | % body fat (UWW) | None (girls) |
| | | | | Negative (boys) |
| Maffeis <i>et al</i> ⁴⁶ | 12.6 y | Extra-curricular + vigorous play, min/day | Obesity based on relative BMI | None |
| | | TV viewing min/day | | |
| Moore <i>et al</i> ¹⁶⁷ | 6.5 y | Total activity Caltrac monitor counts/hour | Increase in triceps skinfold | None/negative ^b |
| | | | Increase in subscapular skinfold | None |
| Parizkova ¹⁷⁷ | 15 y | Total activity | Increase in BMI | None |
| Robinson <i>et al</i> ¹⁷⁸ | 14.4 y | TV watching hours/day | % body lean (UWW) | Negative |
| | | | Change in BMI | None |
| | | | Change in triceps skinfold | None |
| Shapiro <i>et al</i> ¹⁷⁹ | 9 y | Total activity | Sum of four skinfolds | Negative |

Childhood to adulthood studies in bold type. MET = metabolic equivalent, EE = energy expenditure, UWW = underwater weighing. ^aThree papers from same study, various analyses reported. ^bRelationship dependent on method of analysis. ^cThis relationship is positive between *inactivity* and fatness, therefore negative between activity and fatness.

studies.^{46,176,178} The two smaller studies observed no effect of inactivity on later fatness.^{46,178} The third, much larger study, found a positive relationship between time spent watching television and fatness 6 y later in a sample of over 2000 children.¹⁷⁶ Despite marginal levels of significance, these results are made more convincing by the fact that baseline fatness and socio-economic characteristics were adjusted for. An hourly increment in television viewing per day was associated with a 2% increase in obesity prevalence in this population.

Energy expenditure: only one small study examined the influence of energy expenditure, both resting and total, on later childhood fatness.⁴⁴ Resting energy expenditure had no effect, whereas total energy expenditure was *positively* related to increase in fatness.

Summary

The evidence for an influence of childhood activity on later fatness is inconsistent, with studies roughly divided between finding either no effect or a protective effect of activity. Due to different methods of analysis and the variety of methods used to measure physical activity, it is difficult to estimate the size of effect that activity may have on the risk of developing obesity. Sample sizes were generally small, and these

studies have taken very different approaches to data analysis, making comparisons complex. Six studies included potentially confounding variables in analyses. Four studies included parental fatness as a covariate in analyses,^{45,46,167,178} two studies found that higher activity levels were associated with decreased risk of fatness after adjustment,^{45,167} and two studies did not.^{46,178} Both studies investigating the effect of television watching on fatness included SES in analyses,^{176,178} Dietz and Gortmaker found that longer duration of television viewing was associated with greater fatness,¹⁷⁶ Robinson *et al* did not.¹⁷⁸ Klesges *et al*⁴⁵ included dietary variables in their analyses, observing that higher activity was associated with lower risk of fatness, as did some analyses from the Amsterdam Growth and Health Study.¹⁷⁴ Maffeis *et al*⁴⁶ also included energy and macronutrient intake in analyses but found no influence of physical activity. Despite using a questionnaire to gauge hours of television viewing per day, which is a relatively crude method of measuring (in)activity, the largest study identified demonstrated a small, but adverse effect of inactivity on obesity.¹⁷⁶ Small effects should not be disregarded, since they may have considerable importance at the population level, or lead to larger cumulative effects over long periods of time.

Impressions

The evidence from the studies identified for the present review was inconsistent, but suggestive of a protective effect of activity against developing obesity.

Cross-sectional data from the UK and longitudinal data from The Netherlands show that during adolescence, the time that children spend on exercise decreases considerably.^{180–184} The two childhood to adulthood studies identified for the current review included this period, and both observed a protective effect of activity on fatness measured by skinfold thicknesses.^{20,172} It is possible that with the more marked decline in activity in late adolescence, active and inactive subjects are more easily identified, and relationships between activity and fatness become clearer. Some studies suggest that physical activity tends to track, and since tracking seems to become stronger with age from teenage years through early adulthood,²⁰ activity in adolescence may have implications for later life. Although not examining tracking *per se*, the only long-term prospective data in the UK relating to physical activity, indicate that an above average ability in school games was positively associated with activity at age 36.¹⁸⁵

Intervention studies suggest that, whatever its role in the genesis of obesity, increases in activity are an important component of programmes designed to treat obesity and maintain weight. Indeed, although activity may not be as effective as modification of diet for actually losing body weight, it seems to play a crucial role in weight maintenance. There are convincing data to support this in adults,¹⁸⁶ but data are lacking in children. The role of exercise in treatment of obesity in children has been addressed by five randomized controlled trials of at least 1 y in duration.^{10,11} Exercise showed beneficial effects when added to a diet programme¹⁸⁷ or dietary and behavioural management techniques,¹⁸⁸ or as part of a multi-component programme.¹⁸⁹ Aerobic or lifestyle exercise was more effective than callisthenics (control group)¹⁹⁰ in promoting weight loss and, interestingly, reinforcing advice on decreasing time spent in sedentary activity was more effective than reinforcing increased physical activities or a combination of the two.¹⁹¹ As in the studies identified for the current review, outcome measures and exercise programmes or advice varied across studies, yet even so, small but consistent benefits were observed.

A few randomized and non-randomized controlled trials of exercise in non-obese children have also shown small positive effects of exercise. In adolescents, a five day a week exercise programme prevented fat increases in the thigh over 5 weeks. In pre-school children, a 30 week programme of additional exercise 3 days a week showed a greater reduction in the overall prevalence of obesity, although prevention of BMI gain was shown only in girls.¹⁹² After 2 y in another study, children exposed to the exercise arm in

a school based trial tended to have lower levels of body fat.¹⁹³

On the whole, intervention studies in children have not so far provided more insight into the type of exercise most important for prevention of obesity. In the one study comparing types of exercise, lifestyle exercise had the greatest success in weight loss, followed by aerobic exercise. The control group performed callisthenic exercise and gained weight over the year.¹⁹⁰

Although the relationships between activity and obesity are of primary concern for this review, looking one step further to health outcomes, there are data examining the relationships between fitness, obesity and mortality, albeit in adults. Cardiorespiratory fitness has been found to be a more important predictor of all-cause mortality than BMI in a large cohort of middle-aged men.^{194,195} After adjusting for fitness, the positive relationship between BMI and mortality disappeared. Thus it would appear that if fitness is increased, even if fatness does not decrease, some of the risk associated with being fat is reduced. This suggests that levels of activity sufficient to improve cardiorespiratory fitness should be promoted regardless of whether obesity is reduced.

The observational longitudinal studies included in this review are diverse in methods of measuring physical activity and body fatness, and generally of small sample size. There is a lack of studies spanning the childhood into adulthood period. Despite these problems, the two studies with follow-up data in adulthood,^{20,172} and in particular the large study of Dietz *et al*¹⁷⁶ suggest that activity may protect against, or inactivity promote, the development of obesity. Infant activity, which is likely to be a different variable from childhood activity, was not seen to influence fatness in later childhood, although this conclusion is based on few studies.

Dietary factors

Introduction

Energy intake falls on the opposite side of the energy balance equation to physical activity and other components of energy expenditure. If the balance tips in favour of energy intake and is sustained, body fat will accumulate and obesity may develop. However, while the regulation of energy intake is considered crucial in the maintenance of body weight and adiposity, the relationships may be more complicated than is suggested by a simple model of energy balance. There is increasing discussion of the importance of dietary factors other than calorie intake, including infant feeding method, dietary composition, particularly percentage energy from fat, and energy density of the diet.

Paradoxically, National Food Survey data¹⁹⁶ indicate that average total energy intakes have fallen about 20% since 1970, while the prevalence of obesity has doubled. Yet metabolic studies suggest that energy cannot be considered a single currency, but must be recognized in terms of its constituent macronutrients: fat, carbohydrate, protein and alcohol.¹⁹⁷ Each macronutrient follows a particular metabolic pathway, and the ease with which it may be converted to body fat for storage differs. Moreover, carbohydrate and protein stores seem to be closely regulated by adjusting oxidation to intake, whereas fat is almost exclusively used or stored in response to fluctuations in energy balance.¹⁹⁸ *De novo* fat synthesis appears to be a very limited pathway for the deposition of body fat in humans¹⁹⁹ and therefore constitutes only a small proportion of body fat.²⁰⁰ Dietary fat is preferentially stored, and therefore implicated in the major proportion of body fat. If energy intake is in excess, the source of excess energy (fat or carbohydrate) may become important.¹⁹⁸ Since general physical activity levels appear to be falling, it is difficult to determine exactly what is happening to energy balance.

Arguments about the potential influence of infant feeding on later obesity have centred around whether the infant's ability to regulate its intake, and/or the mother's encouragement to feed, differs between breast-feeding and feeding of various formulas. Formula (bottle) feeding is frequently cited as a contributor to infant obesity, since formula-fed infants have been shown to be larger than those who are breast-fed.²⁰¹ Length of breast feeding and age of introduction of solid foods might also influence the infants regulation of energy intake, since it has been suggested that the introduction of solid foods leads to excess energy intake by increasing the energy density of the diet.²⁰¹

Methods

All studies identified from the results of the search strategy which investigated the influence of dietary

intake or composition measured in childhood (< 18 y) on fatness at least 1 y later, were included in this section of the review.

Results

The studies identified fell clearly into two main groups: those investigating infant feeding, and those looking at dietary intake during childhood. In each of these groups, only one study followed participants from childhood into adulthood.

Infant feeding

Limitations of the data. Seventeen papers from 13 studies looked at the relationship between feeding in infancy (first year of life), and fatness later in childhood (12 studies),^{47,73,74,94,96,97,130,202–210} or in adulthood (one study,²¹¹ Table 8 and Appendix I, Table A10). Two papers from a study by Agram *et al*,^{73,94} two papers from the DARLING study,^{203,204} and three papers from a study by Kramer *et al*^{74,96,97} were identified. The only study directly relating infant feeding to the risk of adult obesity²¹¹ was a small subset of a larger study. Of the 12 studies which examined the relationship between mode of infant feeding, duration of breast feeding or energy intake in infancy and obesity in childhood, none had outcomes measured later than age 7. Seven studies made some mention of socio-economic status (SES), five of which incorporated SES into multivariate analyses,^{73,74,211} related it to fatness at follow-up,¹³⁰ or to risk factor at baseline.²⁰² In one study SES was not included in analyses with infant feeding⁴⁷ and in another study it was unclear whether SES was incorporated into the analysis.²¹⁰

Findings. Six of 13 studies investigated breast vs formula feeding, on fatness in childhood (five

Table 8 Relationships between infant feeding and subsequent fatness

| Study | Age at outcome | Breast vs formula feeding | Duration breast feeding | Age at solid food introduction | Energy intake |
|--|----------------|--------------------------------------|----------------------------|--------------------------------|---------------|
| Agram <i>et al</i> ^{73,94a} | 6 y | | Positive/none ^a | None | None |
| Birkbeck <i>et al</i> ²⁰² | 7 y | None | | | |
| Dewey <i>et al</i> ²⁰³ /Heinig <i>et al</i> ^{204c} | 2 y | Breast/formula/none ^b | | None | |
| Dine <i>et al</i> ²⁰⁵ | 5 y | None | | | |
| Kramer <i>et al</i> ^{74,96,97} | 2 y | | Negative | Negative | |
| Marmot <i>et al</i>²¹¹ | 32 y | Breast (men) None (women) | | | |
| O'Callaghan <i>et al</i> ⁴⁷ | 5 y | | None | | |
| Poskitt ²⁰⁶ | 5 y | | None | | None |
| Sveger ²⁰⁷ | 4 y | | | | None |
| Vobecky <i>et al</i> ²⁰⁸ | 3 y | None | | | |
| Wells <i>et al</i> ²⁰⁹ | 2–3.5 y | | Positive | | None |
| Wilson <i>et al</i> ²¹⁰ | 7.2 y | None | | Negative | |
| Zive <i>et al</i> ¹³⁰ | 4 y | | None | None | |

^aRelationship dependent on outcome variable, positive for BMI, null for triceps skinfold. ^bRelationship dependent on outcome variable; weight-for-length, triceps and subscapular skinfolds, estimated % body fat greater in breast-fed children at 2 y. Biceps skinfold greater in formula-fed children at 2 y. Flank and quadriceps and sum of five skinfolds not significantly different in breast and formula fed children at 2 y. ^cMore than one paper from same study, only more recent data used. Childhood to adulthood study in bold type.

studies)^{202–205,208,210} or adulthood (one study).²¹¹ Marmot *et al* found a small and marginally significant association between breast feeding and greater fatness at 32 y in men, and a non-significant association in the same direction in women.²¹¹ Of the studies within childhood, four found no influence on fatness at 2–7 y.^{202,205,208,210} Dewey *et al* reported that, depending on the outcome variable examined, formula feeding, breast feeding or neither was associated with greater fatness at 2 y.²⁰³

Six studies looked at the effect of duration of breast feeding on later fatness, three studies found no relationship,^{47,130,206} one study suggested a negative relationship (longer duration of breast feeding associated with lower risk of fatness),⁷⁴ and one study suggested a positive relationship (longer duration of breast feeding associated with greater fatness).²⁰⁹ Depending on the outcome variable analysed, Agras *et al* reported either no relationship, or an association between longer duration of breast feeding and greater fatness at 6 y.⁹⁴

Five studies addressed the age of introduction of solid food; three studies observed no relationship with fatness at 1.5–6 y^{94,202,210} and two studies suggested that earlier introduction of solids was associated with a lower risk of fatness at 1 or 7 y.^{74,210} Three studies examined the influence of energy intake in infancy, but none found a relationship with fatness at 4–6 y.^{94,206,207}

Summary. Overall, no consistent pattern emerged of a relationship between mode of infant feeding, duration of breast feeding, age of introduction of solids, or energy intake in infancy and later risk of obesity. It should be emphasised that this conclusion is based on twelve studies measuring fatness to 7 y, with only one study following through to adulthood. Few studies

considered potential confounders, particularly the well-documented relationship between social class and breast feeding.²¹²

Childhood dietary intake

Limitations of the data. Twelve papers from eight studies investigated the influence of childhood dietary intake on later fatness (Table 9 and Appendix I, Table A10). Only one study, the Amsterdam Growth and Health Study^{172–174,213} reported data on diet in childhood and adult fatness, in a sample of 182 individuals. The remaining seven studies reported on associations between various aspects of childhood diet at 1–8 y of age, and fatness during later childhood.^{45,46,179,214–218} These differences in age of measurement of both explanatory and outcome variables complicates interpretation of the data. All studies were relatively small, some particularly so.^{216,217} The majority of studies estimated dietary intake using a dietary history method, which although it has the advantage of a longer measurement period, is generally considered a fairly crude method of measuring intake.²¹⁹ Possible confounders of the relationship between dietary intake and fatness include parental fatness, socio-economic status, and energy expenditure or physical activity, which few studies took into account.

Findings. (i) *Childhood to adulthood:* The Amsterdam Growth and Health Study^{172–174,213} was the only study we found which estimated the influence of childhood diet on adult fatness. Even within this study, the influence of macronutrient intake on fatness was inconsistent. This study included measurements of diet and outcome at six time points between age 13 and 27 y.

Energy intake: Two of the four papers addressed energy intake.^{174,213} Using data from all six time-

Table 9 Relationships between childhood dietary factors and subsequent fatness

| Study | Age at outcome | Energy intake | % energy from fat | % energy from carbohydrate | % energy from protein | Cholesterol intake |
|--|----------------|----------------------------|----------------------------|----------------------------|-------------------------|--------------------|
| Twisk <i>et al</i> ^{172a} | 29 y | | Negative/none ^b | None | None | |
| Post <i>et al</i> ^{213a} | 27 y | Negative | None | None | None | None |
| Twisk <i>et al</i> ^{173a} | 27 y | | None | None | Positive | |
| van Lenthe <i>et al</i> ^{174a} | 27 y | None (males) | None (males or females) | None (males) | None (males or females) | |
| | | Negative (females) | | Positive (females) | | |
| Deheeger <i>et al</i> ^{214c} | 8 y | None/positive ^b | | | | |
| Rolland-Cachera <i>et al</i> ^{215c} | 8 y | None | None | None | Positive | |
| Griffiths <i>et al</i> ²¹⁶ | 15 y | None (boys) | | | | |
| | | Negative (girls) | | | | |
| Klesges <i>et al</i> ⁴⁵ | 6.4 y | None | Positive | None | | |
| Maffeis <i>et al</i> ⁴⁶ | 12 y | None | None | None | None | |
| Nicklas <i>et al</i> ²¹⁷ | 7 y | | None | None | None | |
| Shapiro <i>et al</i> ¹⁷⁹ | 9 y | Negative | | | | |
| Shea <i>et al</i> ²¹⁸ | 6 y | | None | | | None |

^aFour papers from same study, various analyses reported.^{172–174,213} ^bRelationship dependent on predictor, absolute or change in value and time-point/interval considered. ^cTwo papers from same study, various analyses reported.^{214,215} Childhood to adulthood study in bold type.

points in the study (from age 13 to 27 y), a negative relationship was noted between *change* in energy intake and *change* in fatness.²¹³ When males and females were separated, this relationship was confined to females, but did not persist when other lifestyle variables including physical activity were accounted for.¹⁷⁴

Fat: all four papers investigated percentage energy from fat, carbohydrate and protein, three using different analytical methods.^{172,173,213} No relationship was observed between *change* in percentage energy from saturated or unsaturated fat,²¹³ or total fat,¹⁷⁴ and *change* in fatness over the study period, nor were those consuming a higher proportion of energy from fat more likely to be in the high risk group of fatness at age 27.¹⁷³ At the most recent follow-up, however, percentage energy from fat at 13–16 y was negatively related to fatness at 29 y.¹⁷² This relationship was not observed for other time periods, (fat intake at 13–21 y or 13–29 y), nor did it persist after adjusting for other lifestyle variables.

Carbohydrate: three papers reported no relationship between percentage energy from carbohydrate and later fatness,^{172,173} or between change in carbohydrate and change in fatness.²¹³ However, when males and females were separated, a positive relationship between carbohydrate intake and fatness was seen in females,¹⁷⁴ which did not persist after including other lifestyle variables in analysis.

Protein: three of the four papers found no relationship between percentage energy from protein and later fatness,¹⁷² or between change in protein and change in fatness,^{174,213} but the fourth paper found that those with a high percentage energy from protein in the diet or those changing to such a diet were significantly more likely to be in the high risk group of fatness at 27 y.¹⁷³

(ii) *Within childhood:* Seven studies estimated dietary intake at 1–8 y and investigated associations with fatness at 5–15 y.

Energy intake: five studies considered energy intake, two found no relationship with fatness at 8–12 y.^{45,46} One study found no relationship between absolute energy intake, but a positive relationship between change in energy intake between 4 and 6 y, and fatness at 8 y.^{214,215} One study found a negative relationship with fatness at 9 y,¹⁷⁹ and another found a negative relationship between energy intake and fatness at 15 y in girls but not boys.²¹⁶

Fat: five studies looked at percentage of energy from fat; four found no influence on fatness at 5–12 y,^{46,215,217,218} and one study found a positive association ($P=0.052$) at 6 y.⁴⁵ One study looked at cholesterol but found no relationship with fatness at 5–7 y.²¹⁸

Carbohydrate: percentage energy from carbohydrate was examined in four studies, none found a relationship with fatness at 2–12 y.^{45,46,215,217}

Protein: three studies investigated the effect of percentage energy from protein; two studies found

no influence on fatness at 4 or 7 y,²¹⁷ or 12 y,⁴⁶ and one found a positive association with fatness at 8 y.²¹⁵

Summary. The discussion is hampered by the small sample sizes, paucity of data spanning childhood to adulthood, and difficulty in measuring childhood dietary intake. Interpretation of data from studies of the relationship between diet at one point in childhood and later fatness is also complicated by the different time points at which explanatory variables were measured. Nonetheless, the studies identified here provide little evidence to suggest a relationship between any aspect of childhood diet and fatness in adulthood. However, childhood dietary intake is very difficult to measure, making it likely that relationships might be obscured. Few studies adjusted for potential confounders; three studies included parental fatness^{45,46,215} as covariates in analyses, two of which also included activity,^{45,46} the third study included SES.²¹⁵ These studies showed a positive relationship between percentage energy from protein and fatness after adjustment for parental fatness or SES,²¹⁵ a negative relationship between percentage energy from fat and fatness after adjusting for parental fatness and activity,⁴⁵ and no relationships between dietary composition and fatness after adjusting for parental fatness and activity.⁴⁶ Physical activity/fitness was also considered in some of the analyses of the Amsterdam Growth and Health Study^{172,174,213} and, after adjustment, the negative relationship between percentage energy from fat and fatness¹⁷² disappeared and the positive relationship between percentage energy from protein and fatness remained in one set of analyses,¹⁷³ but was not observed in another.¹⁷⁴

Impressions

During the past 2–3 decades there has been considerable debate about the role of infant feeding, particularly breast vs bottle feeding, in the promotion of fatness both in infancy and possibly later in life. Over the same period, there have been changes in the composition of infant formulas, and parental attitudes to feeding.²²⁰ As knowledge of breast milk composition has increased, modern infant formulas have been adapted to resemble breast milk more closely. It has been demonstrated that infants fed old-style formulas consumed significantly more energy than those on modern, adapted formulas or breast milk,²²¹ and therefore the relationships between infant feeding and fatness might have changed over time. The recent UK estimated average requirements for energy for formula fed infants,²²² are based on combined evidence from energy expenditure data, and energy intake data from infants receiving modern formulas, and are lower than earlier FAO/WHO/UNU guidelines.²²³ In addition to considerations of energy intake, formula feeding has been suggested to promote infant obesity because the mother tends to insist the child finishes the bottle, or

provides over-strength formula. Other proposed contributors to infant obesity include early weaning, or introduction of solid foods, which are generally related to each other, but might not be of equal importance in their influence on later fatness.

The evidence from longitudinal studies identified for the current review do not suggest that infant feeding method has an influence on fatness in childhood. The only study to follow through to adulthood suggested that breast-feeding might be related to adult fatness,²¹¹ but there are difficulties interpreting these data. This study is based on a subset of the 1946 UK birth cohort, and reported that men who were breast fed had a triceps skinfold of 4 mm higher, corresponding to 1.7% body fat,²²⁴ than men who were bottle fed. Sample sizes were not large, and the difference of marginal significance ($P=0.06$). The authors recognized that this relationship might be confounded by social class, but too few subjects were classified as manual to examine whether the difference existed in the lower social classes, although it remained present in those in the higher, non-manual social classes (itself a heterogeneous group). The relationships between infant feeding method and later fatness may not remain constant over time, so that the relationship might differ for childhood fatness and for adult fatness. Studies with outcome measures at over 7 y of age will be needed to determine if this is the case.

Similarly, no consistent evidence was identified for an influence of duration of breast-feeding on childhood fatness, and almost equal numbers of studies suggested either no association, or a negative relationship between the age solid foods are introduced and later childhood fatness. None of the three studies investigating infant energy intake reported an association with childhood fatness. No study has followed participants for long enough to examine the contributions of these factors to fatness in adulthood.

Although there is a general acceptance that diet plays a role in obesity development, the relative importance of various dietary factors is more controversial. Despite the biological plausibility that higher energy intakes lead to increased energy storage and body fat, many dietary surveys in Western countries have observed inverse relationships between energy intake and BMI. These studies, however, may easily be confounded by other factors, particularly energy expenditure. In the current review we found no evidence that absolute energy intake is positively related to later fatness, and only one study that suggested a change in energy intake was positively related to later fatness.²¹⁴ One of the two studies reporting a negative association between energy intake and later fatness attempted to investigate whether increased energy expenditure explained the relationship seen in all subjects.²¹³ Using fitness as a proxy marker,²¹³ the association was only partly accounted for. If, in the same study, males and females were separated¹⁷⁴ and other lifestyle variables

including physical activity entered into multivariate analyses, the relationship did not persist.

Beyond energy intake itself, the hypotheses for macronutrient composition influencing fatness have concentrated around carbohydrate and fat, the major sources of energy for the body. The levels of fat in the diet have received particular attention. Since the 1970s absolute fat consumption has decreased, but because energy intakes have also fallen, fat as a percentage of energy has decreased very little. A recent meta-analysis has suggested that at the beginning of the century, total fat intake was about 25% of dietary energy, increasing to 33% by the late 1930s and peaking at 40% in the late 1970s before starting to fall.²²⁵ The National Food Survey figures are slightly higher, showing a peak of 42.6% in 1980, falling to 39.1% in 1997.^{1,226} There is still some way to go to meet the 1991 COMA recommendations,²²² which are that population averages for total fat intake should be reduced to 35% of total food energy, and saturated fat to 10% of energy. In the 1987 OPCS Nutritional and Dietary Survey, only 12% of men and 15% of women achieved the 35% limit of fat as percentage of food energy.²²⁷ Young children however, are much closer to the recommended levels, which are considered appropriate for the whole population of children by the age of 5 y.²²⁸ Between infancy and the age of 5 y, energy derived from fat is expected to fall from 50%, as supplied by breast milk or infant formula, to 35%. Recent data collected in 1992–1993 suggest that children aged $1\frac{1}{2}$ – $4\frac{1}{2}$ y, derive, on average, about 36% of energy from fat.²²⁸ Available data for older children suggest that they may not be so close to the recommendations, but it should be noted that these data were collected in 1983. Although the average consumption of fat was about 38% of energy in 10–11 and 14–15 y olds, three-quarters of children took more than 35% of their energy as fat.²²⁹

The relationship between dietary fat and obesity has been recently reviewed.^{230,231} Ecological studies, which investigate the relationship between percentage energy from dietary fat and relative weight across populations, have produced both positive and negative associations. Most cross-sectional studies within populations have observed positive relationships between fatness in the population and fat intake,²³⁰ but prospective studies are less consistent.²³¹ However, the validity of all these epidemiological studies has been criticized. Ecological, cross-sectional and prospective studies may all be subject to serious confounding by population or individual differences in physical activity, smoking, socio-economic factors and genetic susceptibility. Data from men and women are not always separated, yet relationships may differ between sexes. Furthermore, the precision and reproducibility of methods employed to measure energy or fat intake and relative weight vary greatly. Under-reporting of energy and fat intake by obese individuals is well recognized, and limits the possibility of study-

ing the relationship in question in epidemiological studies.

Randomized controlled trials that investigated the effect of a calorically unrestricted low fat diet on weight change, generally showed a modest weight reduction in the low-fat treatment group.²³⁰ However, follow-up data suggest it is less clear whether the weight loss achieved in these relatively short-term studies could be maintained longer term. In a recent review of treatment of obesity, little success of only dietary therapy is seen, but sample sizes are very small.^{10,11} Most of the studies discussed so far, however, do not attempt to disentangle the effect of fat intake and energy intake. Because diets high in fat are energy dense and highly palatable, a diet relatively high in fat usually leads to increased energy intake. Conversely, decreasing fat content of the diet therefore usually simultaneously decreases energy intake. The role of energy density in the regulation of energy intake has been recently reviewed.¹⁹⁷ Experimental studies show that if percentage energy from fat is increased, and energy density (kJ/100 g) allowed to increase in parallel, energy intake increases. If, however, energy density is held constant, energy intake does not increase, despite the increasing fat content of the diet. This would suggest that energy density of the diet has an important role to play in regulating energy intake. In practice, with unmanipulated foods, a diet with low energy density is likely to also be low in fat.

Despite the promotion of low fat foods and avocation of low fat diets, we identified only one study of six that reported a positive relationship between percentage energy from fat in childhood and greater subsequent body fat. The appropriateness of recommending low fat diets for children and from what age remains unclear.^{14,15} Low fat diets in childhood have been associated with deficits in fat soluble vitamins and poorer growth in height in some children.¹⁵ There also remains a danger that the recommendation of low fat diets in childhood may lead to some parents putting very young children onto extremely restricted diets.

Some studies have demonstrated that fatness in the population decreases with increasing carbohydrate or sugar consumption, and that fat:sugar ratio predicts obesity better than fat alone.²³² This can be explained by the reciprocal relationship generally found between intakes of fat and simple sugars.²³³ None of the studies addressing carbohydrate intake in the present review demonstrated any influence on the development of fatness, in child- or adulthood.

The role of protein intake in the development of fatness has received little attention. In the current review, however, two of four studies observed a positive relationship between percentage energy from protein and later fatness.^{173,215} Rolland-Cachera *et al*²¹⁵ found that a higher protein intake at 2 y was associated with an earlier adiposity rebound (see Maturation section), which was associated with

increased adiposity at 8 y. The authors suggest this effect might result from protein intake leading to an increase in IGF-1, triggering early adipocyte multiplication. Participants in the Amsterdam Growth and Health Study were older, and percentage energy from protein between ages 13 and 27 was seen to predict fatness at 27 y, for which the authors do not suggest an explanation.

Overall, despite the prominent role that diet is presumed to play in promoting adiposity, the current review found no consistent evidence that infant or childhood diet is related to later fatness. This may seem surprising in view of the belief that there is likely to be at least some tracking in food preferences and eating habits in addition to any postulated physiological effects. The fact that little evidence was identified linking childhood diet and adult fatness may partly reflect the paucity of studies covering the whole period. In addition, the studies in this section were generally small, the largest number of subjects at follow-up was just over 400, increasing the possibility of missing a relatively small but important association. Of particular importance in this area is the difficulty in accurately estimating all elements of dietary intake. This measurement error leads to random misclassification of exposure status and hence tends to bias studies against finding a relationship between dietary intake and later outcome. It has been demonstrated, in a dataset of over 3000 subjects, that measurement error associated with dietary data may not have a linear effect on regression coefficients and confidence intervals, and may be marked when measurement error variance accounts for more than 10–15% of the total variance.²³⁴

Interestingly, when relatively homogeneous groups with markedly differing eating habits are compared, clear differences in fatness are also observed. Numerous studies (although not all), have observed lower relative body weights in vegetarians,^{235–237} vegan, macrobiotic²³⁸ and Seventh Day Adventist²³⁹ population groups, compared with control groups or national data. These findings have been noted in both males and females and across a large age range. Some studies reported higher intakes of fibre,^{236,240,241} or carbohydrate^{242,243} or lower intakes of fat,^{235,240,244} or energy,^{240,245} which might partly explain the differences in body fatness, but few have simultaneously investigated levels of physical activity. Other factors are also associated with vegetarian and other alternative diets, such as lower incidence of smoking, lower alcohol consumption and generally increased health consciousness. Because of the difficulty of measuring diet and adiposity, it is possible that the influence of diet on fatness only becomes apparent when comparing more distinct, homogenous groups. It is also likely that other health-related lifestyle habits associated with alternative diets, will contribute to the relationship between diet and fatness. The diet–fatness relationship may itself be weak, but become evident when acting in combination with some other

protective lifestyle factors, as observed in groups following alternative diets.

Behavioural and psychological factors

Introduction

There is a diverse body of literature linking behavioural and psychological factors to obesity, much of which concerns data from cross-sectional studies. The list of behavioural and psychological factors studied in relation to obesity or weight loss is varied, and includes, in children, self-esteem, temperament, personality traits, feeding behaviour, nutritional knowledge, and maternal awareness of offspring eating behaviour. These factors have all been proposed by one mechanism or another, to influence energy balance. Temperament, which develops from birth, may influence not only subsequent feeding and activity behaviour of the child,²⁴⁶ but also parental behaviour; for example, difficult children may be fed more often to pacify them. Moreover, they may be fed sugary or energy rich foods in these circumstances.^{246,247} Low self-esteem has long been regarded as a factor which contributes to the development of fatness, and is the central element of the psychosomatic theory of obesity,²⁴⁸ in which food is associated with comfort. Unfortunately, the evidence for this hypothesis is largely from cross-sectional and case history data, with prospective observational studies almost non-existent.²⁴⁹ The relationship between self-esteem and obesity in children and adolescents has been quite widely addressed, as summarized in a recent comprehensive review.²⁴⁹ In contrast, personality has been suggested to affect obesity by a little investigated biochemical mechanism.²⁵⁰ The way in which individuals cope with stress can be described as an 'active' reaction, involving help seeking and problem solving behaviour, or a 'passive' reaction, characterized by avoidance of stressors, also considered a defeat reaction. A defeat reaction has been hypothesized to lead to hyperactivity of the hypothalamic–pituitary–adrenal axis, causing increased cortisol secretion, which promotes fat accumulation.

Additional behavioural and psychological factors have been examined predominantly in studies of adults, although they may have some relevance to children. Greater individual self-control, the ability and propensity to engage in self-reinforcement, appears to enhance success at weight reduction. Eating behaviour patterns, in particular binge eating, are considered likely to influence the development of fatness. Binge eating disorder is defined as having a pattern of regular and sustained binge eating, but in contrast to bulimia nervosa, neither starvation or compensatory behaviours are present. In the United States, binge eating is a common and serious factor

among a subset of the overweight population,²⁵¹ and has been found to be more common in younger and heavier subjects, and significantly related to dietary restraint. Increased restraint, the ability to eat less than desired in resistance to physiological or psychological pressures to eat, has been observed to make an individual more likely to overeat in response to an emotional challenge.²⁵² Lastly, some individuals seem to have the ability to take corrective action when their weight increases above some self-imposed level, a behaviour which may be acquired or learned. Whether this cognitive threshold may be modified by training, however, or whether it is instinctive and cannot be altered, is unknown.

Methodology

No attempt was made to define behavioural or psychological factors, since predictors of fatness were not searched for specifically in the search strategy. This section is a compilation of all studies identified which included a measure of behaviour or a psychological factor during childhood (< 18 y) and a measure of fatness at least 1 y later. Physical activity and inactivity, which could be classified as behaviour, is dealt with in a separate section.

Results

Limitations of the data. Thirteen papers from 10 studies were identified investigating an aspect of behaviour or a psychological factor on the development of fatness^{47,73,74,94,96,131,209,246,253–257} (Tables 10 and 11, and Appendix I, Table A11); two papers from the study of Agras *et al.*,^{73,94} two papers from the study of Kramer *et al.*,^{74,96} and two papers from the study of Wells *et al.*^{209,246} Two studies followed participants from childhood to adulthood^{131,253} and the remainder fell within childhood, as summarized in the Table 10. Details of the behaviour or psychological factors measured are given in Table 11. Some studies tested many variables for relationships with fatness, in relatively small sample sizes, increasing the likelihood of finding significant relationships due to chance. Further problems associated with research in this field include the lack of standardized methods to measure psychological and behavioural variables, which are difficult to quantify. Use of global rather than specific measures of predictors such as self-esteem, personality or anxiety may mean that critical variables are missed. For example, general anxiety or self-esteem may not predict fatness, but that specific to body weight might.²⁵²

Findings. (i) *Childhood to adulthood:* one study spanning the childhood to adulthood period assessed personality traits,²⁵³ and the other investigated variables relating to sweet eating habits and mother's knowledge of these habits in her offspring.¹³¹ Of the

six personality traits investigated in the Amsterdam Growth and Health Study (inadequacy, social inadequacy, dominance, rigidity, achievement motivation and debilitating anxiety), only inadequacy at 13–16 y was related to increased fatness in adulthood.²⁵³ In the study of sweet eating,¹³¹ only mother's lack of knowledge had a significant influence on increasing the likelihood of their offspring being overweight in adulthood, after adjustment for childhood BMI, gender and social factors. The other variables, mother's acceptance of child's sweet eating habits, amount of sweet money, child's frequency of eating sweets, mother's acceptance of sugary food, and amount of pocket money did not significantly affect offspring's obesity after adjustment for covariates.

(ii) *Within childhood*: four studies within the childhood period were concerned with feeding behaviour, maternal attitudes to feeding behaviour, sleeplessness and temperament of the infant.^{47,73,74,246} Agras *et al*^{73,94} found that a vigorous feeding style, characterized by high-pressure sucking, more rapid sucking, longer bursts of sucking and shorter interburst interval, was related to increased adiposity up to 3 y, but not at 6 y. This vigorous feeding style was, however, also related to energy intake at the time of measurement, 2–4 weeks of age (see also dietary section). In another study,⁴⁷ infant feeding problems (undefined) had a protective effect on moderate, but not on severe obesity at 5 y. Kramer *et al*^{74,96} found

that neither infant temperament, maternal preconception of ideal body habitus or maternal feeding attitude was related to fatness at 2 y, whereas Wells *et al*²⁴⁶ found that infant distress to limitations (see Table 11 for definition), was positively related and soothability (definition, Table 11) negatively related to fatness at 2–3.5 y.

The four remaining studies concerned children rather than infants,^{254–257} and addressed the influence of temperament, self-esteem, family functioning, and attitudes towards and knowledge of nutrition, on fatness 1–5 y later. Carey *et al*²⁵⁴ found that, of nine categories of temperament, decreased adaptability, increased intensity and increased withdrawal were related to increased weight-for-height. No influence of the other six categories, activity, distractibility, mood, persistence, rhythmicity/predictability, or sensory threshold was seen. Klesges *et al*²⁵⁶ assessed four types of self-esteem in children: cognitive ability, physical ability, peer relations, general self-esteem and also family function (reported by parents). After a year, higher physical esteem and more positive family situations as viewed by the father, were related to greater increases in body fat in girls, smaller increases in boys. But these relationships became weaker with length of follow-up. At year 3, only family functioning was related to BMI (not triceps skinfold), with opposite effects in boys and girls. French *et al*²⁵⁵ found physical appearance and

Table 10 Behavioural and psychological factors investigated in relation to the development of fatness

| Study | Age at predictor and outcome measurements | Outcome measure | Risk factor |
|--|---|-----------------|--|
| (i) Childhood to adulthood | | | |
| van Lenthe <i>et al</i> ²⁵³ | 13 y (14, 15, 16, 21 y) | 27 y | Sum biceps, triceps, subscapular and suprailiac skinfolds |
| Lissau <i>et al</i> ¹³¹ | 9–10 y | 20–21 y | Obesity and overweight based on BMI |
| (ii) Within childhood | | | |
| Agras <i>et al</i> ⁹⁴ | 2 and 4 weeks | | Triceps skinfold, log BMI |
| Agras <i>et al</i> ⁷³ | 2 y | | Sucking behaviour, active feeding time, sucking pressure, interburst interval, no. feeds/day |
| | 2 and 4 weeks | | As above |
| Carey <i>et al</i> ²⁵⁴ | 3 y, 6 y | | As above |
| | 3–7 y | | Weight-for-height |
| | 8–9 y | | Nine categories of temperament. |
| French <i>et al</i> ²⁵⁵ | 12–15 y | | BMI |
| | 15–18 y | | Nine types of self-esteem |
| Klesges <i>et al</i> ²⁵⁶ | 3–6 y | | % ideal body fat based on triceps skinfold |
| | 6–9 y | | BMI |
| | | | Family Relationship Index (FRI) assessing family function |
| Kramer <i>et al</i> ^{74,96} | 1–3 days for IBH + MFA | | BMI, sum triceps + subscapular + suprailiac skinfolds |
| | | | Infant temperament (ITQ) |
| | | | Maternal preconception of ideal infant body habitus (IBH) |
| | | | Maternal feeding attitudes (MFA) |
| | 2 weeks for ITQ | | |
| | 1 y, 2 y | | |
| Morris <i>et al</i> ²⁵⁷ | 10 y | | Δ weight–height index |
| | 11 y | | Δ skinfold index |
| O'Callaghan <i>et al</i> ⁴⁷ | 6 months | | Obesity based on BMI |
| | 4–6 y | | Attitudes towards nutrition |
| | | | Knowledge of nutrition |
| | | | Feeding problems |
| | | | Sleeplessness |
| Wells <i>et al</i> ^{209,246} | 12 weeks | | Infant temperament |
| | 2–3.5 y | | Sum triceps + subscapular skinfolds, % body fat |

Table 11 Details of behavioural and psychological factors investigated in relation to the development of fatness

| <i>Study</i> | <i>Risk factor</i> | <i>Description of risk factor</i> |
|--|---|--|
| (i) Childhood to adulthood | | |
| van Lenthe <i>et al</i> ²⁵³ | Personality traits: inadequacy social inadequacy dominance rigidity achievement motivation debilitating anxiety | Vague feelings of malfunctioning, anxiety, vague physical/psychosomatic complaints, depressed mood Neurotic shyness, uncomfortable feelings in social situations, avoidance of unfamiliar people or situations Need for regularity, having fixed habits + principles, sense of duty, perseverance Need to achieve, will to reach achievements Fear of failure, leading to lower achievements, especially in unstructured tasks |
| Lissau <i>et al</i> ¹³¹ | Variables relating to sweet eating habits and mother's knowledge of such habits | Mother's acceptance of child's sweet eating habits Amount of sweet money, child's frequency of eating sweets Mother's knowledge of child's sweet eating habits Mother's acceptance of sugary food Amount of pocket money |
| (ii) Within childhood | | |
| Agras <i>et al</i> ^{73,94} | Sucking behaviour, active feeding time, sucking pressure, interburst interval, no. feeds/day | |
| Carey <i>et al</i> ²⁵⁴ | Nine categories of temperament: activity rhythmicity (younger children) or predictability (older children) approach/withdrawal adaptability intensity mood persistence/attention span distractibility sensory threshold | 'Difficulty of child' Amount of physical motion during sleep, eating, play, dressing, bathing etc. Regularity of physiologic functions such as hunger, sleep, elimination (younger children) or predictability and quality of organization e.g. doing things on time (older children) Nature of initial responses to new stimuli — people, places, foods, toys, procedures Ease or difficulty with which reactions to stimuli can be modified in a desired way Energy level of responses, regardless of quality or direction Amount of pleasant + friendly or unpleasant + unfriendly behaviour in certain instances Length of time particular activities are pursued by child with or without obstacles Effectiveness of extraneous environmental stimuli in interfering with ongoing activities Amount of stimulation, such as sounds or light, necessary to evoke discernible responses in child Harter Self-perception Profile for Adolescents |
| French <i>et al</i> ²⁵⁵ | Nine types of self-esteem: global physical appearance athletic scholastic social acceptance job competence romantic appeal behavioural conduct close friendship | |
| Klesges <i>et al</i> ²⁵⁶ | Four types of self-esteem: cognitive ability physical ability peer relations general self-esteem Family Relationship Index (FRI) assessing family function | Harter perceived Competence Scale for Children Emphasizes academic abilities Focuses on sport and outdoor games Assesses standing in peer group Focuses on self-perceptions of general all-round competence (a) cohesion — degree of helpfulness and support among family members (b) expressiveness — extent to which family members are encouraged to be open and express their feelings (c) conflict — extent to which family relations are characterized by open expression of anger and aggression and general conflictual situations Modification of questionnaire as used by Carey |
| Kramer <i>et al</i> ^{74,96} | Infant temperament (ITQ): maternal preconception of ideal infant body habitus (IBH) maternal feeding attitudes (MFA) | |
| Morris <i>et al</i> ²⁵⁷ | Attitudes towards nutrition Knowledge of nutrition | |
| O'Callaghan <i>et al</i> ⁴⁷ | Feeding problems Sleeplessness | Undefined |

Table 11 Continued

Table 11 *Continued*

| <i>Study</i> | <i>Risk factor</i> | <i>Description of risk factor</i> |
|---------------------------------------|---|---|
| Wells <i>et al</i> ^{209,246} | <p>Infant temperament: time spent fussy</p> <p>time spent crying</p> <p>frequency of distress to limitations</p> <p>frequency of soothability</p> | <p>'Irritability of child'</p> <p>Unsettled, irritable, restless, or fractious. may be vocalizing, but not continuously crying</p> <p>Periods of prolonged distressed vocalization</p> <p>Measure of a child's fussing, crying, or showing distress while (a) waiting for food, (b) refusing food, (c) being in a confining place or position, (d) being dressed or undressed, or (e) being prevented access to an object toward which the child was directing his/her attention</p> <p>Child's reduction of fussing, crying or distress when soothing techniques used by parent/caretaker of child (related to intensity, adaptability, sensory threshold and mood as used by Carey²⁵⁸)</p> |

social acceptance self-esteem were inversely related to change in BMI over 3 y in girls, but not boys. Morris *et al*²⁵⁷ found that better knowledge of nutrition or nutritious meals were protective against increasing fatness, but that there was no influence of positive attitudes to good nutrition, knowledge relating to fattening puddings, or preferences of nutritious meals or fattening puddings.

Summary. Few studies have addressed the potential influence of behavioural or psychological factors on the development of fatness, particularly in the longer term. The studies identified for the current review were very diverse in the factors they investigated, making it difficult to draw unifying themes from those identified. There is potential for considerable overlap between behaviour and psychological factors and social, dietary and physical activity factors, and the mechanisms by which behaviour might disturb energy balance appears largely untested.

Impressions

Many different constructs have been used to investigate the relationships between behavioural or psychological variables and fatness, and include among the studies presented here, personality traits, temperament, self-esteem, sweet eating habits, infant feeding behaviour, knowledge of nutrition and various maternal attitudes. Many of these were tested only in one study, providing little evidence to support or refute prospective relationships with the development of fatness. Factors such as child's knowledge of nutrition or mother's knowledge of sweet eating habits, could almost be regarded as socio-economic factors, or might at least be highly correlated with other social factors. Some studies have taken socio-economic factors into account,¹³¹ others have not.²⁵⁷

Temperament and self-esteem were investigated in more than one study, and although assessment tools were not identical, at least some common elements were identified. Kramer *et al*⁷⁴ and Wells *et al*²⁴⁶ investigated temperament in infancy, using different modifications of the questionnaire used by Carey,²⁵⁴

who looked at temperament in childhood. Wells *et al*²⁴⁶ found that greater irritability in infancy, as indicated by distress to limitations, was related to increased fatness, and less irritability, as indicated by the child being more easily soothable, was related to lower fatness. Soothability in this study is related to intensity, adaptability, sensory threshold and mood, as used in the study of Carey *et al* and described by Rothbart.²⁵⁸ The findings in the two studies broadly agree, with the study of Carey *et al*,²⁵⁴ finding that increased 'difficulty' of the child, as indicated by decreased adaptability or increased intensity, was related to greater subsequent fatness. Conversely, in a larger but shorter study, Kramer *et al*⁷⁴ found no relationships between temperament and later fatness. In addition to being less fat, Wells *et al*²⁴⁶ found that more easily soothable children were more 'awake and active' at follow-up, suggesting that infant temperament may also relate to subsequent physical activity behaviour. There was no relationship between distress to limitations and activity, but more irritable children, who were fatter, watched less television, an observation which does not easily reconcile with the greater activity of more soothable (less irritable) children. Measurement of activity however, was fairly crude. Neither soothability nor distress to limitations in infancy were related to energy intake at follow-up, as measured by dietary history. Neither Carey *et al*²⁵⁴ nor Kramer *et al*⁷⁴ investigated the possibility that temperament might also be related to energy intake or expenditure. A recent study of overfeeding in adults has reported that the 10-fold differences in fat storage were largely accounted for by changes in non-exercise activity, such as the activities of daily living, fidgeting, spontaneous muscle contraction and maintaining posture when not recumbent.²⁵⁹ Those who increased their non-exercise activity the most gained less fat, suggesting that activity such as fidgeting, which is very difficult to measure in epidemiological studies, may be an important factor in resisting weight gain. Whether non-exercise activity is related to temperament requires confirmation.

Two studies looked at relationships between self-esteem and subsequent fatness, one in children, the

other in adolescents. Each used a self-esteem scale specific to the age of the subjects, but both scales were designed by Harter, and overlapped in the areas of self-esteem assessed. In children, lower self-esteem in their physical ability was related to greater subsequent fatness, a relationship which weakened with length of follow-up. However, in adolescents, lower self-esteem in their physical appearance or social acceptance was related to greater subsequent fatness, whereas athletic (or physical ability) self-esteem did not relate to later fatness. However, this may not be surprising; if physical appearance and social acceptance is more important to adolescents than their physical or sporting ability, the self-esteem relationships might be expected to change over time. Although self-esteem and fatness has been quite widely investigated, since the vast majority of the studies are cross-sectional,²⁴⁹ these issues have yet to be addressed. Existing research into self-esteem and obesity also shows contradictory findings which deserve further attention. The review of French *et al*²⁴⁹ suggested that self-esteem was often lower in obese adolescents, yet one large study has shown that self-esteem in overweight and non-overweight 16–24 y olds was similar, and that their weight status did not predict their self-esteem 6 y later.⁸ This apparent contradiction may be due to differences in measurement methods, or because the relationships are complex, and may differ according to the weight status, social and cultural background of the population. The same review found that weight loss treatment programmes increased self-esteem, although the increase was not predictive of weight loss.²⁴⁹ Which aspects of self-esteem are related to obesity, and whether they protect against obesity or are more important for weight loss, remains unclear.

Perhaps the best current evidence that behavioural and psychological factors may be important, is provided by intervention studies which aim to treat obesity, incorporating a behavioural component. Behaviour modification, usually related to management of eating behaviour and/or physical activity, has shown success in increasing weight loss in those with mild to moderate obesity, but whether behaviour change and weight loss are maintained is questionable.²⁶⁰ Recently, data from randomized controlled trials has been reviewed.^{10,11} Studies were few, sample sizes small and interventions varied, yet some aspects of behaviour appeared to aid weight loss. In adults, cue avoidance (avoidance of situations that provide the temptation to overeat), daily weight charting, behavioural therapy by correspondence, and extension of the intervention period for behavioural therapy all showed promise as a component of a weight-loss programme. In children, there is some evidence that either treating obese parents and children together,¹⁹⁰ or involving parents without directly targeting them for weight loss, may enhance weight loss or prevent obesity progressing in children.^{261,262} The 'Shapedown' programme, a combination of cog-

nitive, behavioural, affective and interactional techniques to encourage 'small sustainable modifications in diet, exercise, relationships, lifestyle, communications and attitudes', has been successful in treating obesity in adolescents.²⁶³ The manner in which childhood behaviour is influenced may be crucial; reinforcing decreased sedentary behaviour alone has been found to be more effective for weight loss than reinforcing increased physical activity or a combination of the two.¹⁹¹

Further studies support the hypothesis that the way in which behaviour is modified matters. Both positively reinforcing children for being less sedentary or punishing them for being sedentary seems to be more effective in increasing activity than restricting access to sedentary activities.¹⁹¹ Making sedentary activities contingent on being physically active also results in decreased time spent on sedentary activities.²⁶⁴ These changes in activity patterns may contribute to useful strategies to enhance weight loss or prevent obesity. Eating behaviour may also be modified. Children have been shown to learn to dislike foods they are encouraged to eat to obtain rewards (e.g. eat your vegetables and you can watch TV) and prefer foods eaten in positive social contexts as rewards (e.g. clean up your toys and you can have some cookies).²⁶⁵ How the development of food preferences relates to later fatness remains unknown.

Finally, there are interesting findings from behavioural intervention studies where the intervention was not aimed to influence obesity itself, yet an effect on obesity was found. Children whose mothers took part in practitioner–mother interviews during the first 5 y of their child's life, were less obese at 27–29 y.²⁶⁶ The interviews aimed to enhance the mother's self-worth, foster gentle physical interaction with child, and adopt a positive attitude to modifying child's behaviour, with the intention of improving the mother–child relationship. In the Know Your Body Evaluation Project, a health education programme to reduce coronary heart disease, increased baseline self-esteem was related to greater decreases in ponderosity in those who were obese at baseline but not at follow-up.²⁶⁷

Although longitudinal, observational evidence for the contributions of behaviour and psychological factors to the development of fatness is limited, the range of data provided by other types of study suggest that further longitudinal investigation may prove useful. From existing literature, the picture appears complex, since behaviour and psychological factors clearly overlap with social factors, dietary intake and physical activity. How these factors interrelate is unclear, but it may be important if elements of behaviour or personality lead to disruptions of energy balance, and therefore predict susceptibility to obesity. Studies of behavioural modification for weight loss and treatment of obesity give clues to these interrelationships, yet because treatment strategies are frequently multifactorial, they can be difficult

to disentangle. Behavioural strategies that are successful in aiding weight loss may or may not be effective in protecting against obesity at the population level, and caution may need to be exercised in comparing the two.

Discussion

Although the links between adult obesity and adverse health outcomes are established, those between child or adolescent obesity and morbidity⁵ and mortality⁶ in adulthood are less well documented. The health consequences of obesity in youth have been recently reviewed²⁶⁸ and evidence for cross-sectional relationships between childhood obesity and factors related to morbidity in adults summarized. Increased blood lipids, glucose intolerance, hypertension, and increases in liver enzymes associated with fatty liver, have all been observed to be more common in obese children or adolescents. Since these factors tend to cluster and track with age, it seems likely that an increased risk for morbidity will carry forward into adulthood. However, as pointed out by Dietz,²⁶⁸ it is unclear whether the risk of morbidity associated with obesity varies with age of onset, severity, duration or the factors responsible for the onset. Childhood obesity is also associated with adverse social consequences,^{7,8} and as the prevalence of obesity in childhood increases the potential associated morbidity and psychosocial consequences in adulthood may be a cause for concern.

Limitations of the evidence

Few studies have addressed long term predictors of obesity, and therefore the current review identified few datasets with measurements of risk factors in childhood, and outcome measures in adulthood. Part of the problem appears to be that existing data are not fully utilized. In particular, there are several large cardiovascular disease, blood pressure and cancer studies with few or no published *longitudinal* analyses of predictors of obesity that we identified. These data could be usefully analysed (or analysed more extensively) since sample sizes are generally large, and height and weight data are usually available, so that fatness can be gauged in terms of body mass index (BMI). Obesity, hypertension, cardiovascular disease and cancer share common lifestyle factors that are related to all four conditions, and so in many cases it is likely that data for risk factors such as those considered in the current review will be available, even if they have been measured relatively crudely. The lack of available analyses from these studies is likely to be due, first to the fact that obesity was not the main interest of the study, and second to publication bias. Publication bias has been defined as a tendency towards preparation, submission and pub-

lication of research findings based on the nature and direction of the results. It has been demonstrated that positive and significant results are more likely to be published, and published more rapidly than negative, null or non-significant findings.²⁶⁹ Therefore those involved in relevant longitudinal studies, but where obesity is not the main focus or outcome measure, may be less inclined to prepare and publish non-significant findings. In addition, selective reporting of outcomes and risk factors may occur so that only those demonstrating positive or significant results are published. The result of publication bias is to increase the observed size of effect.²⁷⁰ For some of the risk factors in the current review, for example diet and activity, there were few studies and effects varied, but it might be more of a problem in studies concerning birth weight, maturation or social class, for which data are more routinely collected and therefore readily available.

Because risk factors need to be measured prospectively, longitudinal studies provide the strongest data for evidence of relationships between risk factors and development of obesity. Unfortunately, inherent problems in defining and measuring risk factors and outcomes may tend to obscure real relationships, although this problem extends to most other study designs (as well as longitudinal studies). The definition of obesity remains a matter of debate, although it is anticipated that the recent work of the WHO expert committee,²⁷¹ and the International Task Force on Obesity,²⁷² in establishing a consensus of definition of obesity in adults and children and adolescents, respectively, will lead to greater consistency of reporting measures of obesity. The appropriateness of measurement methodologies to assess lifestyle factors such as diet and physical activity, and for behavioural characteristics is equally debated. This lack of consensus was reflected in the studies identified for this review, in which many different definitions and methods of measurement were employed for both risk factors and outcome measures, complicating the synthesis and interpretation of the findings. We acknowledge that measurement standardization of some risk factors, notably physical activity and dietary intake would be very difficult to achieve, and in some cases, such as for a social group, possibly not desirable, since it will be country specific. However, it would be particularly useful if raw data were presented, and used in analyses, rather than using groups defined by arbitrary cut-offs, to facilitate comparisons across studies.

Summary of findings

Parental obesity has been consistently shown to increase the risk of fatness in offspring, although few studies have looked at this relationship over longer periods of childhood and into adulthood. The contribution of genes and inherited lifestyle factors to the parent-child fatness association remains largely

unknown, and may well vary between societies exposed to different environment influences. The relationship between low SES in childhood and increased fatness in adulthood is remarkably consistent in studies of large sample sizes. In relation to fatness in childhood, however, the association with SES is less consistent. Women who change social class (social mobility) show the prevalence of obesity of the class they join, an association which is not present in men. The influence of other social factors, family size, number of parents at home, child-care and others, have been little researched. There is good evidence from large and reasonably long-term studies for an apparently clear relationship for increased fatness with higher birth weight, but in studies which attempted to address potential confounding, the relationship was less consistent. The relationship between earlier maturation and greater subsequent fatness was investigated in predominantly smaller, but also a few large studies. Again, this relationship was very consistent, but could easily be confounded by other factors which were generally not accounted for. Studies investigating the relationships between diet or activity and fatness were generally based on small samples, and included diverse methods of risk factor measurement. There was almost no evidence for an influence of activity in infancy on later fatness, and inconsistent but suggestive evidence for a protective effect of activity in childhood on later fatness. No clear evidence for an effect of infant feeding on later fatness emerged, but there were gaps in the timing of outcome measure, with only one study with a measurement after 7 y. A few, diverse studies investigated associations between behaviour or psychological factors and fatness, but mechanisms by which energy balance might be influenced were rarely addressed.

In summary, although the associations may be subject to confounding, a number of childhood factors were predictive of later obesity; parental fatness, low SES, higher birth weight, earlier/more rapid maturation and inactivity.

Interrelationships between risk factors

There are several issues that concern the interrelationships between risk factors: confounding, effect modifiers and cumulative effects. Many of the suggested risk factors are highly correlated, or may operate as proximal and distal aetiological factors on the same causal pathways. Controlling for confounding in individual studies, and attempts to disentangle relationships when combining studies is complicated by problems in precisely defining many of these risk factors.

Among interrelationships we identified, higher socio-economic status (SES) is associated with greater birth weight, and in most studies, earlier maturation, although recent data from developed countries show an association with later maturation.^{136,137} Mothers in higher social groups are more likely to breast feed, and breast feed for longer.²¹² Those of higher SES

have been reported to eat a healthier diet, and to have an increased level of physical activity, as demonstrated by data from the 1946 and 1958 British birth cohorts.^{93,273} Data from the USA show similar patterns; the prevalence of regular, and regular and vigorous activity has been reported to be greater among Whites, and to increase with level of education.²⁷⁴ In children, SES has been reported to be unrelated to energy intake, but fat consumption to be lower and protein and carbohydrate intake higher, in children of higher social groups.²⁷⁵ The recent Health Survey for England data also demonstrates associations between higher social class and more healthy eating habits, in children but not in young adults (aged 16 to 24 y).⁶³ The proportion of children aged 2–15 y consuming fruit and vegetables more than once every day, decreased from the higher to lower social classes or income, while the proportion of children consuming sweet foods, soft drinks and crisps more than once a day, or chips at least 5 days a week, increased. There was no relationship between overall activity and social class but participation in different activity types varied with social class or income group. In younger children (aged 2–10 y), participation in sports and exercise (structured activities) decreased from higher to lower social classes or income group. A similar trend was seen in girls aged 11–15 y, but not in boys. Participation in active play or walking did not vary across social classes, but the proportion of children involved in active play or walking on 5 days or more in the previous week was higher among the manual social classes. Among girls, levels of participation in housework or gardening were higher in the manual social classes, a trend that was not seen in boys. The Health Survey for England also showed that behavioural, emotional or relationship difficulties were significantly related to SES, with the lowest proportion of children with difficulties in social class I, increasing to the highest proportion in social class V. Since in adults, women from higher social groups tend to be leaner,⁶⁹ SES may also be linked to inheritance of obesity, because it is associated with maternal fatness.

Nutrition and physical activity have both been linked to timing of maturation. Childhood malnutrition has been associated with retarded maturation, as measured by delayed bone age and age at menarche,^{276,277} whereas earlier maturation has been closely correlated with improved nutrition.¹³⁵ Menarcheal age of athletes who begin training before menarche is delayed, each year of premenarcheal training delaying menarche by about 5 months.¹³⁸ Similar relationships have been observed in cohort studies: late maturing boys and girls have been found to be more active than early maturers,¹⁴⁷ and increased sports activity to be associated with later menarche.¹³⁹ Energy and protein intakes have been suggested to be higher in late maturers after adjusting for body weight,¹⁴⁷ and energy-adjusted fat intake to be associated with earlier menarche.¹³⁹

Increased energy expenditure or overall level of physical activity is necessarily accompanied by an increase in energy intake if body weight is to be maintained, and therefore each has the potential to confound the relationship between the other and fatness. Unfortunately, both risk factors are very difficult to measure, and only three studies identified in the current review investigated (or reported analyses of) diet and activity simultaneously.^{45,46,173,174} Beyond the relationship between energy intake and expenditure, the macronutrient composition of energy intake might vary with energy expenditure/activity level. Children with a high activity level have been observed to consume more energy than those with a low activity level, but to consume a higher percentage of energy as carbohydrate and a lower percentage as fat.²⁷⁸ Increased inactivity, particularly television watching, might also be related to increased dietary intake, either during viewing, or in response to food advertising, a subject addressed in a recent review of eating behaviour in children.²⁶⁵ In the USA the majority,²⁶⁵ and in the UK approximately half²⁷⁹ the advertisements during children's television are for food products. Of these, 80% in the USA are for foods with low nutritional value, including breakfast cereals high in simple sugars and snack foods high in sugar, fat and salt.²⁶⁵ Similarly in the UK, 60% of food advertisements were for breakfast cereals, snack foods and confectionery.²⁷⁹ There are few data linking television advertising and dietary intake, but children exposed to advertisements have been reported to select more sugared foods,²⁸⁰ and in Australia, teenagers who watch television more extensively have been suggested to eat healthy foods less often and unhealthy foods more often.²⁸¹ Inactive pastimes such as watching television may also displace periods of more active play or exercise from children's lives, further reducing overall activity. Increased television viewing has been associated with decreased level of physical activity, albeit weakly.¹⁷⁸ A small study suggests that, while watching television, children's metabolic rate is lower than that during rest, which might contribute to the relationships between television watching and fatness.

Certain aspects of behaviour might also be related to physical activity and thereby fatness. Children reported by teachers to have high levels of energy in adolescence, and who were judged to be good at games and were extrovert in personality, reported taking more exercise as adults, at 36 y of age.¹⁸⁵ Behavioural and dietary factors are also likely to be related in infancy. For example, mothers may use food as a pacifying technique with less soothable or more irritable infants, and wean infants to reduce infant distress.^{209,246} Thus dietary factors could contribute to the relationships between soothability, or weaning, and later fatness.

Some relationships between risk factors and fatness seem paradoxical. For example, babies with higher birth weights, and babies born to parents of lower SES

subsequently become fatter. However, babies of higher birth weight are those from higher social groups and would therefore be expected to be leaner, rather than fatter, in later life. As previously discussed (Intra-uterine growth section), the relationship between birth weight and later fatness may itself be confounded by other factors such as maternal body size or fatness.

Perhaps one of the most important potential confounders of the relationship between early life risk factors and later obesity, is parental fatness. Parents provide genetic predispositions, but also the environment in which these predispositions are expressed, and therefore the inheritance of parental fatness is inevitably a combination of inherited genes and shared environment. The shared environment is likely to include behaviours that children have learnt from their parents. Statistical adjustment for parental fatness in analyses therefore not only takes account of parental fatness, but also parental lifestyle factors which might be associated with their child's as well as their own fatness. For example, fatter parents may have lower physical activity levels, which could in turn be related to offspring activity, which itself might be a predictor of offspring fatness. The contributions of parental genes and shared common environment are almost impossible to disentangle, but for this reason we would strongly advocate that study results should be presented both with and without adjustment for parental fatness.

Confounding is clearly a problem in attempting to determine the important risk factors for obesity. Acknowledgement of the many interrelationships between risk factors and attempts to address them in analyses, however, is important.

Factors associated with obesity might act either as confounding factors, as described above, or as effect modifiers, that is, when one particular factor modifies the effect of another. The concept of genetic susceptibility is really only useful in the context of other contributors to obesity, and gene-environment interactions have been reported in relation to obesity in adults. Data from the Finnish Twin Cohort Study have suggested that, in men, although not in women, genetic effects on change in BMI were dependent on physical activity level.²⁸² In Swedish women, those with a family history of obesity had a much higher risk of weight gain on a high fat diet.²⁸³ Since the gene pool has remained relatively constant while obesity has been increasing, it is likely that changes in the environment and lifestyle are allowing expression of genes that were previously not expressed. If, for example, an individual has genes predisposing to obesity, under extreme conditions of food limitation or enforced activity, the individual is unlikely to become obese. Today's environment in developing countries, with sophisticated transport systems, sedentary jobs and abundance of low cost, relatively high-fat foods, may be considered to highly predispose to obesity. Such factors may well modify the risk for

those children with pre-existing risks, such as having an overweight parent(s).

Finally, cumulative effects on the development of obesity are likely, both over time for specific risks or at any particular time over a range of risk factors. The relationship between each risk factor and obesity is usually weak, but small effects should not be disregarded, since they may amount to larger cumulative effects over time. So far, it remains unknown how risk factors in childhood exert their effects on adulthood. There might be a physiological effect of, for example, physical activity in childhood on fatness in adulthood, or the effect might be due to tracking behaviour, with physical activity in childhood influencing later fatness via later activity patterns. In addition, 'healthy' and 'unhealthy' lifestyle factors tend to cluster together, so that those who exercise are more likely to eat a diet closer to current recommendations and are less likely to smoke.²⁷³ Moreover, 'healthy' lifestyle factors tend to be associated with higher social groups.^{93,273} Clearly, the relative contribution of each risk factor to the development of obesity is very difficult to assess, and because the effects of individual risk factors are weak, an effect may manifest only in combination with other factors.

Population and individual level influences

Influences on obesity may occur at either or both the population level, determining the position of the distribution as a whole, and at the individual level, affecting the position of an individual within the population distribution. Because the gene pool is relatively stable, genetic make-up may have a stronger effect on an individual's position in the distribution, than on the position of the distribution itself. At least in developed countries, socio-economic conditions have changed greatly, and there has been an overall improvement in living standards, which is likely to have contributed to the upward shift in adiposity distribution of the population. Socio-economic group, however, is a relative measure that determines the individual's position, rather than the population position. Lifestyle factors such as diet and activity, are also likely to exert an influence at both population and individual levels. Overall decreases in physical activity, or increases in fat in the diet, might shift the population adiposity distribution upwards, but change in an individual's diet and exercise habits could also change their position within the distribution. The multi-level influences of diet and activity might explain why small effects of each on obesity are observed, but that larger effects are seen on comparison of groups at the extremes of a risk factor (for example, very active compared with inactive). Possible population changes in behaviour are beyond the scope of this review, but behavioural factors might well influence where an individual is located in the distribution.

From the data currently available, it is difficult to draw conclusions for simple policy implementation.

There is an added need for caution in childhood, to ensure normal processes of growth and maturation are not disrupted.

Research priorities

The major research gap identified by the current review is the lack of long-term follow-up data spanning the childhood to adulthood period. This gap could be filled to some extent, firstly by follow-up of groups on whom good quality baseline data have already been collected, and secondly, by further exploitation of existing datasets. For example, the British National Diet and Nutrition Survey of Children aged 1½–4½y²²⁸ and the Nutritional Surveillance Survey of the Diets of British Schoolchildren²²⁹ have collected extensive dietary data on over 1500 and 3000 children, respectively. In addition, the Health Survey for England data from children might prove a valuable source of baseline data.⁶³ In the current review, parental fatness, low SES, higher birth weight, earlier maturation and inactivity are shown to be associated with later fatness. Further work might examine the sensitivity and specificity of using such measures or combinations to predict risk as a basis for future intervention strategies. Furthermore, given the failure of many previous studies to account for confounding variables, further insight into the causal chains of risk for obesity could be gained. Issues that remain unresolved include identification of the mechanism by which SES in early life influences obesity in adulthood and whether the relationships between birth weight and maturation and later obesity persist after accounting for confounding factors. Evidence for relationships between dietary factors and activity and later fatness are largely confined to small datasets, and there is little information as to whether these relationships are due to a direct effect, or to tracking in dietary or activity behaviour. The inter-relationships between psychological factors and behaviours that could influence energy balance, and therefore fatness, are largely unexplored. Given that the present day Western environment may predispose to obesity, an additional neglected area of research is the identification of factors which predict the maintenance of an appropriate and healthy relative weight, which may or may not be the opposite of predictors of obesity. Acknowledging the inherent problems in measurement of risk factors and outcomes, an additional approach to addressing these issues may be to use large samples for whom more basic measures of risk factors have been collected. This might allow the influences of tracking of lifestyle behaviours and how groups particular clusters of risk factors predict obesity to be investigated.

The relationships between some of the risk factors considered in the current review and secular trend data would seem compatible, for example, higher birth weight is associated with greater fatness and both birth weight and fatness have increased over recent

years. Similarly, maturation has been occurring earlier, and earlier maturation is related to fatness. The secular changes and evidence from longitudinal studies for dietary factors and physical activity are less consistent, yet secular data may inform further studies. If for example, the population distribution of activity has shifted so that the majority is inactive and at risk of obesity, and the range of activity level is narrow, relationships between activity and obesity might be obscured. There is therefore more agreement between secular trend data and findings from population studies for risk factors which are more easily measured, and measured with more precision, but less for risk factors which are more difficult to measure. The extent to which lifestyle factors track over the lifecourse is debatable, but could be highly relevant to the development of obesity. Cumulative effects of different risk factors also deserve attention, since weak effects may amplify over time. The challenge to future research will be to reconcile current knowledge on individual and population data in order to discern the important, and modifiable risk factors, or clusters of factors, on the causal pathways in the development of obesity.

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Appendix I. Tables of included studies

Abbreviations used in tables

| | |
|-------|---|
| Δ | change in |
| ANOVA | analysis of variance |
| BMI | body mass index (weight/height ²) kg/m ² |
| CI | confidence interval, 95% unless stated otherwise |
| DZ | dizygotic |
| f | female |
| f-up | follow-up |
| hgt | height |
| m | male |
| max | maximum |
| METs | metabolic equivalents |
| min | minimum |
| mo. | month |
| MZ | monozygotic |
| OR | odds ratio |
| ref. | reference |
| RR | relative risk |
| TBW | total body water |
| vs | versus |
| wgt | weight |
| wks | weeks |
| y | year |

The number superscripted after the first author of each paper included in the following tables refers to the reference list in this review. The reference numbers given as part of the description of the papers included in the following tables refer to the reference list in those papers.

Tables A1 –A4: Genetic — inheritance of phenotype papers

| Table A1 | Genetic — longitudinal studies | | | | |
|---|--|--|--|--|--|
| <i>Author/journal/ year/ country</i> | <i>Ages at outcome, measurements/ no. of subjects</i> | <i>Design/ participants</i> | <i>Outcome measure</i> | <i>Risk factor</i> | <i>Main finding</i> |
| Childhood to adulthood Kaplowitz <i>et al.</i> ⁴¹ 1988, UK London Longitudinal Group | Every 3 mo. in 1st y; 1.5 y; annually until 20 y ⁷ , <i>n</i> = 224, ~ 50% female | Prospective longitudinal. Mothers selected in approximately random sample in west London during pregnancy | Mean of log biceps, triceps, subscapular, suprailiac skinfolds | Parental mean of log biceps, triceps, subscapular, suprailiac skinfolds. All mothers and 57 fathers measured | Mother – child correlations (<0.1 to ~0.4), greater than father – child correlations (<0.1 to ~0.25). Mother – child correlations tended to increase with time, with the exception of a decrease in late adolescence. Father – child correlations tended to remain stable or decrease over time |
| Lake <i>et al.</i> ⁴² , 1997, UK UK 1958 birth cohort | 0, 7 y, 11, 16, 23 and 33 y; Duration = 33 y; <i>n</i> ≈ 17,378; baseline, <i>n</i> = 11,407 at 33 y | Prospective longitudinal. Participants members of 1958 birth cohort, all births 3 – 9 March 1958. Data from immigrants subsequently included | BMI | Parental obesity based on BMI, self-reported when child 11 y. Parents classified as underweight, normal weight, overweight, obese. Obese: BMI > 85th percentile. | In general, child's BMI at all ages increased with increasing parental obesity. For males and females, odds ratio of being obese at 33 y increased with increasing parental fatness. OR = 8.4 for both parents obese compared to both normal weight. Tracking of obesity (correlation between BMI at 7 y and at 33 y) was stronger if both parents obese than if both normal weight. Males: both parents obese, <i>r</i> = 0.46, both normal <i>r</i> = 0.25; Females: both parents obese, <i>r</i> = 0.54, both normal <i>r</i> = 0.32 |

Table A1 Continued

Table A1 Continued Genetic — longitudinal studies

| <i>Author/journal/ year/country</i> | <i>Ages at outcome, measurements/no. of subjects</i> | <i>Design/participants</i> | <i>Outcome measure</i> | <i>Risk factor</i> | <i>Main finding</i> |
|---|--|---|---|---|--|
| Whitaker <i>et al.</i> , ⁴³ 1997, USA | 1–2 y; 3–5 y; 6–9 y; 10–14 y; 15–17 y; 21–29 y; duration = 24.5 y (mean). Target <i>n</i> = 1333, <i>n</i> = 854, 64% female | Retrospective longitudinal. Participants long-term members of Group Health Cooperative in Washington State. Included if born 1965–1970, with measured weight at ≥ 21 y, height at ≥ 18 y (males) or at ≥ 16 y (females). Data extracted from medical records. 94% non-Hispanic Whites | Obesity based on BMI expressed as Z-score. Childhood — obese: BMI ≥ 85th percentile; very obese: BMI > 95th percentile (based on NHANES I+II data). Adulthood (21–29 y) obesity: men BMI ≥ 27.8; women BMI ≥ 27.3 | Parental obesity: men BMI ≥ 27.8; women BMI ≥ 27.3 Based on single adult height and multiple weight measurements used to estimate parent BMI at mid-point of each child age interval. 794 pairs parents (some with > 1 child) measured. 747 mother's records, 699 father's records | Calculation of odds ratios showed risk of obesity at all ages significantly greater if either parent obese. OR for obese mother 2.8–3.6, for obese father 2.4–2.9. Prevalence of obesity much greater in children with ≥ 1 obese parent at all ages. Multivariate logistic regression using gener- alized estimating equa- tions, with BMI weighted for time interval and incorporating adjustment for correlations between sibling data with child- hood and parental obesity included as variables of interest: before 3 y, parental obesity, childhood obesity not a predictor; over 3 y, both parental obesity and childhood obesity predictors, > 9y parent obesity less important than child obesity. Parental obesity especially important up to 6 y |

Table A2 Genetic—cross-sectional and sibling studies

| Author/journal/year/country | Ages at outcome measurements/ no. of subjects | Participants | Outcome measure | Parent/sibling fatness measure | Main finding |
|---|---|---|--|---|--|
| Health Survey for England ⁶³ 1995–1997, UK | 2–15 y, $n = 6114$ (father–child association); $n = 8163$ (mother–child association) | Nationally representative sample selected by random sampling technique | BMI | BMI | Proportion of children in top BMI quintile increases with mother's or father's increasing BMI in boys and girls |
| Ayatollahi, ⁴⁹ 1992, Iran | 6–12 y, $n = 1207$ (642 m, 565 f) | Participants representative sample of school children in Shiraz (see refs. 7 and 8) | Obesity index: weight/height ^{2.5} | Obesity index: weight/height ¹ | Correlation: father's obesity related to child's obesity, $r = 0.15$, $P < 0.00001$. Mother's obesity related to child's obesity, $r = 0.17$, $P < 0.00001$. Father's obesity related to son's obesity, $r = 0.199$, $P < 0.001$. Mother's obesity related to daughter's obesity, $r = 0.195$, $P < 0.001$ |
| Burns <i>et al.</i> ⁵⁰ 1989, USA Muscatine Family Study | ~ 15.5 y, $n = 284$ families (four groups individuals selected, $n = 70$ random, $n = 72$ lean, $n = 70$ weight gain, $n = 72$ heavy) | Participants—see ref. 11 | Relative weight (weight/median weight for appropriate age and sex group $\times 100$) grouped: lean, random, lean gain, heavy gain, heavy | BMI | ANOVA: after adjusting for age, BMI of mothers, fathers, siblings and aunts/uncles increased with increasing fatness group of offspring (lean, random, weight gain, heavy). Similar results for skinfolds, data not shown |
| Moll <i>et al.</i> ⁵¹ 1991, USA Muscatine Study | 12–24 y, numbers approximately as above | As above | As above | As above | From abstract, % variation in BMI explained by: 35% explained by single recessive locus; 42% by polygenic loci; 23% adjusted variation NOT explained by genetic factors; spouses—12% by shared environment; siblings—10% by shared environment |
| Garn <i>et al.</i> ⁵² 1981, USA Tecumseh | $n = 11,015$, individuals in four member families | Participants—see refs. 5–7 | Triceps skinfold | Parent and sibling triceps skinfold | Probability of individual being obese rises dramatically when all three remaining family members are obese, compared to all lean |

Table A2 Continued

Table A2 Continued Genetic — cross-sectional and sibling studies

| <i>Author/journal/year/country</i> | <i>Ages at outcome measurements/no. of subjects</i> | <i>Participants</i> | <i>Outcome measure</i> | <i>Parent/sibling fatness measure</i> | <i>Main finding</i> |
|---|--|--|--|---|--|
| Goran <i>et al.</i> ⁴⁴ 1998, USA | 5.2 y, annual to 8.3 y, duration 3 y, <i>n</i> = 75 baseline, <i>n</i> = 53 at 8.3 y | Participants recruited by newspaper advertisements and word of mouth | Rate of change in fat mass adjusted for fat-free mass (fat and fat free mass measured by DXA) | Parental obesity: BMI > 85th percentile for age and sex | ANOVA; rate of change in fat mass adjusted for fat-free mass related to parental obesity, <i>P</i> < 0.001. Rate of change in fat mass adjusted for fat-free mass higher in children with two obese parents than one or no obese parents in boys and girls |
| Grinker <i>et al.</i> ⁵³ 1989, USA Ten State Study | 1–12 y | Participants—no detail given | Triceps skinfold | Obesity of mothers based on triceps skinfold: lean, medium or obese | Children of obese mothers had greater triceps skinfolds from 3 y onwards |
| Grinker <i>et al.</i> ⁵³ 1989, USA Michigan Study | 0–4 y | Participants—see ref. 6 | Weight-for-height | Obesity of mothers based on weight/height. Mothers selected for obesity or normal weight criteria | Infants of obese mothers were of higher weight/height than those of normal weight mothers at 1–18 mo. but not thereafter |
| Guillaume <i>et al.</i> ⁵⁴ 1995, Belgium | 6–13 y, 70% eligible children participated. <i>n</i> = 1028 (527 m, 501 f) | Participants selected at random from school classes. Population Wallonian origin, little admixture by immigration. Rural | BMI Triceps skinfold | Parental BMI based on reported weight and height | Children's BMI was related to parents BMI (statistical method unclear, no summary statistics given). In boys, after adjusting for age, triceps skinfold related to BMI quintile of fathers, <i>P</i> = 0.038 |
| Hashimoto <i>et al.</i> ⁵⁵ 1995, Japan | 1–6 y, <i>n</i> = 3187 (1716 m, 1471 f) | Volunteers for 'Prevention of Childhood Obesity in Niigata City' project, from municipal schools | Obesity: > 15% overweight for age, height and sex using ideal weights from Welfare Ministry of Japan, 1990 | Parental BMI — reported. Obesity: BMI > 95th percentile. BMI > 27.4 fathers; BMI > 26.0 mothers | Using Chi-squared test, incidence of obesity in children with obese fathers greater than incidence in those with non-obese fathers, 11.5% vs 6.2%, <i>P</i> < 0.01. Incidence of obesity in children with obese mothers greater than with non-obese mothers, 14.5% vs 6.2%, <i>P</i> < 0.01. If sexes separated, no significant difference in boys or girls obese vs non-obese fathers. Boys and girls with obese mothers have higher incidence obesity. Overall obesity incidence for two parents obese = 30.8%, for one parent obese = 22.7%, two parents non-obese 5.9% |

Table A2 Continued

Table A2 Continued Genetic—cross-sectional and sibling studies

| <i>Author/journal/year/country</i> | <i>Ages at outcome measurements/no. of subjects</i> | <i>Participants</i> | <i>Outcome measure</i> | <i>Parent/sibling fatness measure</i> | <i>Main finding</i> |
|---|--|---|---|--|--|
| Khoury <i>et al.</i> , ⁵⁶ 1983, USA Princeton School District Study | Two groups, < 20 y and ≥ 20 y, total <i>n</i> = 177 | Participants—random recall subset of larger study | BMI | BMI | Father—son, mother—son, father—daughter, mother—daughter BMI correlations all significant for those < 20 y not for those ≥ 20 y. Sibling correlations also significant |
| Klesges <i>et al.</i> , ⁴⁵ 1995, USA | 4.4, 5.4, 6.4 y, duration 2 y, <i>n</i> = 203 baseline, <i>n</i> = 146 at 6.4 y | Participants recruited through paediatricians, day-care centres, churches. Obese over-sampled | Δ BMI | Number of parents overweight (BMI > 75th percentile for age) | Multiple regression analysis (dependent variable = ΔBMI), including adjustment for baseline BMI, gender, age, lifestyle factors (diet and activity). After adjustment, boys with both parents overweight showed average increase in BMI of 0.67 kg/m ² compared with a decrease of 0.31 kg/m ² in boys with both parents normal weight. In girls, those with both parents normal weight or overweight, BMI decreased by 0.23 kg/m ² , but increased by 0.4 kg/m ² in those whose father was overweight |
| Maffei <i>et al.</i> , ⁵⁷ 1994, Italy | 4, 8, 10, 12 y, <i>n</i> = 1523 total, <i>n</i> = 1363 this paper (676 m, 687 f) | Retrospective. Participants a random sample from four school classes in six areas of north-east Italy. This paper excludes children with low gestational age and includes subjects for whom complete data was available | Obesity: BMI > 95th percentile for age and gender, using French tables as reference (see ref. 13) | Parental BMI from reported weight and height | Odds ratios calculated for likelihood of obesity, parental BMI categories unspecified. After adjusting for age, increasing paternal and maternal BMI increased likelihood of obesity, in boys OR = 1.14, <i>P</i> < 0.001 and OR = 1.11, <i>P</i> < 0.001, and in girls OR = 1.19, <i>P</i> < 0.001 and OR = 1.16, <i>P</i> < 0.001 respectively |

Table A2 Continued

Table A2 Continued Genetic — cross-sectional and sibling studies

| <i>Author/journal/year/country</i> | <i>Ages at outcome measurements/no. of subjects</i> | <i>Participants</i> | <i>Outcome measure</i> | <i>Parent/sibling fatness measure</i> | <i>Main finding</i> |
|---|--|--|---|--|---|
| Maffei <i>et al.</i> ⁴⁶ 1998, Italy | 8.6 y; 12.6 y; duration 4 y, <i>n</i> = 298 baseline, <i>n</i> = 112 at 12.6 y | Participants recruited through schools | BMI; obesity: relative BMI > 120% (Rolland-Cachera BMI tables as reference) | Parental BMI (unclear whether measured or reported) | Three different types of multivariate analysis, with age, gender, dietary variables, parental BMI, TV viewing time and vigorous activity included as covariates. 1. Mother's and father's BMI significantly positively related to child's relative BMI at 12 y, $\beta = 2.5$, $P < 0.0001$, $\beta = 2.1$, $P < 0.0001$ respectively. 2. In logistic regression model based on whether child increased or decreased BMI over study period, parental BMI not related to change in child BMI. 3. If change in child BMI expressed relative to average change in BMI, no relationship with parental BMI. |
| O'Callaghan <i>et al.</i> ⁴⁷ 1997, Australia MUSP (Mater University Study of Pregnancy) | Baseline 0 y; follow-up 5 y; <i>n</i> = 7357 baseline, <i>n</i> = 4062 at 5 y (2133 f, 1929 m) | Mothers (<i>n</i> = 8556) enrolled at pregnancy hospital visit (ref. 20). At birth <i>n</i> = 7357, not all of these children followed up due to insufficient funds | Obesity — severe obesity: BMI > 94th percentile; moderate obesity: BMI 85–94th percentile within cohort | Maternal pre-pregnant BMI based on reported weight, measured height. Paternal BMI based on maternal reports of height and weight | Univariate analyses: RR, 95% CIs, Chi-squared test for significance. RR for moderate and severe obesity at 5 y increases with maternal or paternal obesity. Multivariate analyses including maternal BMI, maternal education, maternal income and paternal BMI, adjusted ORs calculated with 95% CIs. OR for moderate and severe obesity at 5 y increases with maternal or paternal obesity. |
| Rice <i>et al.</i> ⁵⁸ 1995, Canada Quebec family Study | 15 y, <i>n</i> = 903, parents <i>n</i> = 727 | Families recruited via media 1978–1981 to study genetic effects on several physiological and biochemical traits. | BMI; % body fat by underwater weighing | BMI; % body fat by underwater weighing | Cross-trait correlations suggesting familial determinants significant only for BMI. |

Table A2 Continued

Table A2 Continued Genetic—cross-sectional and sibling studies

| Author/journal/year/country | Ages at outcome measurements/no. of subjects | Participants | Outcome measure | Parent/sibling fatness measure | Main finding |
|---|---|--|---|--|--|
| Rosenbaum <i>et al.</i> , ⁴⁸ 1987, USA Bogalusa Heart Study | 0 y; 1, 2, 3, 4 y; 7 y; duration 7 y, <i>n</i> = 440 baseline, <i>n</i> = 250 at 7 y, analyses on <i>n</i> = 69–180 | All live births to mothers residing in ward of Bogalusa, Louisiana from 1/1/74 to 30/6/75 eligible. Of 447, seven refused. Population mixed, Black and White | BMI; subscapular skinfold | Parental BMI; parental subscapular skinfold, both measured when child aged 2 y. 192 mothers, 99 fathers measured | Univariate regression analyses. Mother's and father's BMI positively related to child's BMI at all ages, <i>P</i> < 0.05. Regression coefficient for father–child BMI increased from 0.15 at 6 mo. to 0.29 at 7 y, and for mother–child BMI from 0.11 at 6 mo. to 0.19 at 7 y. Father–child subscapular skinfold coefficient decreased from 0.18 at 6 mo. to 0.09 at 7 y, and mother–child coefficient increased from –0.1 at 6 mo. to 0.16 at 7 y |
| Tienboon <i>et al.</i> , ⁵⁹ 1992, Australia | 14.9 y, <i>n</i> = 126 families | School children in Geelong district. All families with child born Jul–Dec 1972, at school in 1987 invited. 1215 families invited, low response rate justified adequate for within family correlations. | BMI; skinfolds: biceps, triceps, subscapular, suprailiac, thigh | Parental BMI; skinfolds (as for children); obesity: BMI > 30 | Obesity in one or two parents associated with increase of one unit BMI in adolescents (not significant). Correlations: fathers vs children, <i>r</i> = 0.21, <i>P</i> < 0.05; mothers vs children <i>r</i> = 0.04, ns |
| Vuille and Mellbin, ⁶⁰ 1979, Sweden | 10 y, <i>n</i> = 345 | Participants selected from larger original sample—see ref. 18 | Relative weight; sum triceps + subscapular skinfolds | Parental BMI calculated from reported weight and heights, at or after birth of child | Mother's BMI positively related to BMI in boys and girls, and sum skinfolds in girls (contributed at least 0.015 to <i>R</i> ² in multivariate regression model). No contribution of parent fatness to sum skinfolds in boys |
| Wada and Ueda, ⁵⁸ 1990, Japan | 14–15 y, 19–20 y, duration 5 y, baseline <i>n</i> = 522; at 19–20 y <i>n</i> = 400 (198 m, 212 f) | No information on participant recruitment | BMI | Parent BMI, based on reported weight and height, seemingly at baseline | BMI at 19–20 y significantly correlated to parental BMI in males and females, mother–child slightly higher than father–child correlations. At 19–20 y in males and females, BMI significantly higher if one or both parents obese than if neither parent obese, <i>P</i> < 0.05. Prevalence of obesity, at 19–20 y increased with number of parents obese. Multiple regression: after adjusting |

Table A2 Continued

Table A2 Continued Genetic — cross-sectional and sibling studies

| Author/journal/year/country | Ages at outcome measurements/no. of subjects | Participants | Outcome measure | Parent/sibling fatness measure | Main finding |
|---|--|---|--|---|---|
| Wada and Ueda ³⁹ (continued) | | | | | for BMI at 14–15 y, in boys, mother's and father's BMI positively related to BMI at 19–20 y, $\beta = 0.12$, $P = 0.04$, $\beta = 0.06$, $P = 0.04$ respectively. In girls, mother's and father's BMI positively related to BMI at 19–20 y, $\beta = 0.07$, $P = 0.04$, $\beta = 0.07$, $P = 0.04$, respectively |
| Weinberg et al, ⁶² 1982, USA Bogalusa Heart Study | 5–17 y, $n = 1856$ (1740 full siblings, 116 half-siblings) | Participants of Bogalusa Heart study screened during the 1973–4 and 1976–7 school years — see refs 1, 2, 13. | Triceps skinfold | Full siblings vs half siblings | Intraclass correlation coefficients and heritability analysis. Intraclass correlation coefficients for triceps skinfold greater in full siblings than half siblings. Heritability estimate not significant, shared common environment statistic significant in Whites, not Blacks. |
| Wilkinson et al, ⁶¹ 1977, UK Newcastle Study of Child Development (NSCD) | 0, 6, 12 mo., 5, 10, 12 y; at 12-y subgroup of $n = 60$ obese, $n = 60$ controls studied; $n = 8000$ in NSCD at 10 y | Cases and controls identified at 10 y from a longitudinal cohort study. Cases and controls born in one of the three years of recruitment (1961) provided more detailed information ($n = 120$) | Obese cases (wgt for hgt > 97th percentile using Scott's tables) or controls (25–75th percentile) and matched for age, sex and school | Parents triceps skinfold (of subgroup of children $n = 120$) measured when child 12 y. Obesity — mother: triceps > 20 mm; father: triceps > 10 mm | Mothers and fathers of obese children had significantly greater triceps skinfolds. 39% siblings of obese group had weight-for-height > 90th percentile, only 10% siblings in control group $\chi^2 = 23.2$, $P > 0.001$ |

Table A2 Continued

Table A3 Genetic — twin studies

| Author/journal/year/country | Ages at outcome, measurements/no. of subjects | Participants | Outcome measure | Risk factor | Main finding |
|---|--|---|--|------------------------------|--|
| Allison <i>et al.</i> , ⁶⁴ 1994, USA | 12–18 y; <i>n</i> = 238 pairs; 39 pairs MZ males, 31 pairs DZ males, 69 pairs MZ females, 51 pairs DZ females, 48 pairs DZ m/f | Participants — no information on recruitment, see ref. 24 | BMI | Heritability, MZ vs DZ | Correlation and variance-covariance matrices for Box-Cox transformed BMIs calculated. MZ correlations much higher than DZ correlations and much higher than opposite sex DZ correlations suggesting substantial heritability. Difference tends to be greater in females, suggesting greater heritability in females. No meaningful differences in heritability of BMI between Blacks and Whites. Degree to which genetic and environmental factors influence BMI varies by race but not gender. |
| Brook <i>et al.</i> , ⁶⁵ 1975, UK | 3–15 y; <i>n</i> = 222 pairs; 38 pairs MZ males, 67 pairs DZ males, 40 pairs MZ females, 44 pairs DZ females | Participants all twins registered at GOSH aged 3–15 y living in greater London. Additional volunteers by appeal after TV programme on twins | Triceps skinfold, subscapular skinfold | Heritability, MZ vs DZ twins | Correlation coefficients: triceps — females MZ > DZ, genetic factors important; males MZ ~ DZ, genetic factors not so important; subscapular — males and females MZ > DZ, genetic factors important. Heritability estimates: boys — < 10 y, environment important for triceps, genetic for subscapular; > 10 y, genetic factors important for triceps and subscapular; girls — < 10 y, environment important for triceps + subscapular; > 10 y subscapular; > 10 y genetic factors important for triceps + subscapular |

Table A4 Genetic — adoption studies

| <i>Author/journal/year/country</i> | <i>Ages at outcome measurements/no. of subjects</i> | <i>Participants</i> | <i>Outcome measure</i> | <i>Risk factor</i> | <i>Main finding</i> |
|---|---|---|--|--|---|
| Vogler <i>et al.</i> ⁶⁷ 1995, Denmark Danish adoption study | 42 y; <i>n</i> = 660 adoptees; 225 bio fathers, 330 bio mothers, 202 adopt fathers, 309 adopt mothers, 177 bio siblings, 321 of 568 maternal half sibs included, 252 of 491 paternal half sibs included | All adoptions in Denmark 1924 – 1947 in which neither parent is a biological relative of their child. Information on 3691 of 5455 adoptions | BMI based on self-reported weight and height | BMI of biological and adoptive parents when participant at school — reported either by parent or offspring | Complex statistical analysis — refer to appendix of paper. Conclude that familial resemblance in BMI is entirely due to genetic relationships. Shared family environment has no influence |
| Sorensen <i>et al.</i> ⁶⁶ 1992, Denmark Danish adoption study | 7 – 13 y, <i>n</i> = 269 | As above, subgroup selected | BMI based on weights and heights from school records | BMI of biological and adoptive parents when participant at school — reported either by parent or offspring | Correlations between adoptees and adoptive parents weaker than those with biological parents. Correlations between adoptive parents and full siblings greater than for adoptees |
| Stunkard <i>et al.</i> ⁶⁸ 1986 Danish adoption study | 42 y, <i>n</i> = 60 thin, <i>n</i> = 61 median, <i>n</i> = 61 overweight, <i>n</i> = 57 obese | As above, subgroup selected to represent range of BMI | BMI based on self-reported weight and height | BMI of biological and adoptive parents — assumed current, not explicit. Overweight: BMI > 25; obese BMI > 30 | Proportion of overweight plus obese biological mothers and fathers increased with increasing weight class of offspring, <i>P</i> = 0.001, <i>P</i> = 0.04, respectively. Proportion of overweight plus obese adoptive mothers no relationship with fatness of offspring (<i>P</i> = 0.98); proportion adoptive fathers decreased with increasing weight class of offspring, <i>P</i> = 0.04. Proportion of only obese biological and adoptive mothers and fathers increased with increasing fatness of offspring, only significant for biological mothers, <i>P</i> = 0.03 |

Tables A5 and A6: Social factors papers

Table A5 Social—longitudinal studies

| Author/journal/year/country | Ages at measurements/ no. of subjects | Design/participants | Outcome measure | Risk factor | Main finding |
|---|--|--|--|--|---|
| (i) Childhood to adulthood Arnesen and Forsdahl, ⁷⁵ 1985, Norway Tromso Heart Study | 20–54 y; n = 14,652 (7405 m, 7247 f) | Retrospective. Population not selected, municipal birth cohort (refer to ref. 3), 69% of those invited | BMI | Economic conditions in family during childhood, reported retrospectively. Four groups: very difficult, difficult, good, very good. | Using equality of means, linear trend and difference between extreme groups, no relationship found between poverty in childhood and BMI in adulthood in men or women |
| Blane et al., ⁷⁶ 1996, UK (Scotland) | 35–64 y, duration 30– 60 y; n = 5645 all men | Retrospective. Participants from 27 work places selected to represent all levels and types of employment. Men < 35 y excluded | BMI | Father's main occupation, social mobility | Using multiple regression analyses, adjusting for age and own social class, BMI significantly related to father's social class, $\beta = 0.2$, $P < 0.0001$. Those in lower social class higher BMI. No relationship between social mobility and BMI |
| Braddon et al., ⁷⁷ 1986 1946 UK birth cohort | 0, 7, 11, 14, 20, 26, 36 y; duration 36 y; n = 5362 baseline, n = 3249 at 36 y | Prospective. Participants in birth cohort, all births in 1 week, March 1946. Stratified sample only included in this paper. Pregnant women excluded | Overweight—men: BMI = 25.0–29.9; women: BMI = 24.3–29.1; obesity— men: BMI > 29.9; women: BMI > 29.1 | SES based on 'social class and education' of parents. Four groups: upper and lower non-manual and manual. Social mobility: change between SES in childhood (as above) and SES in adulthood (unspecified) | At 36 y both men and women from non-manual class homes were significantly more likely to have normal or underweight BMI (men $\chi^2 = 30$, $P < 0.001$, women $\chi^2 = 52$, $P < 0.001$) than others, and those from manual homes more likely to be overweight or obese. In multiple regression models—men: after adjusting for relative weight at 11 y, SES in childhood and education were significantly related to BMI at 36 y as in above analyses, $P < 0.05$; women: after adjusting for relative weight at 11 y, parity and marital status, SES in childhood and education were significantly related to BMI at 36 y as in above analyses, $P < 0.05$. Social mobility: women who moved up from manual to non- manual SES showed significantly lower prevalence of obesity than those who remained in manual SES, 4.7% vs 11.2%. No effect in men |

Table A5 Continued

Table A5 Continued Social—longitudinal studies

| Author/journal/year/country | Ages at measurements/ no. of subjects | Design/participants | Outcome measure | Risk factor | Main finding |
|--|--|---|--|--|---|
| Hardy <i>et al.</i> , ⁷⁸ 1998, UK 1946 UK birth cohort | 0, 7, 11, 14, 20, 26, 36, 43 y; duration 43 y; <i>n</i> = 3262 baseline, <i>n</i> = 3249 at 43 y | Prospective. Participants in birth cohort, all births in 1 week, March 1946 | Obesity: BMI \geq 30 kg/m ² | Childhood social class | Using generalized estimating equations, children from manual social class have greater risk of obesity at 43 y than those from non-manual, OR = 1.56 (1.12, 2.18) No correlation between ordinal position of child and overweight. Prevalence of overweight was 18% in social class 1, 17% in social class 2, 20% in social class 3, and 31% in classes 4 and 5 |
| Charney <i>et al.</i> , ⁷⁹ 1976, USA | 20–30 y; duration 20– 30 y, <i>n</i> = 366 | Retrospective. Participants born 1945–1955, birth weight > 2270 g, with height and weight recorded several times in childhood. 90% of those contacted participated | Overweight: > 10% above median weight for height and age. Obese: > 20% weight for height and age (National Health Survey 1960–1962 used as reference data) | Social class based on education and occupation. Ordinal position of child in family | |
| Garn <i>et al.</i> , ⁸⁰ 1981, USA Tecumseh Community Health Study | 8 y, 26 y; duration 18 y, <i>n</i> = 1117 (553 m, 564 f); drop-out nos not given, similar for high and low income groups | Prospective. Subjects were those measured in rounds 1 and 4 of Tecumseh Community Health Study | Subscapular skinfold, Δ subscapular skinfold | Income per capita seems to be measured at baseline | At 26 y (in Garn's paper table 1 and text disagree about length of study), low income group had greater skinfold than medium and high income groups in boys and girls. In girls, medium group was also greater than high income group. No mention of statistical test. Change in skinfold thickness was greatest in low income group and lowest in high income group using data from all subjects, significant using <i>t</i> -tests and test for trend (level not specified) |

Table A5 Continued

Table A5 Continued Social—longitudinal studies

| <i>Author/journal/year/country</i> | <i>Ages at measurements/ no. of subjects</i> | <i>Design/participants</i> | <i>Outcome measure</i> | <i>Risk factor</i> | <i>Main finding</i> |
|---|--|--|---|--|--|
| Garn <i>et al.</i> , ⁸⁰ 1991, USA Tecumseh Community Health Study | 0–34 y, 5–39 y, duration 5 y; <i>n</i> = 8648 at 5–39 y, drop-out < 5% | Prospective. Participants of Tecumseh Community Health Survey | Obesity based on summed triceps and subscapular skinfolds normalised Z- scores. Obesity = Z-score ≥ 1 | Normalized Z-score of years of parental education /likely to be reported at baseline although not explicitly stated | Parents of children who became obese by 5–9 y were slightly above average educational level, Z-score = 0.11, not significantly different from zero, but significantly different from other groups. Parents of children who became obese by 10–14 y were slightly below average educational level, Z = -0.15, not significantly different from zero. Parents of those who became obese by 15–19 y and 20–39 y were of lower than average education level, Z = -0.21 and Z = -0.16, respectively, significantly different from zero |
| Goldblatt <i>et al.</i> , ⁸¹ 1965, USA Midtown Manhattan Study | 20–59 y; <i>n</i> = 1,660 (690 m, 970 f) | Unclear whether prospective or retrospective—see refs. 1–33. Participants—see refs. 1–3 | Obesity based on self- reported height and weight | SES based on father's occupation and education when participant 8 y. Social mobility: change in SES between 8 y and adulthood based on occupation, education, income, rent | SES of origin inversely related to obesity in women $\chi^2 = 66.5, P < 0.001$ and in men although relationship much weaker, statistics not given. More obesity in downward socially mobile than socially static, and more obesity in socially static than in upward socially mobile; in women $\chi^2 = 20.5, P < 0.001$, in men not significant |

Table A5 Continued

Table A5 Continued Social—longitudinal studies

| <i>Author/journal/year/country</i> | <i>Ages at measurements/ no. of subjects</i> | <i>Design/participants</i> | <i>Outcome measure</i> | <i>Risk factor</i> | <i>Main finding</i> |
|--|--|---|--|--|--|
| Lasker and Mascie-Taylor, ⁹⁰ 1989, UK 1958 UK birth cohort (National Child development study, NCDS) | 0, 7, 11 y; 16 y; duration between each follow-up used in analyses, f- up <i>n</i> = 18,559 | Prospective. Participants in birth cohort, 98% births 3–9 March 1958. Data from ethnic minorities excluded for this paper | BMI, Δ BMI | SES based on father's occupation, reported at baseline and follow-ups. Social mobility: change in SES between follow-ups | No influence of SES (assume at age 7 y) on change in BMI 7–11 y. Significant effect of SES. Those in lowest SES group had greatest increase in BMI at 11–16 y. Δ SES (social mobility) 0 (assumed) —7 y not related to BMI at 7 y. Δ SES 7–11 y not related to BMI at 11 y. Δ SES 11–16 y not related to BMI at 16 y. No mention of statistical tests or significance levels |
| Power and Moynihan, ⁹¹ 1988, UK 1958 UK birth cohort | 7 y; 23 y; duration 16 y; <i>n</i> = 12,274 (6133 m, 6141 f) | Prospective. Participants in birth cohort, 98% births 3–9 March 1958 | Standard deviation score (SDS) for BMI from reported height and weight at 23 y | Father's occupation, reported at baseline | At age 23, significantly greater percentage of men and women from manual classes had standard deviation score 0.5–1.5 and > 1.5 compared with non- manual classes, 22 vs 16% and 8.2 vs 4.6% for men, 18 vs 14% and 9.2 vs 4.2% for women. Between age 7 and 23, more from manual classes maintained SDS of > 1.5 compared to non- manual classes, 31 vs 18% for men, 39 vs 22% for women, <i>P</i> < 0.01. More from manual classes increased SDS to 0.5–1.5 or to > 1.5 |

Table A5 Continued

Table A5 Continued Social—longitudinal studies

| <i>Author/journal/year/country</i> | <i>Ages at measurements/ no. of subjects</i> | <i>Design/participants</i> | <i>Outcome measure</i> | <i>Risk factor</i> | <i>Main finding</i> |
|---|---|---|--|--|---|
| Power et al, ⁹² 1997, UK 1958 UK birth cohort | 0, 7, 11, 16, 23 y; 33 y; duration 33 y; <i>n</i> = 17,414 baseline, <i>n</i> = 8,459 at 33 y | As above | Overweight and obese based on reported BMI. Overweight— men: BMI = 25.0–30.0; women: BMI = 23.8–28.6. Obesity—men: BMI > 30.0; women: BMI > 28.6 | Father's occupation reported at baseline. Four groups | Results presented as odds ratios, representing proportional increase in odds from top to bottom of social hierarchy. In men and women, OR of being overweight or obese at 23 y or 33 y significantly greater for lowest compared to highest social group, <i>P</i> < 0.05. At 33 y, OR for overweight was 1.60 in men, 1.54 in women and for obesity, 2.19 in men and 1.99 in women. Social group differences in overweight and obesity lessened between 23 and 33 y but not significantly |
| Power and Matthews, ⁹³ 1997, UK 1958 UK birth cohort | 0, 7, 11, 16, 23 y; 33 y; duration 33 y; <i>n</i> = 17,414 baseline, <i>n</i> = 12,264 at 7 y; <i>n</i> = 10,121 at 33 y | As above | BMI (measured at 7 y, reported at 33 y) | Father's occupation reported at baseline. Four groups | In both males and females, no difference in BMI across social groups at 7 y but significant increasing BMI with lower social class at 33 y, <i>P</i> < 0.01, Mantel Haenzel χ^2 test for trend |
| Lissau-Lund-Sorensen and Sorensen, ⁸² 1992, Denmark | 9–10 y, 20–21 y, duration 10 y; <i>n</i> = 1258 baseline, <i>n</i> = 522 at 20–21 y | Prospective. Participants a random sample, 25% of 3rd graders in Copenhagen | Obesity: BMI > 95th percentile; overweight: BMI > 90th percentile from reported height and weight, males and females separately | Maternal and paternal education, occupation of householder, childhood rearing areas (good vs poor), reported at baseline | Bivariate regression; those with mother of low education more likely to be overweight than those with mother of middle level education, OR = 2.4, <i>P</i> < 0.05. Those reared in poor area more likely to be overweight than those in good area, OR = 2.2, <i>P</i> < 0.001. No relationships for father's education or householder occupation. After controlling for BMI in childhood and gender, only effect of rearing area remained (OR = 2.4, <i>P</i> < 0.001). Results similar if those already overweight in childhood excluded |

Table A5 Continued

Table A5 Continued Social—longitudinal studies

| <i>Author/journal/year/country</i> | <i>Ages at measurements/ no. of subjects</i> | <i>Design/participants</i> | <i>Outcome measure</i> | <i>Risk factor</i> | <i>Main finding</i> |
|---|---|----------------------------|------------------------|--|--|
| Lissau and Sorensen, ⁸³ 1993, Denmark | 9–10 y, 20–21 y, duration 10 y; <i>n</i> = 1258 baseline, <i>n</i> = 756 at 20–21 y | As above | As above | Scholarly difficulties, learning difficulties, scholastic proficiency, special education, reported by form teacher at baseline | Using logistic regression analysis and adjusting for childhood BMI, gender and confounder score to account for parents education, householder's occupation and residential area, obesity more likely if learning difficulties present in childhood, OR = 4.2, <i>P</i> = 0.0003, scholastic proficiency below average, OR = 2.8, <i>P</i> = 0.006, received special education, OR = 2.7, <i>P</i> = 0.007, or had scholarly difficulties in childhood, OR = 1.5, <i>P</i> = 0.006. Odds ratios for being overweight, values 1.5–2.0 and less significant. Results similar if separate adjustment made for social factors rather than using confounder score. If children already overweight or obese in childhood excluded, results similar but less significant |
| Lissau and Sorensen, ⁸⁴ 1994, Denmark | 9–10 y, 20–21 y, duration 10 y; <i>n</i> = 1258 baseline, <i>n</i> = 756 at 20–21 y | As above | As above | Family factors, no. of siblings, parental support, child's hygiene, at baseline | Using logistic regression analysis and adjusting for childhood BMI, age, gender and confounder score (as above), family structure had no significant effect on adult obesity. Children receiving no parental support had higher risk of adult obesity, OR = 7.1, <i>P</i> < 0.0001 than those with harmonius support. Those receiving overprotective support had non-significantly increased risk of obesity, OR = 2.3, <i>P</i> = 0.2. Dirty/neglected children had much greater risk of obesity than averagely groomed children, OR = 9.8, <i>P</i> < 0.0001. Risks for overweight as for obesity |

Table A5 Continued

Table A5 Continued Social — longitudinal studies

| <i>Author/journal/year/country</i> | <i>Ages at measurements/ no. of subjects</i> | <i>Design/participants</i> | <i>Outcome measure</i> | <i>Risk factor</i> | <i>Main finding</i> |
|---|--|---|---|---|--|
| Rasmussen and Johansson, ⁸⁵ 1998, Sweden | 18.2 y, n = 75,638 | Retrospective. Single male births identified from birth registry 1973–1976. Linked to follow-up data from military service registry. 78% of identified births were term births, and height and weight data was available at 18 y. Analyses relevant to this review completed on 1975–1976 births only, 36% of identified births | Overweight: 25 ≤ BMI < 30. Severe overweight: BMI ≥ 30 | Mother's age, mother's education, mother's parity, mother's marital status, living area | Multiple logistic regression analyses, odds ratios and 95% CIs calculated. Independent variables included: birth weight, mother's age, mother's education, mother's parity, mother's marital status, living area. Compared with living in a large city, likelihood of being overweight significantly greater living in a medium-sized town, country area or rural environment, <i>P</i> < 0.05. Compared with mothers aged 25–30 y, mothers < 20 y more likely to have overweight (OR = 1.20, CI 1.10–1.32) offspring at 18 y. Compared with mothers of high education level, mothers of low education level more likely to have offspring overweight at 18 y, OR = 1.72, CI 1.62–1.82. Likelihood of obesity increased with ≥ 4 children in family compared with two children, OR = 1.22, CI 1.10–1.35, and increased if only one parent in family compared with two, OR = 1.11, CI 1.06–1.17. Results for severe overweight were similar |

Table A5 Continued

Table A5 Continued Social—longitudinal studies

| <i>Author/journal/year/country</i> | <i>Ages at measurements/ no. of subjects</i> | <i>Design/participants</i> | <i>Outcome measure</i> | <i>Risk factor</i> | <i>Main finding</i> |
|--|--|--|---|--|--|
| Ravelli and Belmont, ⁸⁶ 1979, The Netherlands | 19 y, $n = 283,028$, all male | Retrospective. Dutch men examined for military induction, born between 1/1/44 and 31/12/47 | Obesity: $\geq 120\%$ weight-for-height. WHO data used as reference | Father's occupation (manual or non-manual), birth order, family size | Overall prevalence of obesity higher in manual classes (2.09%) than non-manual (1.54%). Obesity declined with family size, from 3.2% in only children to 1.5% in children who were 1 of 5, $\chi^2 = 218$, $P < 0.0005$ for trend. Prevalence of obesity in single children significantly more than children who were 1 of 2, 3, 4 or 5, $\chi^2 = 221$, $P < 0.0005$. Only in manual social group, in families of four children, obesity among first-born significantly less than among last-born. Prevalence of obesity not consistently related to birth order |
| Teasdale <i>et al.</i> , ⁸⁷ 1990, Denmark Copenhagen adoptions | 3 mo. (median), 42 y, duration ~ 42 y, $n = 2015$ (1155 m, 860 f) | Prospective. Copenhagen register of non-familial adoptions, total $n = 5455$ | Log BMI from reported weight and height | Biological and adoptive father's occupation at time of adoption. Geographical region of adoption | Pearson correlations: log BMI negatively related to biological father's social class ($r = -0.05$, $P < 0.05$), adoptive father's social class ($r = -0.09$, $P < 0.05$). Log BMI to geographical region $r = 0.08$, $P < 0.05$, those adopted into provincial region had higher BMI than those into urban. Multiple regression: log BMI = age + sex + geographical region + bio father's SES + adoptive father's SES + bio father's SES*age, all SES variables significant. log BMI = age + sex + geographical region + bio father's SES + adoptive father's SES + bio father's SES*age + adoptive's SES, all SES variables significant. In both models, relationship between biological father's SES and log BMI is inverse and increases with age |

Table A5 Continued

Table A5 Continued Social — longitudinal studies

| <i>Author/journal/year/country</i> | <i>Ages at measurements/ no. of subjects</i> | <i>Design/participants</i> | <i>Outcome measure</i> | <i>Risk factor</i> | <i>Main finding</i> |
|---|---|---|---|--|---|
| Wannamethee <i>et al.</i> , ⁸⁸ 1996, UK British Regional Heart Study | 53–72 y, <i>n</i> = 5643 | Retrospective. Men selected from age – sex registers of general practices, one for each of 24 towns in the UK. 6528 of original 7735 men alive and contacted, 5934 replied, 5643 (73% of original cohort) with complete information | Obesity undefined in paper but cut off used was — obesity: BMI \geq 80th percentile of original 7735 men aged 40–59 y, equivalent to \geq 28 kg/m ² (personal communication) | SES based on father's longest held occupation, manual or non-manual | <i>t</i> -Test: % obese in group of non-manual father vs manual father. Greater prevalence of obesity in manual father group (20.1%) vs non-manual father group (14.1%), <i>P</i> < 0.0001. After adjustment for age and own adult social class, 19.7% obese in manual father group, 14.5% in non-manual group, <i>P</i> < 0.0001 |
| (ii) Within childhood Agras <i>et al.</i> , ⁷³ 1990, USA | 2, 4 wks, 12 mo.; 20 mo.; 2 y; 3 y; 6 y; <i>n</i> = 99 baseline, <i>n</i> = 54 at 6 y | Prospective. Children recruited from hospital in first week of life | Triceps skinfold, log BMI | Age, marital status, education and occupation of parents | Univariate correlations: at 6 y, education of parents negatively related to log BMI, <i>r</i> = – 0.31, <i>P</i> < 0.05. Multivariate regression: after adjustment for BMI at birth, several dietary factors and parent BMI: at 6 y, parent education significantly negatively related to log BMI, partial <i>R</i> ² = 0.13, <i>P</i> = 0.02. No association between parent education and child BMI at any age < 6 y or child triceps skinfold at any age. No analyses for other risk factors |
| Agras <i>et al.</i> , ⁹⁴ 1987, USA | 2, 4 wks, 12 mo.; 20 mo.; 2 y; duration 2 y; <i>n</i> = 99 baseline, <i>n</i> = 79 at 2 y | As above | Triceps skinfold, log BMI | Parent education | Multivariate regression: after adjustment for triceps skinfold at 2 wks, several dietary factors and parent triceps: parent education negatively related to triceps skinfold at 1 y, <i>P</i> < 0.02. No influence at 2 y or on BMI at 1 or 2 y |
| Berkowitz <i>et al.</i> , ⁹⁵ 1985, USA | 1–3 days, 6.5 y; duration ~6.5 y; <i>n</i> = 112 baseline, <i>n</i> = 52 (25m, 27f) at 6.5 y | Prospective. Recruitment details not given, see ref. 9 | Log BMI. Triceps skinfold | SES based on father's occupation and education as reported by mother | No significant correlation between SES and log BMI or triceps skinfold at 6.5 y |

Table A5 Continued

Table A5 Continued Social—longitudinal studies

| <i>Author/journal/year/country</i> | <i>Ages at measurements/ no. of subjects</i> | <i>Design/participants</i> | <i>Outcome measure</i> | <i>Risk factor</i> | <i>Main finding</i> |
|--|---|--|--|--|--|
| Kramer <i>et al.</i> , ⁷⁴ 1986, Canada | Baseline 1–3 days, further measurements at 2, 4, 6 wks, 2, 3, 4 mo. Follow-up at 1, 2 y. Duration 2 y, <i>n</i> = 462 baseline, <i>n</i> = 347 at 2 y | Prospective. Recruitment from large university teaching hospital serving socially diverse population. <i>n</i> = 553 eligible, 462 enrolled | BMI, sum triceps + subscapular + suprailliac skinfolds | SES based on mother's education and head of household's occupation | Multiple regression models, stepwise procedure, constructed for BMI and skinfolds at 1 and 2 y. Independent variables included: sex, birth weight, parental relative weights, social class, and behaviour and dietary variables. No influence of social class on BMI or skinfolds at 1 or 2 y |
| Kramer <i>et al.</i> , ⁹⁶ 1985, Canada | As above | As above | As above | As above | As above |
| Kramer <i>et al.</i> , ⁹⁷ 1985, Canada | As above | As above | As above | As above | As above |
| Lindgren, ⁹⁸ 1976, Sweden SLU Project | 9 y; semi-annually until 17 y f, 18 y m; duration 8–9 y; <i>n</i> = 380 baseline, <i>n</i> = 294 at 17–18 y | Prospective. Participants a sample of randomly selected children (see Ljung, 1974) | Weight-for-height | Father's occupation family income, combination of the above two | Using ANOVA, in girls, those from manual workers tended to have higher weight-for- height than higher social groups at all ages, only significant at age 11–15 y. Similar relationships in boys but weaker and not significant at any age. Comparing extreme groups (father's occupation and family income combined) greater weight-for-height in boys of lower social group not significant, but significant in girls at ages 11, 13 and 15, <i>P</i> < 0.05. No relationship between no. of parents or mothers working out of home/in home and weight- for-height at any age |

Table A5 Continued

Table A5 Continued Social—longitudinal studies

| Author/journal/year/country | Ages at measurements/ no. of subjects | Design/participants | Outcome measure | Risk factor | Main finding |
|---|--|---|---|--|---|
| Seidman <i>et al.</i> ⁹⁹ 1991, Israel. Jerusalem Perinatal Study | Baseline 0 y, f-up 17 y, duration 17 y; baseline $n = ?$; f-up $n = 33,413$ | Prospective. All births from three major wards in Jerusalem 1964–1971. At 17 y those not in defence forces not included (61.7% females missing) | Overweight: BMI \geq 90th percentile for 17 y olds in defence forces, 24.6 for m + f. Severe overweight: BMI \geq 97th percentile, 27.8 for m, 27.7 for f | Paternal education (years of schooling) seems to be assessed in childhood/at birth. Municipal tax level (area of residence) | Chi square test. Males: % severe overweight/less in lowest paternal education level, $P < 0.01$. Females: % overweight and severe overweight greater in lowest paternal education level, $P < 0.001$, $P < 0.01$ respectively. % overweight /less in highest tax level. Multiple regression also used but not clear whether paternal education, included in model, was significant covariate |
| de Spiegelaere <i>et al.</i> , ¹⁰⁰ 1998, Belgium | 12.3 y 14.8 y, duration 2.4 y, $n = 2607$ | Retrospective. Participants from six school medical centres in Brussels region. Medical files chosen in filing order to obtain ~500 files from each of five social groups | Obesity based on BMI. Moderate obesity: BMI 120– 140% for age and sex; severe obesity: BMI $>$ 140% for age and sex (Rolland-Cachera's data used as reference) | SES based on parent's profession and activity status (in or out of work). Unclear when measured, presumably from medical records | Chi square test: in girls, at 12 and 15 y significant inverse relationship between SES and obesity prevalence (P value not quoted). In boys, no relationship. Using all data (girls and boys), obesity prevalence significantly decreased, – 4%, in intermediate social group and significantly increased, 3.3% in lowest social group. RR (95% CI) of obesity in lowest vs highest SES group at 12 y in girls 1.8 (1.3–2.6) and in boys 1.0 (0.7–1.5), and at 15 y in girls 2.1 (1.5–3.1) and in boys 1.4 (1.0–2.0). RR (95% CI) of severe obesity in lowest vs highest SES group at 12 y in girls 5.1 (2.0–13.1) and in boys 1.6 (0.7–3.9) at 15 y in girls 6.7 (2.4–18.7) and in boys 1.6 (0.7–3.4). Change in obesity: greater percentage of children from low SES became obese (8.6%) than from inter- mediate (3.5%) or high SES (4.5%), χ^2 test, $P < 0.01$. RR (95% CI) of becoming obese, lowest vs highest SES group: girls 2.1 (0.9–4.6); boys 1.8 (0.9–3.8); all 1.9 (1.1–3.3); RR of becoming normal BMI, lowest SES group vs highest group: girls 0.5 (0.3– 1.0); boys 0.4 (0.2–1.1); all 0.5 (0.3–0.9) |

Table A5 Continued

Table A5 Continued Social—longitudinal studies

| Author/journal/year/country | Ages at measurements/ no. of subjects | Design/participants | Outcome measure | Risk factor | Main finding |
|---|---|--|--|--|--|
| Weststrate <i>et al.</i> , ¹⁰¹ 1986, The Netherlands | 1–5 y, 6–10 y, duration 5.5 y, <i>n</i> = 171 (104 m, 67 f) | Prospective. Participants— no information on recruitment—see ref. 23 | Change in tertile of sum triceps, biceps, subscapular and suprailiac skinfolds | SES based on mean educational level of both parents, three levels, reported at baseline and f-up (author communication), unclear which used, little difference | Using χ^2 test, significantly more children of parents of lowest education level increased fatness (tertile of skinfold thickness) than children of parents of highest education level, 19/45 vs 13/70, $\chi^2 = 7.62$ |
| Wilkinson <i>et al.</i> , ⁶¹ 1977, UK Newcastle Study of Child Development (NSCD) | 0, 6, 12 mo., 5, 10, 12 y, duration 12 y, <i>n</i> = 8000 in NSCD at 10 y, <i>n</i> = 262 (161 obese, 161 controls). Subgroup of <i>n</i> = 60 obese, <i>n</i> = 60 controls | Cases and controls identified at 10 y from a longitudinal cohort study. Cases and controls born in one of the three years of recruitment (1961) provided more detailed information | Obese cases (wgt for hgt > 97th percentile using Scott's tables) or controls (25–75th percentile) and matched for age, sex and school | All children: social class (at birth?); subgroup <i>n</i> = 120; age of mother at birth, care of child and state of home reported by health visitors at 0–3 y | A greater percentage of obese children (<i>n</i> = 161) came from social classes IV and V than expected compared with NCDS population (<i>n</i> ~ 8000), $\chi^2 = 11.4$, <i>P</i> < 0.001. In subgroup (60 obese, 60 controls), more obese children had mother over 35 y at birth (χ^2 not given) compared with controls. Care of children and state of their homes during the first 3 y of life similar in obese compared with control children |
| Zack <i>et al.</i> , ¹⁰² 1979, USA Data source: NHANES II and III | 6–11 y, 12–17 y; duration 2–5 y, <i>n</i> = 2177 | Prospective. Participants from US Health Examination Surveys, cycle II (NHANES II) and cycle III (NHANES III), probability samples of non- institutionalized population | Sum triceps and subscapular skinfolds | Family income at baseline | Using multiple regression, family income added slightly, but significantly, to the prediction of subsequent body fatness in white males (<i>n</i> = 339), in the presence of skinfolds and age at baseline. Direction of association not given. No influence in other sex/race groups |

Table A6 Social — cross-sectional studies

| <i>Author/journal/year/country</i> | <i>Ages at measurements/ no. of subjects</i> | <i>Design/participants</i> | <i>Outcome measure</i> | <i>Risk factor</i> | <i>Main finding</i> |
|--|--|---|---|--|---|
| Boulton <i>et al.</i> , ¹⁰³ 1995, Australia Adelaide Children WHO | 8.6 Y, $n=856$ (448 m, 408 f); mothers $n=838$, fathers $n=640$ | Participants from cohort of 2000 children born in particular hospital, 1975– 1976 | Sum biceps, triceps, subscapular and supra-iliac skinfolds (SSF). BMI | Mother's education, father's education, father's occupation | Using ANOVA, both mother's and father's education negatively related to child's BMI and SSF, $P < 0.0001$, $P < 0.01$ respectively. Father's occupation negatively related to child's BMI and SSF, $P < 0.01$, $P < 0.0001$ respectively |
| Gortmaker <i>et al.</i> , ⁸ 1993, USA NLSY (National Longitudinal Survey Of Labour Market Experience, Youth cohort) | 16–24 Y; $n=10,039$ (4901 m, 5138 f) | National probability sample which oversampled Blacks, Hispanics and poor non- Hispanic Whites | Overweight based on BMI from reported height and weight. Overweight: BMI > 95th percentile for age and sex from NHANES I data | Mother's education, father's education. Household income | In women, overweight associated with lower maternal and paternal education level, and household income, $P < 0.01$ (t -test? unclear). In men, no association |
| Greenlund <i>et al.</i> , ¹⁰⁹ 1996, USA CARDIA | 18–30 Y; $n=4006$ | Participants randomly recruited. Sampling designed to include equal numbers by age, sex, ethnicity, education level | BMI | Father's education, mother's education | Using ANCOVA, adjusting for father and mother's body size and participant's baseline age and education, BMI decreased with increasing father's education level in Black men, $P=0.03$, and White women, $P=0.0001$, not in Black women and White men, $P > 0.05$ |
| Hunter <i>et al.</i> , ¹⁰⁴ 1979, USA Bogalusa Heart Study | 5–14 Y, $n=2837$ with parental data, of total $n=3524$ | Participants 93% of all children 5–14 y living in Bogalusa, Louisiana during 1973–1974 school year, i.e. $n=3524$ | Weight – height index: weight/height ^{2.77} ; triceps skinfold | Head of household's education, seven categories ($n=2837$) | Age- and sex- adjusted means compared across parental education categories for Black and Whites separately. Linear and quadratic models fitted. Blacks: quadratic model significant, indicating children of parents with highest and lowest education levels had highest skinfolds, $P < 0.01$. Whites: no difference in skinfolds over education categories. Similar results for weight – height index |

Table A6 Continued

Table A6 Continued Social—cross-sectional studies

| Author/journal/year/country | Ages at measurements/ no. of subjects | Design/participants | Outcome measure | Risk factor | Main finding |
|--|--|---|--|---|---|
| Khoury et al, ¹⁰⁷ 1981, USA | 6–19 y; n = 893 (459 m, 434 f) | Participants a random 15% sample from a study of six communities, biracial | BMI. Triceps skinfold | Education of head of household. Occupation of head of household | Head of household's education negatively correlated with BMI $r = -0.17$, $P < 0.001$ and triceps skinfold, $r = -0.12$, $P < 0.05$ in girls and with BMI, $r = -0.13$, $P < 0.01$ but not triceps in boys. Head of household's occupation negatively correlated with BMI, $r = -0.12$, $P < 0.05$, but not skinfold in boys and no relationships in girls. None of these relationships significant after adjusting for age and race |
| Kimm et al, ¹⁰⁸ 1996, USA NGHS of NHLBI | 10 y; n = 2357 girls (1158 White, 1199 Black) | Schools in two states selected for approximately equal proportions of Black and White children and least racial disparity in income. Girls only, sampled to obtain even distribution in SES. 74– 81% eligible girls enrolled | Obesity: BMI > 75th percentile (20.5 kg/m ²) from combined data all subjects | Parental education. Total household income. No. parents at home | Using Bartholomew's test for monotonic trend: White girls—prevalence of obesity lower at higher household income, $P < 0.001$ for trend; prevalence of obesity lower at higher parental education, $P < 0.001$ for trend; black girls—no relationship between prevalence of obesity and household income, parental education or no. parents. Univariate regression analyses gave same results and white girls with two parents significantly less likely to be obese, $P < 0.001$. Multiple regression: White girls— lower education and one parent family significant predictors, income not significant. Black girls— neither education, income or no. parents significant |

Table A6 Continued

Table A6 Continued Social—cross-sectional studies

| <i>Author/journal/year/country</i> | <i>Ages at measurements/ no. of subjects</i> | <i>Design/participants</i> | <i>Outcome measure</i> | <i>Risk factor</i> | <i>Main finding</i> |
|--|--|---|--|---|--|
| Patterson <i>et al.</i> , ¹⁰⁶ 1997, USA NGHS of NHLBI | 9–10 y, <i>n</i> = 2357 girls (1158 White, 1199 Black) | As above | Obesity: BMI \geq 85th percentile, age and sex- specific data from NHANES II. BMI \geq 19.6 at 9 y; BMI \geq 20.9 at 10 y | Household income. Max parent education. Parent employment status over prior 12 mo. No. parents/ guardians. Age female guardian. No. siblings | Using ANOVA, White girls from household with lower income significantly more likely to be obese. Black girls no relationship. Whites with parents of lower education more likely to be obese, Blacks no relationship. Whites with neither parent employed more likely to be obese than those with one or two parents employed, Blacks no relationship. Whites with one parent more likely to be obese than those with two parents, Blacks no relationship. Both Black and White girls were more likely to be obese if mother older, less likely to be obese with more siblings. Multiple logistic regression: mother's age and no. of siblings significant predictors for both Black and White girls, max parent education and employment significant for Whites only |
| Persson, ¹⁰⁸ 1984, Sweden | 4, 8 or 13 y, <i>n</i> = 738, 935 invited, 901 eligible | Random selection of children from three specified geographical areas | Overweight: 110–119.9% of standard obesity: \geq 120% of standard weight-for-height, from Swedish reference values, Karlberg, 1976 | SES based on mother's education | Using logistic multiple regression, after adjusting for age group, sex and geographical area, SES was not a significant predictor for overweight but was for obesity. The shorter the mother's education the more likely the child to be obese |

Table A7 Intrauterine growth papers

| <i>Author /journal/year/country</i> | <i>Ages at outcome measurements/ no. of subjects</i> | <i>Design /participants</i> | <i>Outcome measure</i> | <i>Birth weight^a</i> | <i>Main finding</i> |
|---|---|---|---|---------------------------------|--|
| (i) Childhood to adulthood Allison <i>et al.</i> , ¹²² 1995, USA Minnesota Twin Registry | 28–52 y (mean 40 y), <i>n</i> = 4020 twin pairs; 699 pairs MZ males, 609 pairs DZ males, 939 pairs MZ females, 880 pairs DZ females, 893 pairs DZ male/female | Retrospective. Members of Minnesota twin Registry (refs. 22, 23). One pair twins excluded due to intra-pair difference in birth weight > 1.9 kg | BMI | Birth weight | Correlations: Birth weight positively associated with adult BMI, <i>r</i> = 0.078, <i>P</i> < 0.0005 (similar results whether all individuals included or 1 member of each pair). Intra-pair difference in birth weight not associated with intra-pair difference in adult BMI, <i>r</i> = 0.026, <i>P</i> = 0.331. Multivariate analyses—ordinary least squares regression: with dependent variable intra-pair difference in adult weight, independent variable of interest intra-pair difference in birth weight and covariates intra-pair difference in adult height, age, sex, mean birth weight; semi-partial <i>r</i> ² for difference in birth weight and difference in adult weight = 0.03, <i>P</i> = 0.034, thus birth weight has no influence on adult relative weight |
| Braddon <i>et al.</i> , ⁷⁷ 1986, UK 1946 UK birth cohort | 0, 7, 11, 14, 20, 26 y; 36 y; duration 36 y; <i>n</i> = 5362 baseline; <i>n</i> = 3249 at 36 y | Prospective. Participants in birth cohort, all births in 1 week, March 1946. Stratified sample only included in this paper. Pregnant women excluded | Overweight—men: BMI = 25.0–29.9; women: BMI = 24.3–29.1. Obesity—men: BMI > 29.9; women: BMI > 29.1 | Birth weight | Up to 14 y, prevalence of overweight (relative weight 111–130%) and obesity (relative weight > 130%) was greater in boys and girls of greater birth weight. At age 36, overweight or obese men had significantly higher birth weights, <i>F</i> = 12.29, <i>P</i> < 0.01. No difference at 36 y in women |

Table A7 Continued

Table A7 Continued Intrauterine growth papers

| Author/journal/year/country | Ages at outcome measurements, /no. of subjects | Design/participants | Outcome measure | Birth weight ^a | Main finding |
|--|--|--|--|---|--|
| Charney et al, ¹⁷⁹ 1976, USA | 20–30 y; duration 20–30 y; n=366 | Retrospective. Participants born 1945–1955, birth weight > 2270 g, with height and weight recorded several times in childhood. 90% of those contacted participated | Overweight: > 10% above median weight for height and age. Obese: > 20% weight for height and age (National Health Survey 1960–1962 used as reference data) | Birth weight unclear whether from records or reported | Higher birth weight associated with greater prevalence of overweight in adulthood. For those with birth weight > 75th percentile, 32% were overweight as adults, for those with birth weight ≤ 75th percentile, 20% were overweight as adults. Statistical test not stated, P=0.04 |
| Curhan et al, ¹²³ 1996, USA Nurses Health Studies (NHS) I+II | NHS I, 30–55 y, n = 52,824 in this paper NHS II, 25–42 y, n = 84,839 in this paper BMI | Retrospective. NHS I: 121,700 female registered nurses in 1976. 96% White. NHS II: 116,686 female registered nurses in 1989. 94% White | BMI | Birth weight reported by participants | Logistic regression analysis, OR and 95% CI calculated. After adjusting for age, OR of being in highest quintile vs lowest quintile of BMI was increased for women with birth weight greater than reference group (7.1–8.5 lb). For birth weight 8.6–10.0 lb, OR = 1.2, CI 1.1–1.3 in NHS I and OR = 1.3, CI 1.2–1.4 in NHS II. For birth weight > 10.0 lb, OR = 1.6, CI 1.4–1.9 in NHS I and OR = 2.0, CI 1.6–2.4 in NHS II. OR also higher than reference group for low birth weight < 5.0 lb, shown diagrammatically only. In NHS I, subjects also divided according to mother's figure at age 50 y, thin, medium or heavy. Only in medium group was increased birth weight associated with increased BMI |
| Hulman et al, ¹²⁴ 1998, USA | 28 y, n = 137 (67 m, 70 f) (with complete anthropometric data from birth–28 y, a sample of cohort) | Retrospective. African Americans, offspring of mothers enrolled in Philadelphia Perinatal Collaborative Project—see ref.15 | Obesity: BMI > 27.3 females; BMI > 27.8 males | Birth weight | Chi square test to compare groups: no relationship between birth weight group (< 2500, 2500–3200, 3201–3800 and > 3800 g) and obesity, $\chi^2 = 0.88$, P = 0.83 |

Table A7 Continued

Table A7 Continued Intrauterine growth papers

| Author/journal/year/country | Ages at outcome measurements/no. of subjects | Design/participants | Outcome measure | Birth weight ^a | Main finding |
|---|---|--|--|---|--|
| Rasmussen and Johansson, ⁸⁵ 1998, Sweden | 18.2 y, n = 75,638 | Retrospective. Single male births identified from birth registry 1973–1976. Linked to follow-up data from military service registry. 78% of identified births were term births and height and weight data was available at 18 y. Analyses relevant to this review completed on 1975–1976 births only, 36% of identified births | Overweight: 25 ≤ BMI < 30; severe overweight: BMI ≥ 30 | Birth weight | Multiple logistic regression analyses, odds ratios and 95% CIs calculated. After adjusting for living area and mother's age, educational level, parity and marital status: for groups lower than the reference group, no association between birth weight for gestational age and overweight or severe overweight; for higher than reference groups, positive associations between birth weight for gestational age and overweight and severe overweight; for overweight, odds ratios increased from 1.07 to 1.67 with birth weight group, for severe overweight, odds ratios increased from 1.09 to 1.66 with increasing birth weight group |
| Sorensen et al, ¹²⁵ 1997, Denmark | 18–26 y, n = 4305, all males | Retrospective. Men in 5th conscription district of Denmark, born after 1/1/73 and examined at draft board 1/8/93–31/7/94. 505 men excluded due to asthma | BMI | Birth weight obtained from birth registry | Steady increase in mean BMI from 22.7 at birth weight < 2500 g to 24.9 at birth weight of 4500 g, no statistical test mentioned. Multiple regression models, adjusting for gestational age, birth order, mother's marital status, age and occupation, birth weight associated with BMI, $\beta = 0.82$, <i>s.e.</i> = 0.17 |
| (ii) Within childhood Barker et al, ¹²⁶ 1997, UK | 14–16 y, n = 216, 348 recruited, maternity records obtained for 216 of 242 singleton girls born locally | Retrospective. Girls recruited from five schools in Southampton area, chosen to give mix of social classes. 83% agreed to participate (n = 348). This paper includes singleton girls born locally | BMI. Subscapular and triceps skinfold | Birth weight | Correlation: birth weight positively associated with BMI, significance borderline <i>P</i> = 0.08. After adjusting for BMI, birth weight negatively associated with subscapular skinfold. Subscapular skinfold increased by 7% for every 1 kg decrease in birth weight, CI 1–13%, <i>P</i> = 0.02. No association between birth weight and triceps skinfold. These associations independent of social class |

Table A7 Continued

Table A7 Continued Intrauterine growth papers

| Author/journal/year/country | Ages at outcome measurements/no. of subjects | Design/participants | Outcome measure | Birth weight ^a | Main finding |
|--|---|---|---|--|--|
| Fomon <i>et al.</i> , ¹²⁷ 1984, USA | 8 y, <i>n</i> = 471 at baseline, <i>n</i> = 432 at 8 y (244 m, 188 f) | Prospective. Participants were children who had completed a previous study (ref. 16). For this paper, infants fed formulas containing butterfat or differing appreciably in composition from commercial formulas excluded | BMI, Triceps, subscapular skinfolds | Birth weight measured | Results analysed by feeding and sex groups: breast fed males, <i>n</i> = 73, formula fed males <i>n</i> = 171, breast fed females <i>n</i> = 83, formula fed females <i>n</i> = 105. Using correlation analyses, birth weight was positively related to BMI at 8 y, significant only in formula fed males (<i>r</i> = 0.16, <i>P</i> < 0.05). Association not significant in other groups. Using multiple regression analyses to adjust for weight at 112 days, gain in weight 5–112 days and BMI at 112 days, birth weight was positively related to BMI at 8 y in formula-fed males and breast fed females, <i>P</i> < 0.05. No analyses reported for skinfolds |
| Guillaume <i>et al.</i> , ⁵⁴ 1995, Belgium | 6–13 y, <i>n</i> = 1028 (527 m, 501 f) | Retrospective. Participants selected at random from school classes. Population Wallonian origin, little admixture by immigration. Rural. 70% eligible children participated | BMI | Birth weight reported by mother (a sub-sample checked with written records, no statistical difference) | Using ANOVA, adjusting for age, birth weight positively related to BMI at 6–13 y, <i>P</i> < 0.0001 |
| Harland, <i>et al.</i> , ¹²⁸ 1997 UK | 12–16 y, <i>n</i> = ? at baseline, <i>n</i> = 188 at 12–16 y | Retrospective. Participants students of a state school in socially deprived area in England | BMI (assumed measured) | Birth weight—not stated how obtained | Using correlations; birth weight not related to BMI |
| Kramer <i>et al.</i> , ^{7,4,96,97} 1986, and 1985, Canada | 1, 2 y; <i>n</i> = 462 at baseline, <i>n</i> = 347 at 2 y | Prospective. Recruitment from large university teaching hospital serving socially diverse population. <i>n</i> = 553 eligible, 462 enrolled | BMI, sum triceps + subscapular + superiliac skinfolds | Birth weight | Multiple regression models, stepwise procedure, constructed for BMI and skinfolds at 12 and 24 mo. Birth weight associated with BMI at 12 and 24 mo. (<i>β</i> = 0.23, <i>P</i> < 0.0001 for both relationships) in model including duration of breast feeding, gender and maternal score of 'ideal infant body habitus'. Birth weight associated with sum skinfolds at 12 mo. (<i>β</i> = 0.13, <i>P</i> = 0.02) and 24 mo. (<i>β</i> = 0.17, <i>P</i> = 0.002) in model including age at solid food introduction, duration breast feeding, gender, maternal relative weight and maternal feeding attitude |

Table A7 Continued

Table A7 Continued Intrauterine growth papers

| Author/journal/year/country | Ages at outcome measurements/ no. of subjects | Design/participants | Outcome measure | Birth weight ^a | Main finding |
|--|--|--|--|-------------------------------------|---|
| Maffei et al. ⁵⁷ 1994, Italy | 4, 8, 10, 12 y; n = 1523 total, n = 1363 this paper (676 m, 687 f) | Retrospective. Random sample from four school classes in six areas of north- east Italy. This paper excludes children with low gestational age and includes subjects for whom complete data was available | Obesity: BMI > 95th percentile for age and gender, using French tables as reference (see ref. 13) | Birth weight reported by parents | Odds ratios calculated for likelihood of obesity, birth weight categories unspecified. After adjusting for age, higher birth weight increased likelihood of obesity, in boys OR = 1.001, P < 0.002, and girls OR = 1.001, P = 0.004. Using logistic regression, adjusting for age and including both parents' BMIs, birth weight was positively related to BMI in boys (OR = 1.001, P = 0.02) and girls (effect varies with mother's BMI), P < 0.05 |
| O'Callaghan et al. ⁴⁷ 1997, Australia MUSP (Mater University Study of Pregnancy) | 5 y; n = 7357 at baseline, n = 4062 at 5 y (2133 f, 1929 m) | Prospective. Mothers (n = 8556) enrolled at pregnancy hospital visit (ref. 20). At birth n = 7357, not all of these children followed up due to insufficient funds | Obesity: severe obesity — BMI > 94th percentile; moderate obesity — BMI 85–94th percentile within cohort | Birth weight | Univariate analyses: RR, 95% CIs. Birth weight in 85–94 percentile and ≥ 95th percentile positively related to severe obesity, RR = 1.7, CI 1.2–2.9, RR = 1.8, CI 1.1–2.9 respectively. Birth weight ≥ 95th percentile positively related to moderate obesity RR = 1.8, CI 1.3–2.5. Multivariate analyses included birth weight, size for gestational age, gender, feeding problems, duration of breast feeding, sleeplessness, parental BMIs, maternal education, level of income. Adjusted ORs calculated with 95% CIs. Birth weight ≥ 95th percentile positively related to severe obesity OR = 1.8, CI 1.1–2.9, not moderate obesity. Birth weight 85–94 percentile positively related to moderate obesity OR = 2.0 CI 1.3–3.2, not to severe obesity |

Table A7 Continued

Table A7 Continued Intrauterine growth papers

| <i>Author/journal/year/country</i> | <i>Ages at outcome measurements/no. of subjects</i> | <i>Design/participants</i> | <i>Outcome measure</i> | <i>Birth weight^a</i> | <i>Main finding</i> |
|---|--|---|---|--|---|
| Prescott-Clarke and Primates ⁶³ 1998, UK Health Survey for England | 2–15 y, n = 13,563 (6846 m, 6717 f) | Retrospective. Nationally representative sample selected by random sampling technique | BMI | Birth weight reported | In males and females, with increasing birth weight the proportion of children in the top quintile of BMI increased, and the proportion in the bottom quintile decreased |
| Seidman et al. ⁹⁹ 1991, Israel. Jerusalem Perinatal Study | 0 y, 17 y; duration 17 y; n = ? at baseline, n = 20,747 at 17 y, all males | Prospective. All births from three major wards in Jerusalem 1964–1971. At 17 y those not in defence forces not included. 61.7% females missing | Overweight: BMI ≥ 90th percentile, 24.6; severe overweight: BMI ≥ 97th percentile, 27.8 | Birth weight | When prevalence of overweight and severe overweight expressed by birth weight, strong and linear relationship observed at birth weights > 3000 g (no statistical test mentioned). Using multiple logistic regression, including parental education, area of residence, ethnic origin and birth order, OR for being overweight increased with increasing birth weight, compared to birth weight of 3000–3499 g as reference group. Model for severe overweight included paternal education and ethnic origin, similar results obtained |
| Taylor et al. ¹²⁹ 1997, UK Ten Towns Study | 8–11 y, n = 3010 | Retrospective. Participants from a stratified random sample of 10 schools in each of 10 towns selected on basis of adult cardiovascular mortality. 3728 participated, 3181 with data on birth weight, 3010 singleton births | BMI | Birth weight reported by parents when child age 8–11 y | Birth weight positively correlated to BMI at 8–11 y, r = 0.09, P < 0.0001 |
| Zive et al. ¹³⁰ 1992, USA SCAN study | 4 y, n = 270, ~50% m, 50% f | Retrospective. Recruitment via state-funded pre-schools, children's centres. Low SES families targeted for study. Anglo- and Mexican Americans. 331 respondents, complete data for 270 | BMI, sum triceps + subscapular skinfold | Birth weight reported by mother | Correlations: birth weight significantly correlated with BMI, r = 0.28, P < 0.001 and with sum skinfolds r = 0.16, P ≤ 0.01 |

^aBirth weight measured unless stated otherwise.

Table A8 Maturation papers

| <i>Author/journal/year/country</i> | <i>Ages at risk factor and outcome measurements/no. of subjects</i> | <i>Design/participants</i> | <i>Outcome measure</i> | <i>Risk factor</i> | <i>Main finding</i> |
|--|---|--|---|--|--|
| (i) Childhood to adulthood | | | | | |
| Beunen <i>et al.</i> , ¹⁵⁸ 1994, Belgium Leuven Growth Study | 13 y, annually until 18 y, 30 y, duration 17 y; <i>n</i> = 588 at baseline, <i>n</i> = 115 at 30 y (all male) | Prospective. Subgroup of boys (<i>n</i> = 588) from nationally representative sample of Belgian secondary school boys (<i>n</i> = 21,175) | BMI, triceps, subscapular, suprailiac, medial calf skinfolds | Maturity based on PHV _{age} . Early maturers: PHV _{age} < 13.37 y; average maturers: 13.85 ≤ PHV _{age} ≤ 14.8 y; late maturers PHV _{age} > 15.27 y | One-way ANOVA with Duncan multiple range test used to compare groups. At 30 y, no difference between maturity groups in BMI, triceps, medial calf skinfolds. Subscapular and suprailiac skinfolds significantly largest in early > average > late maturers, <i>P</i> < 0.05, <i>P</i> = 0.07, respectively. Correlations used to relate PHV _{age} and body dimensions at 30 y. Correlation coefficients mostly low, <i>r</i> < 0.1. PHV _{age} related to subscapular (<i>r</i> = -0.34, <i>P</i> < 0.05) and medial calf (<i>r</i> = -0.18, <i>P</i> < 0.05) skinfolds |
| Burke <i>et al.</i> , ¹⁵⁴ 1992, USA CARDIA | 18–30 y, <i>n</i> = 2658 (all female) | Retrospective. Participants randomly recruited. Sampling designed to include equal numbers by age, sex, ethnicity, education level | BMI | Age at menarche | No statistical test reported. In both Black and White women, BMI increased with decreasing age of menarche |
| Garn <i>et al.</i> , ¹⁵⁰ 1986, USA National Collaborative Perinatal Project (NCPP) | 20–35 y, <i>n</i> = 16,868 (all female) | Unclear whether prospective or retrospective (see refs. 6–8). Participants of National Collaborative Perinatal Project. Teenagers in sample excluded | BMI | Age at menarche: early < 11 y; intermediate 12–13 y; late ≥ 14 y | ANOVA and <i>t</i> -tests used to compare groups. Early maturers showed greater BMI than later maturers, 61.17 vs 56.96, <i>P</i> < 0.001. Difference persisted if subjects grouped by age, 20–25 y, 26–30 y, 31–35 y, but became larger with increasing age. Difference persisted if sample restricted on basis of parity or education |
| Garn <i>et al.</i> , ¹⁵⁰ 1986, USA Tecumseh Community Health Study | 20–35 y, <i>n</i> = 476 (all female) | Unclear whether prospective or retrospective (see refs. 6–8). Participants of Tecumseh round II. Teenagers in sample excluded | Triceps, subscapular, iliac, abdominal skinfolds; obesity: abdominal skinfold > 85th percentile | Age at menarche: early < 11 y; intermediate 12–13 y; late ≥ 14 y | ANOVA and <i>t</i> -tests used to compare groups. All skinfolds greater in early maturers than in later maturers, <i>P</i> ≤ 0.001. Subjects of similar age and SES. RR of obesity in early maturing group = 1.7 |

Table A8 Continued

Table A8 Continued Maturation papers

| <i>Author/journal/year/country</i> | <i>Ages at risk factor and outcome measurements/no. of subjects</i> | <i>Design/participants</i> | <i>Outcome measure</i> | <i>Risk factor</i> | <i>Main finding</i> |
|--|--|--|---|--|--|
| Gasser et al., ¹⁴² 1994, Switzerland Zurich Growth Study | 1, 3, 6, 9, 12, 18, 24 mo. annually until 9 y (f), 10 y (m); biannually, then annually depending on hgt until ~18 y; additional adulthood measurement for most subjects 17–25 y, baseline $n = 321$, follow-up $n = 232$ (112 f, 120 m) | Prospective. Participants—no information given, see ref. Gasser 1990 | BMI. 'Fat' group: adult BMI in top tertile; 'lean' group: adult BMI in bottom tertile. Extremity skinfolds (triceps + biceps). Trunk skinfolds (subscapular + suprailiac) | Pattern of velocity of change in BMI throughout childhood | Structural average growth curves constructed. Relationship of mean velocity BMI with age for 'fat' and 'lean' groups displayed graphically, males and females separately. 'Fat' and 'lean' groups compared. 'Fat' males and females showed larger peaks and troughs in BMI velocity. Difference especially clear at puberty where 'fat' groups showed larger peak. In general 'fat' males and females showed higher BMI velocity from 3 y into adulthood. Comparing mean increase in BMI between 3 and 10 y (3 and 12 y boys) and between 15.7 y and adult (16.6 y boys), 'lean' groups showed much smaller increases during these pre- and postpubertal periods. During puberty, 'fat' males and females demonstrated higher pre- and postpubertal fat spurts, as measured by extremity and trunk skinfolds |
| Gasser et al., ¹⁴³ 1994, Switzerland Zurich Growth Study | As above | As above | As above | Pattern of velocity of change in fat and lean area of upper arm throughout childhood | As above but growth curves constructed for mean velocity fat and lean areas of arm. 'Fat' and 'lean' groups compared. 'Fat' males and females showed higher velocity of lean area from age 6 to puberty. In males, maximum velocity ~6 mo. earlier in 'fat' males. 'Fat' males and females showed larger peaks and troughs in velocity of fat area. Difference especially clear at puberty in males |

Table A8 Continued

Table A8 Continued Maturational papers

| <i>Author/journal/year/country</i> | <i>Ages at risk factor and outcome measurements/no. of subjects</i> | <i>Design/participants</i> | <i>Outcome measure</i> | <i>Risk factor</i> | <i>Main finding</i> |
|--|---|--|--|---|--|
| Guo <i>et al.</i> , ¹⁴⁸ 1997, USA Fels Longitudinal Study | 8 y, 23 y; duration 15 y, <i>n</i> = 244 (114 m, 130 f) | Prospective. Participants of Fels study (see ref. 17) with ≥ 6 body composition measurements between 8 and 23 y. All Caucasian | % body fat based on underwater weighing | Rate of maturation (skeletal age – chronological age (SA – CA). Mean SA – CA over period from 8 y to maturity obtained. Rapid maturers: value > 1. Intermediate: –1 < value < 1. Slow maturers: value < –1 | Using multiple regression analyses and adjusting for age, age ² and age ³ , in males, slow maturers have lower skinfolds than rapid maturers, <i>P</i> < 0.05. In females, intermediate maturers have lower skinfolds than rapid maturers, <i>P</i> < 0.05 and also lower than slow maturers, not significant |
| Stiervogel <i>et al.</i> , ¹⁴⁹ 1991, USA Fels Longitudinal Study | 2 y, semi-annually until 18 y; duration 16 y; <i>n</i> = 473 at baseline, <i>n</i> = 330 at 18 y (167 m, 163 f) | Prospective. Participants of Fels study. All Caucasian and from wide range of SES | BMI | Age at maximum BMI; age at maximum velocity BMI; age at minimum BMI | Participants analysed in two groups depending on whether three or four parameter polynomial growth curve used to fit data. For four parameter model, <i>n</i> = 417. Age at minimum BMI negatively correlated to BMI at 18 y in boys <i>r</i> = –0.46, and girls <i>r</i> = –0.54, <i>P</i> < 0.01. Age at maximum BMI positively correlated to BMI at 18 y in boys <i>r</i> = 0.57, and girls <i>r</i> = 0.64, <i>P</i> < 0.01. Age at maximum velocity BMI unrelated to BMI at 18 y. For three parameter model <i>n</i> = 56. No correlations between age at max BMI, min BMI or max velocity BMI and BMI at 18 y |
| Miller <i>et al.</i> , ¹⁵⁰ 1972, UK Newcastle Thousand Families Study | 0 y, 3, 5, 9, 13, 14, 15 y; 22 y; <i>n</i> = 1142 (m + f) at baseline; <i>n</i> = 442 (m + f) at 22 y (201 m, 241 f) | Prospective. Original sample all babies born in Newcastle in May and June 1947. Only data from females relevant to this review | Weight-for-height (Kemsley 1962 British data used as reference data) | Age at menarche (unclear whether recorded prospectively or retrospectively) | No statistical test reported. Age of menarche negatively related to weight-for-height at 22 y |

Table A8 Continued

Table A8 Continued Maturation papers

| Author/journal/year/country | Ages at risk factor and outcome measurements/no. of subjects | Design/participants | Outcome measure | Risk factor | Main finding |
|---|---|---|--|--|--|
| Power et al. ¹⁷ 1997, UK 1958 UK birth cohort | 7, 11 y, 33 y, duration 26 y (for this paper); <i>n</i> = ~17,000 at baseline; <i>n</i> = 11,407 at 33 y | Prospective. Participants all births 3–9 March 1958. Immigrants born in same week subsequently included in follow-ups | BMI | Tanner stage at 11 y (assessed by doctor). At 16 y: age at menarche (girls, reported) stage of axillary hair (boys) | Test for trend across maturation groups. Based on puberty assessed at 11 y: BMI at 33 y increased with advancing stage of puberty in females, <i>P</i> = 0.003, and males (relationship not significant), <i>P</i> = 0.3. Based on maturation assessed at 16 y: BMI at 33 y increased with earlier age of menarche in females, <i>P</i> = 0.003, and advancing maturation in males, <i>P</i> = 0.003 |
| Freeman et al. ¹⁴⁰ 1995, UK 1958 UK birth cohort | 7, 11 y, 23 y; duration 16 y (for this paper); at 7 y <i>n</i> = 6874 m, 6422 f; at 11 y <i>n</i> = 6381 m, 6118 f; at 23 y <i>n</i> = 6134 m, 6145 f | As above | Obesity: BMI ≥ 30 | Maturation: early maturer—tall at 7 y compared to at 16 y; late maturer—short at 7 y compared to at 16 y (both at 7 and 16 y, subjects classified as short, medium and tall, separate cut-offs for males, females at 16 y) | No statistical test mentioned. At 23 y, prevalence of obesity higher in early maturers (tall at 7, short at 16 y) greater than in late maturers (short at 7, tall at 16 y), 4.3% vs 1.4% in males, 9.5% vs 3.8% in females |
| Stark et al. ¹⁴¹ 1989, UK 1958 UK birth cohort | 7 y, 11 y, 16 y, duration 9 y, <i>n</i> = ~17,000 baseline (boys and girls); <i>n</i> = 3018 girls with complete data for this paper | As above. Only girls included in this paper | Relative weight (observed wgt as % of expected wgt obtained by regression of log wgt on log hgt) | Age at menarche (reported) | No mention of statistical test. With increasing age of menarche, percentage of girls with relative weight 101–120% and > 120% decreased, those with relative weight < 100% increased, at all ages, i.e. 7 y, 11 y, 16 y |
| Prokopec and Bellisle, ¹⁵⁶ 1993, Czech Republic Co-ordinated by Centre International de l'Entrance (CIEP) | 1, 3, 6, 9, 12 mo. every 6 mo. until 18 y; duration 18 y, <i>n</i> = 300 baseline, <i>n</i> = 158 at 18 y (80 m, 78 f) | Prospective. Children selected at random from particular district, children born on Wednesday included | BMI | Adiposity rebound | Not all data used, participants with BMI > 90th percentile (fat) and BMI < 10th percentile (lean) at 18 y compared. Males: fat <i>n</i> = 9, lean <i>n</i> = 10. Females: fat <i>n</i> = 10, lean <i>n</i> = 8. ANOVA used to test for group mean differences. Age at adiposity rebound significantly lower in males fat at 18 y (4.6 y vs 7.8 y, <i>P</i> < 0.02) and females fat at 18 y (5.3 y vs 7.4 y, <i>P</i> < 0.05) |

Table A8 Continued

Table A8 Continued Maturation papers

| Author/journal/year/country | Ages at risk factor and outcome measurements/no. of subjects | Design/participants | Outcome measure | Risk factor | Main finding |
|--|--|---|---------------------------|---|--|
| Rolland-Cachera <i>et al.</i> , ¹⁴⁴ 1987, France Co-ordinated by Centre International de l'Enfance (CIEP) | ~ every 6 mo. until 16 y; 21 y; duration 21 y; n = 164 (85 m, 79 f) | Prospective. Participants — no information given, see below. This paper includes subjects measured beyond 16 y | BMI, subscapular skinfold | Adiposity rebound: <5.5 y = advanced; 5.5–7 y = average; >7 y = delayed | ANOVA used to compare maturity groups. Boys with advanced adiposity rebound showed higher BMI ($P=0.01$) and subscapular skinfold ($P=0.02$) than those with delayed adiposity rebound. Similar relationships observed in girls, but less significant. Girls with advanced adiposity rebound showed higher BMI ($P=0.12$) and subscapular skinfold ($P=0.08$). Significance greater if comparison made only between advanced and delayed adiposity rebound groups (for BMI, $P=0.04$, subscapular skinfold $P=0.03$) |
| Rolland-Cachera <i>et al.</i> , ¹⁴⁵ 1984, France As above | ~ every 6 mo. until 16 y, 16 y; duration 16 y, n = ? at baseline, n = 151 at 14 y, n = 114 at 16 y (62 m, 52 f) | Prospective. Participants — no information given, see refs. 10, 11, or 12. This paper includes subjects measured until 14 y | BMI | Adiposity rebound: <5.5 y = advanced; 5.5–7 y = average; >7 y = delayed | Variance analysis: boys and girls with advanced adiposity rebound were more adipose at 16 y than those with average adiposity rebound, who were in turn more adipose at 16 y than those with delayed adiposity rebound ($P < 0.01$) |
| St. George <i>et al.</i> , ¹⁵⁷ 1994, New Zealand Dunedin Multidisciplinary Health and Development Study | 3 y, every 2 y until 15 y, 18 y; duration 15 y, n = 1139 baseline (m + f), n = 415 girls with complete data in this paper | Prospective. Participants of birth cohort — all births in Dunedin over 1 y, 1/4/72–31/3/73 | BMI | Age at menarche | Data displayed graphically, no statistical test mentioned. BMI at 18 y was greatest in those with menarche < 12 y, less in those with menarche at 12–13 y, less again in those with menarche at 13–14 y and least in those with menarche > 14 y. This was true at all time-points 9–18 y inclusive |

Table A8 Continued

Table A8 Continued Maturation papers

| <i>Author/journal/year/country</i> | <i>Ages at risk factor and outcome measurements/no. of subjects</i> | <i>Design/participants</i> | <i>Outcome measure</i> | <i>Risk factor</i> | <i>Main finding</i> |
|--|--|---|---|---|--|
| Sherman <i>et al.</i> , ¹⁵⁵ 1981, USA | 19–73 y, <i>n</i> = 1221 | Retrospective. Participants female university student volunteers and daughters of this cohort | BMI current—based on reported current hgt and wgt. BMI at 18 y—based on reported current hgt and recalled wgt at 18 y | Age of menarche, recalled while student at university for original cohort, recorded prospectively for daughters | Groups compared using Student's <i>t</i> -test. Based on BMI at 18 y, menarche in the highest quartile of BMI was 12.4 y, compared to 13.0 y in the lowest BMI quartile, <i>P</i> < 0.001. Based on current BMI, menarche in the highest quartile of BMI was 12.5 y, compared to 12.8 y in the lowest BMI quartile, <i>P</i> < 0.001 |
| van Lenthe <i>et al.</i> , ¹⁴⁶ 1996, The Netherlands AGHS (Amsterdam Growth and Health Study) | 13, 14, 15, 16, 21, 27 y; duration 14 y; <i>n</i> = 307 at baseline, <i>n</i> = 177 at 27 y (79 m, 98 f) | Prospective. Participants complete 1st and 2nd form of Amsterdam school, no refusals to participate. SES above population average | BMI, sum of skinfolds (biceps, triceps, subscapular, suprailliac) | Maturation as measured by: (i) skeletal age—rapid maturer: skeletal age > 3 mo. in advance of chronological age at all four time-points 13–16 y; slow maturer: skeletal age > 3 mo. behind chronological age at all four time-points 13–16 y; (ii) PHV _{age} (boys only); (iii) age at menarche (girls); (ii) and (iii) early maturer = bottom tertile, late maturer = top tertile | Group differences analysed by sex-specific MANOVA for repeated measures. Boys: skinfolds—whether maturity based on skeletal age or PHV, decrease in adolescence occurred earlier in rapid maturers. BMI—larger increase in slow maturers at age 16–21 y. If maturity based on skeletal age, BMI and skinfolds greater in rapid maturers 13–27 y but difference not significant if maturity based on PHV _{age} . Girls: skinfolds—whether maturity based on skeletal age or age at menarche, increase at 16–21 y larger in slow maturers. Whether maturity based on skeletal age or age at menarche, both skinfolds and BMI greater in rapid/early maturers throughout time period 13–27 y |

Table A8 Continued

Table A8 Continued Maturation papers

| <i>Author/journal/year/country</i> | <i>Ages at risk factor and outcome measurements/no. of subjects</i> | <i>Design/participants</i> | <i>Outcome measure</i> | <i>Risk factor</i> | <i>Main finding</i> |
|--|--|--|---|--|--|
| Post and Kemper, ¹⁴⁷ 1993, The Netherlands AGHS (Amsterdam Growth and Health Study) | 13, 14, 15, 16, 21 y; duration 8 y; baseline $n = 307$ at 21 y $n = 200$ (93 m, 107 f) | As above | Sum of skinfolds (biceps, triceps, subscapular, suprailiac) | Maturation as measured by skeletal age, see above for definition | Only early (rapid) and late maturers compared with each other. Group differences analysed using ANOVA. In both boys and girls, early maturers demonstrated higher skinfolds throughout study, $P \leq 0.05$ |
| Wellens et al, ¹⁵¹ 1992, USA 1970 cohort | 19.2 y, $n = 342$ | Retrospective. Participants: cohorts of White female university students recruited in 1970 | BMI, triceps skinfold | Age of menarche | Differences in BMI and skinfold across four groups of menarcheal age analysed by ANOVA. As age of menarche increased, BMI and triceps skinfold decreased although not statistically significant |
| Wellens et al, ¹⁵¹ 1992, USA 1987 cohort | 18.8 y, $n = 109$ | Retrospective. Participants: cohorts of White female university students recruited in 1987 | BMI, triceps skinfold | Age of menarche | Differences in BMI and skinfold across four groups of menarcheal age analysed by ANOVA. As age of menarche increased, BMI and triceps skinfold decreased although not statistically significant |
| Wellens et al, ¹⁵¹ 1992, USA Fels study | 18.3 y, $n = 234$ | Retrospective. Participants: females in Fels study, similar to cohorts described above | BMI, triceps skinfold | Age of menarche | Differences in BMI and skinfold across four groups of menarcheal age analysed by ANOVA. Those with menarche < 12 y showed greater BMI than those with menarche 12–12.9 y, 13–13.9 y or > 13.9 y, $P < 0.01$. Those with menarche < 12 y showed greater triceps skinfold than those with menarche > 13.9 y, $P < 0.05$ |

Table A8 Continued

Table A8 Continued Maturations papers

| Author/journal/year/country | Ages at risk factor and outcome measurements/ no. of subjects | Design/participants | Outcome measure | Risk factor | Main finding |
|--|--|--|--|--|--|
| (ii) Within childhood | | | | | |
| Hediger et al, ¹⁵³ 1995, USA | 14–18 y, 15–19 y, duration 1 y, n = 851 at baseline, n = 668 at 15–19 y (all female) | Prospective. Participants female volunteers from two high schools, grades 9–11. Three rounds of recruitment. 70–93% of total volunteered. Ethnicity: 61% White, 16% Black, 17% Puerto-Rican, 6% other | Δ triceps skinfold, Δ subscapular skinfold | Gynaecologic age (chronologic age – age at menarche) | Multiple linear regression analysis with either ΔBMI, and Δ triceps or Δ subscapular skinfold as dependent variable, and which appears to include all of following as covariates (not entirely clear): age, gynaecologic age, ethnicity, late maturation, cigarette smoking, initial skinfold status. No influence of gynaecologic age or late maturation on Δ triceps or subscapular skinfold |
| Knishkowsky et al, ¹⁵² 1989, Israel | 6 y, 14 y, duration 7.5 y, n = 312 f at baseline, n = 288 f at 14 y | Prospective. Participants boys and girls at two elementary schools in Jerusalem. Only data from girls relevant to review. 39% all mothers Israeli born, 18% Asian, 26% N. African, 16% European/American | BMI | Age at menarche (reported at follow-up) | ANOVA used to test group differences between girls menstruating and not menstruating at follow-up (14 y). Menstruating girls (n = 215) had higher BMI, 20.1 ± 2.6 than non-menstruating girls 18.3 ± 2.1, P < 0.001. Menstruating girls (n = 215) had higher age, 13 y 10 mo. ± 3.9 mo. than non-menstruating girls 13 y 8.8 mo. ± 3.6 mo., P = 0.01. Differences in BMI persisted after adjusting for age using ANCOVA, P < 0.001 |

Adiposity rebound = lowest BMI recorded before second rise in adiposity, which usually occurs at ~ 6 y on BMI and skinfold charts. PHV_{age} = age at peak height velocity.

Table A9 Physical activity papers

| Author/year/country | Ages at measurements/ no. of subjects | Design/participants | Outcome (measured unless stated otherwise) | Activity risk factor/method of measurement | Main finding |
|--|--|---|---|---|---|
| (i) Childhood to adulthood Raitakari <i>et al.</i> , ²⁰ 1994, Finland Cardiovascular Risk in Young Finns Study | 12–18 y, 15–21 y, 18–24 y, duration 6 y, <i>n</i> = 3,596, baseline <i>n</i> = 961 at 18–24 y | Prospective. Original sample randomly chosen from population register. No information on dropouts | BMI, subscapular skinfold | Leisure-time physical activity assessed by questionnaire and index calculated accounting for intensity, duration and monthly frequency | Student <i>t</i> -test used to compare subjects who were active (<i>n</i> = 23 f, 44 m) with those who were sedentary (<i>n</i> = 55 f, 33 m) at all three measurements. Subscapular skinfold was lower in active subjects in males (9.9 vs 12.1, <i>P</i> = 0.01) and females (10.5 vs 14.2, <i>P</i> = 0.005). No difference in BMI in males (21.8 vs 21.9) or females (22.6 vs 22.1) (active vs sedentary) |
| Twisk <i>et al.</i> , ¹⁷² 1997, The Netherlands Amsterdam Growth and Health Study (AGHS) | 13, 14, 15, 16, 21, 27, 29 y, duration 16 y, <i>n</i> = 233 baseline, <i>n</i> = 181 at 29 y (98 f, 83 m) | Prospective. Participants complete 1st and 2nd form of Amsterdam school, no refusals to participate. SES above population average | Sum of biceps, triceps, subscapular, suprailiac skinfolts (SSF) | Total activity by structured interview concerning previous 3 mo. Duration and intensity combined to give weighted score expressed in MET's | Multiple linear regression analyses including variables based on significance in univariate analyses. Daily physical activity in adolescence and young adulthood was negatively related to SSF ($\beta = -0.13$, <i>P</i> = 0.05). Relationship stronger if entire longitudinal period considered (further into adulthood; $\beta = -0.20$, <i>P</i> < 0.01) |
| Twisk <i>et al.</i> , ¹⁷³ 1997, The Netherlands AGHS | 13, 14, 15, 16, 21, 27 y, duration 14 y, <i>n</i> = 233 baseline, <i>n</i> = 181 at 27 y (98 f, 83 m) | As above | As above | As above | Statistical model incorporating parameters calculated using generalized estimating equations. After adjusting for biological age, sex, sum of skinfolts, VO _{2max} , and high risk for sum skinfolts ($\geq 20\%$ body fat for males, 30% for females), was negatively related to daily activity (OR = 0.81, <i>P</i> = 0.01). Participants who are inactive/become inactive are more likely to be in or move into risk group for sum of skinfolts |

Table A9 Continued

Table A9 Continued Physical activity papers

| Author/year/country | Ages at measurements/ no. of subjects | Design/participants | Outcome (measured unless stated otherwise) | Activity risk factor/method of measurement | Main finding |
|--|--|---|--|--|--|
| van Lenthe <i>et al.</i> , ¹⁷⁴ 1998, The Netherlands AGHS | 13, 14, 15, 16, 21, 27 y, duration 14 y, <i>n</i> = 233 baseline, <i>n</i> = 182 at 27 y (98 f, 84 m) | As above | Δ subscapular skinfold | As above | 'General estimating equations' (longitudinal regression analysis), adjusting for sum skinfolds between 13 and 27 y: males — no influence of activity on Δ subscapular skinfold; females — activity negatively related to Δ subscapular skinfold, $\beta = -0.10$, CI -0.15 , -0.05 , $P < 0.001$. Multiple regression analysis with subscapular skinfold at 27 y as dependent variable and initially including activity, diet, smoking and alcohol intake as covariates, non-significant covariates removed by backwards elimination procedure: males — activity positively related to subscapular skinfold, $\beta = 0.15$, SE = 0.07, $P = 0.03$. Females — no influence |
| (ii) <i>Within childhood</i> Baseline measurement pre-1 year of age (i.e. pre-walking) Berkowitz <i>et al.</i> , ⁹⁵ 1985, USA | 1–3 days, 6.5 y, duration ~6.5 y, <i>n</i> = 112 baseline, <i>n</i> = 52 at 6.5 y (25 m, 27 f) | Prospective. Participants recruited from cohort of another study — see ref. 9 | Log BMI, triceps skinfold | Neonatal non-crying activity — frequency and intensity by electronic activity monitor | Neonatal non-crying amplitude (intensity of movements) negatively correlated to triceps skinfold at 6.5 y, $r = -0.34$, $P < 0.05$ |
| Davies <i>et al.</i> , ¹⁶⁸ 1991, UK | 12 wks, 2 y, duration 2 y, <i>n</i> = 52 baseline, <i>n</i> = 33 at 2 y | Prospective. Participants recruited from large maternity hospital | BMI, sum triceps + subscapular skinfolds | Energy expenditure measured by doubly labelled water | No significant correlation between energy expenditure at 12 wks and BMI or skinfolds at 2 y |
| Davies <i>et al.</i> , ¹⁶⁹ 1993, UK | 12 wks, 6.7 y, duration 6 y, <i>n</i> = ? baseline, <i>n</i> = 24 at 6 y | As above | Fat mass relative to fat-free mass | Energy expenditure measured by doubly labelled water | No significant correlation between body fatness at 6.7 y and energy expenditure at 12 wks expressed relative to fat-free mass, $r = 0.11$, $t = 0.52$ |

Table A9 Continued

Table A9 Continued Physical activity papers

| Author/year/country | Ages at measurements/ no. of subjects | Design/participants | Outcome (measured unless stated otherwise) | Activity risk factor/method of measurement | Main finding |
|---|---|---|--|--|--|
| Ku <i>et al.</i> ¹⁷⁰ 1981, USA NB. Also falls into 'post- walking' group below | 6 mo., 1, 2, 3, 4, 8 y, duration 8 y, n = 450 baseline, n = 170 at 8 y (83 m, 87 f) | Prospective. Participants — for recruitment details see refs. 13–17 | % body fat at 8 y by underwater weighing | Physical activity measured at 6 mo., 1, 2, 3, 4 y by parents by 24 h activity record for every 5 min. At 8 y children recorded activity assisted by parents | Girls: no significant correlation between activity at 6 mo. and % body fat at 8 y. Boys: no significant correlation between activity at 6 mo. and % body fat at 8 y |
| O'Callaghan <i>et al.</i> ⁴⁷ 1997, Australia MUSP (Mater University Study of Pregnancy) | Baseline 0 y, follow-up 5 y, n = 7357 baseline, n = 4062 at 5 y (2133 f, 1929 m) | Prospective. Mothers (n = 8556) enrolled at pregnancy hospital visit (ref. 20). At birth n = 7357, not all of these children followed up due to insufficient funds | Obesity: severe obesity— BMI > 94th percentile; moderate obesity—BMI 85– 94th percentile within cohort | Frequency of 'overactivity' reported by mother when child 6 mo. No definition given to mother | Univariate analyses: RR, 95% CIs. No relationship between activity at 6 mo. and obesity at 5 y |
| Wells <i>et al.</i> ¹⁷¹ 1996, UK | 12 wks, 2–3.5 y, duration 2– 3.5 y, n = 50 baseline, n = 24– 28 at 2–3.5 y | Prospective. Participants recruited from large maternity hospital | % fat (deuterium dilution), skinfolds, BMI | Components of infant energy expenditure: sleeping metabolic rate, total energy expenditure, energy spent on physical activity | Multiple regression analyses: after adjusting for body weight or fat-free mass, no relationships between total or resting infant energy expenditure and childhood fatness |
| Baseline measurement post- 1 year of age (i.e. post- walking) | | | | | |
| Beunen <i>et al.</i> ¹⁷⁵ 1992, Belgium Leuven Growth Study | 12, 13, 14, 15, 16 17 y; duration 5 y, n = ? baseline, n = 588 at 17 y | Prospective. Subgroup of boys (n = 588) from nationally representative sample of Belgian secondary school boys (n = 21,175) | Triceps, subscapular, suprailiac, calf skinfolds | Active (> 5 h/wk sport) vs inactive (\leq 1.5 h/wk sport) in addition to compulsory physical education for first 3 y of study period | General linear model with repeat measures; no difference in any of skinfolds between active (n = 32) and inactive (n = 32) boys |
| Dietz and Gortmaker, ¹⁷⁶ 1985, USA (NHES) | 6–11 y, 12–17 y, duration 6 y, n = 2,153 | Prospective. Participants those who had participated in both NHES I and II | Triceps skinfold. Obesity: triceps \geq 85th percentile; superobesity: triceps \geq 95th percentile | Hours of television watching per day by questionnaire to parents | Weighted stepwise regressions: positive relationships between television viewing at age 6–11 y and obesity ($\beta = 0.08$, $P < 0.07$) and superobesity ($\beta = 0.06$, $P < 0.03$) at age 12–17 y, after controlling for obesity at age 6–11 and socio-economic characteristics |

Table A9 Continued

Table A9 Continued Physical activity papers

| Author/year/country | Ages at measurements/ no. of subjects | Design/participants | Outcome (measured unless stated otherwise) | Activity risk factor/method of measurement | Main finding |
|--|---|--|--|---|---|
| Goran et al, ⁴⁴ 1998, USA | 5.2 y, annual to 8.3 y, duration 3 y, n = 75 baseline, n = 53 at 8.3 y | Prospective. Participants recruited by newspaper advertisements and word of mouth | Rate of change in fat mass adjusted for fat-free mass (fat and fat free mass measured by DXA) | TEE (total energy expenditure) by doubly labelled water, REE (resting energy expenditure) by indirect calorimetry | Using ANOVA, rate of change in fat mass adjusted for fat- free mass positively related to TEE ($r = 0.28$, $P = 0.025$), TEE adjusted for REE ($r = 0.33$, $P = 0.08$) and unrelated to REE ($r = 0.14$, $P = 0.24$). Assume (not explicit but likely) TEE, REE are from baseline measurements |
| Klesges et al, ⁴⁵ 1995, USA | 4.4 y, 5.4, 6.4 y, duration 2 y, n = 203 baseline, n = 146 at 6.4 y | Prospective. Participants recruited through paediatricians, day-care centres, churches. Obese over-sampled | Δ BMI | Leisure activity, structured activity, and aerobic activity all assessed by both parents (asked whether children did much less, about the same, or much more than other children) | Multiple regression analysis including adjustment for baseline BMI, gender, age, parental fatness and interactions between gender and parental fatness. In model including these covariates and also dietary intake, over 2 y study period higher baseline aerobic activity and increased leisure activity were associated with decrease in BMI ($\beta = -0.32$, $P = 0.03$, and $\beta = -0.04$, $P = 0.01$ respectively) |
| Ku et al, ¹⁷⁰ 1981, USA NB. Also falls into 'pre-walking' group above | 6 mo., 1, 2, 3, 4, 8 y, duration 8 y, n = 450 baseline, n = 170 at 8 y (83 m, 87 f) | Prospective. Participants— for recruitment details see refs. 13–17 | % body fat at 8 y by underwater weighing | Physical activity measured at 6 mo., 1, 2, 3, 4 y by parents by 24 h activity record for every 5 min. At 8 y children recorded activity assisted by parents | In girls no significant correlation between activity at any age and % body fat at 8 y. In boys no significant correlation between activity at 6 mo., 1 y, 2 y or 8 y and % body fat at 8 y, but significant negative correlations between activity at 3 y, 4 y and % body fat at 8 y, and between and sum activity 6 mo.–8 y and % body fat at 8 y |

Table A9 Continued

Table A9 Continued Physical activity papers

| <i>Author/year/country</i> | <i>Ages at measurements/ no. of subjects</i> | <i>Design/participants</i> | <i>Outcome (measured unless stated otherwise)</i> | <i>Activity risk factor/method of measurement</i> | <i>Main finding</i> |
|--|--|--|---|--|--|
| Maffei <i>et al.</i> ⁴⁶ 1998, Italy | 8.6 y, 12.6 y, duration 4 y, <i>n</i> = 298 baseline, <i>n</i> = 112 at 12.6 y | Prospective. Participants recruited through private and public schools. All Caucasian | BMI, obesity: relative BMI > 120% (Rolland-Cachera BMI tables as reference) | Extra-curricular physical activity and vigorous play (min/day) and television watching min/day by questionnaire completed by parents with paediatrician | Three different types of multivariate analysis, with age, gender, dietary variables, parental BMI, television viewing time and vigorous activity included as covariates. No significant effects of television viewing time or vigorous play/activity in any analysis |
| Moore <i>et al.</i> ¹⁶⁷ 1995, USA (Framingham Offspring Study) | 4 y, 5 y, 6 y, duration 2.5 y, <i>n</i> = 106 baseline, <i>n</i> = 97 at 6 y | Prospective. Participants invited on basis of geographical area, good health, and both parents at home | BMI, triceps, subscapular skinfolds | Physical activity measured twice each year by Caltrac accelerometer for 2 × 5 days, 6 mo. apart | Change in triceps skinfold over study period: active girls + 1.0 mm, inactive girls + 1.75 mm active boys − 0.75 mm, inactive boys + 0.25 mm. Odds ratios calculated for relative risk of increasing anthropometry slope with low levels of activity (below median). For inactive children, risk was greater for increasing triceps skinfold OR = 2.6, subscapular skinfold OR = 1.3, or BMI OR = 1.5, non-significant. Logistic regression analysis: after adjusting for age, hours of television watched per day, energy intake and BMI of parents, lower activity increases risk of increasing skinfold slope for leaner subjects (triceps ≤ median) OR = 2.9, CI 0.6–13.1, and fatter subjects (triceps > median) OR = 5.8, CI 1.1–31.3 |

Table A9 Continued

Table A9 Continued Physical activity papers

| Author/year/country | Ages at measurements/ no. of subjects | Design/participants | Outcome (measured unless stated otherwise) | Activity risk factor/method of measurement | Main finding |
|---|---|---|---|--|--|
| Parizkova, ¹⁷⁷ 1968, Czech Republic | 11 y annual until 15 y, duration 4 y, $n = 146$ baseline, $n = 96$ at 15 y | Prospective. No details except $n = 146$ selected from larger number of subjects. Similar SES, all attended similar state schools | % body fat and % body lean by underwater weighing | Record of activity in sport clubs and questionnaires concerning time and intensity in unorganized activities. Four groups based on consistent physical activity | Statistical tests not stated, probably t -tests. At baseline % lean similar across activity groups. At 15 y, all three more active groups higher % lean than least active group. Over study period, % lean increased significantly in most active group but no change in least active group. At 15 y, % body fat lower in most active group |
| Robinson, ¹⁷⁸ 1993, USA | 12.4 y, 13.4 y, 14.4 y; duration 2 y, $n = 536$ baseline, $n = 279$ at 14.4 y | Prospective. Participants the randomly allocated control group of all 6th and 7th grade girls from four schools unless withdrawn/refused | Δ BMI, Δ triceps skinfold both adjusted for sexual maturity. Obesity: BMI > 85th percentile or triceps > 85th percentile, adjusted for sexual maturity | Television watching hours/day and physical activity by questionnaire to children | Spearman's rank correlation analyses: television viewing was not related to sexual maturity adjusted Δ BMI ($r = 0.03$, $P = 0.62$) or triceps skinfold ($r = 0.03$, $P = 0.54$). Multiple linear regression: after adjusting for age, race, parent education, parent fatness and baseline physical activity, television viewing was not related to sexual maturity, adjusted Δ BMI ($\beta = 0.05$, $P = 0.82$) or Δ triceps skinfold ($\beta = -0.2$, $P = 0.67$). Results were unchanged if analysed with obesity as dependent variable in logistic regression |
| Shapiro et al, ¹⁷⁹ 1984, USA | 6 mo., 1, 2, 3, 4, 6 y, 9 y; duration 8.5 y, $n = 450$ baseline, $n = 170$ at 9 y (83 m, 87 f); analyses on $n = 149$ | Prospective. Sample selected from 1193 birth records filed in 1969. Parents who could be located at 6 mo. invited to participate if planning to be in area for 3 y | Sum of triceps, subscapular, suprailiac and chest skinfolds | Physical activity assessed at each time point by 1 day records, completed by parents at 6 mo. - 6 y and children at 9 y | Sum of activity score over study period negatively related to sum of skinfolds at 9 y, $r = -0.18$, $P < 0.05$ |

Table A10 Dietary papers

| Author/journal/year/country | Ages at measurements/ no. of subjects | Design/participants | Outcome (measured unless stated otherwise) | Activity risk factor/ method of measurement | Main finding |
|--|---|---|---|--|--|
| Infant feeding Agras et al, ⁹³ 1990, USA | 2, 4 wks, 12 mo., 20 mo., 2 y; duration 2 y, n = 99 baseline, n = 54 at 6 y | Prospective. Children recruited from hospital in first week of life | Triceps skinfold, log BMI | Sucking behaviour (measured in laboratory) — caloric intake during sucking, active feeding time, sucking pressure, interburst interval, no. feeds/day, duration of breast-feeding, introduction solid foods | Univariate analyses: at 3 y, solid food delayed > 5 mo., breast-feeding > 5 mo., sucking pressure all positively related to log BMI. At 6 y, breast-feeding > 5 mo. positively related, solid food, sucking pressure, no. feedings/day unrelated to log BMI. Multivariate analyses: after adjustment for several factors including parent education and BMI: at 3 y, sucking pressure and solid food significantly positively related to log BMI. At 6 y, breast-feeding > 5 mo. positively related to log BMI. At 6 y, no relationships between feeding behaviour and triceps skinfold |
| Agras et al, ⁹⁴ 1987, USA | 2, 4 wks, 12 mo., 20 mo., 2, 3 y, 6 y; duration 6 y, n = 99 baseline, n = 79 at 2 y | As above | As above | As above | In multiple regression models adjusting for several factors, including parent education and BMI, shorter interburst interval was associated with greater triceps skinfold at 1 y ($P < 0.02$). Pressure of suckling ($P < 0.006$) and no. of feeds per day ($P < 0.01$) were positively associated with triceps skinfold at 2 y. Number of feeds per day was positively associated with (log) BMI at 1 y. No significant predictors for (log) BMI at 2 y. The more adipose infant at 1 and 2 y sucked more rapidly, at higher pressure, with longer suck and burst duration and shorter burst interval. Associated with higher caloric intake |

Table A10 Continued

Table A10 Continued Dietary papers

| Author/journal/year/country | Ages at measurements/ no. of subjects | Design/participants | Outcome (measured unless stated otherwise) | Activity risk factor/ method of measurement | Main finding |
|---|--|---|---|--|---|
| Birkbeck et al. ²⁰² 1985, New Zealand Dunedin Multidisciplinary Health and Development Study (DMHDS) | 0, 3 y, 7 y; duration 7 y, n > 2000 baseline, n = 565 at 7 y | Prospective. Participants derived from a cohort of > 2000 consecutive births at Dunedin's only maternity hospital in 1972–1973 | Triceps skinfold, subscapular skinfold, BMI | Exclusive breast vs formula feeding for at least first 12 wks | Student t-test used to compare breast fed and formula fed groups. At 7 y, no difference in triceps or subscapular skinfold or BMI |
| Dewey et al. ²⁰³ 1993, USA | Anthropometry: monthly from 1–18 mo., +21, 24 mo. diet: every 3 mo. from 3– 18 mo.; duration 2 y, n = ? baseline, n = 75 at 1 y (41 breast-fed, 34 formula fed) | Prospective. Stratified matching for breast and formula groups for several factors including SES. Recruitment refer to refs. 15–17 | Weight-for-length Z-scores, skinfold thicknesses, estimated % body fat from wgt, hgt, age and sex (prediction equation) | Breast or formula feeding, energy intake by 4 day weighed records, breast milk included | ANOVA: weight-for-length significantly greater for formula-fed infants between 7 and 24 mo. Triceps skinfold significantly greater in formula-fed infants at 9– 24 mo., subscapular skinfold greater at 3–18 mo. and 24 mo., flank and quadriceps skinfold greater at 9–16 and 10–13 mo. respectively. Biceps skinfold significantly greater in formula-fed infants at 13 mo., greater in breast- fed infants at 21 and 24 mo. Sum skinfolds (five sites) greater in formula-fed infants at 9–17 mo. Estimated % body fat significantly greater in formula-fed infants between 5 and 24 mo. |
| Heinig et al. ²⁰⁴ 1993, USA | As above, duration 18 mo., n = 44 breast-fed | As above | Weight-for-length Z-scores | Time of introduction of solid foods | Multiple regression model including gender, birth weight, parental body size as covariates. No relationships between energy intake, feeding mode or introduction of solid foods, and sum skinfold thicknesses at 2 y Within breast-fed group, weight-for-length Z-scores similar throughout first 18 mo. for infants who received solid food before 26 wks and those received solid food at 26+ weeks (not enough formula infants in each group for analysis) |

Table A10 Continued

Table A10 Continued Dietary papers

| Author/journal/year/country | Ages at measurements/ no. of subjects | Design/participants | Outcome (measured unless stated otherwise) | Activity risk factor/ method of measurement | Main finding |
|---|---|--|--|---|--|
| Dine <i>et al.</i> , ²⁰⁵ 1979, USA | 0, 3, 6, 12 mo., 2, 3, 4, 5 y, duration = 5 y, <i>n</i> = 620 baseline, <i>n</i> = 476 at 5 y with complete data | Prospective. Recruitment through paediatric practice. Birth weight \geq 5.5 lb. White, mid-upper social class | Weight/height, height/weight ^{1/3} , weight/height ² | Breast vs bottle feeding | Student's <i>t</i> -test used to compare breast and bottle fed groups. No difference in weight/height, ponderal index or BMI between breast and bottle-fed infants over first 5 y of life (<i>P</i> > 0.1; breast- fed <i>n</i> = 70, bottle-fed <i>n</i> = 406) |
| Kramer <i>et al.</i> , ⁷⁴ 1986, Canada | 1–3 days, 2, 4, 6 wks, 2, 3, 4 mo., 1, 2 y; duration 2 y, <i>n</i> = 462 baseline, <i>n</i> = 347 at 2 y | Prospective. Recruitment from large university teaching hospital serving socially diverse population. <i>n</i> = 553 eligible, 462 enrolled | BMI, sum triceps + subscapular + suprailliac skinfolds | Age at introduction of solids, duration of breast feeding | Multiple regression models, stepwise procedure, including sex, birth weight, parental relative weights, social class, behaviour and dietary variables as covariates. Duration of breast feeding negative predictor of BMI at 1 y (β = -0.21, <i>P</i> < 0.0001), 2 y (β = -0.11, <i>P</i> = 0.008), and of skinfolds at 2 y (β = -0.16, <i>P</i> = 0.003). Age at introduction of solids negative predictor of skinfolds at 1 y (β = -0.14, <i>P</i> = 0.009) |
| Kramer <i>et al.</i> , ⁹⁶ 1985, Canada | As above | As above | As above | As above | As above |
| Kramer <i>et al.</i> , ⁹⁷ 1985, Canada | As above | As above | As above | As above | As above |
| Marmot <i>et al.</i> , ²¹¹ 1980, UK 1946 UK birth cohort | 2 mo., 2 y, 32 y, duration 32 y, <i>n</i> = 238 baseline, <i>n</i> = 172 at 32 y | Prospective. Sample selected from UK 1946 birth cohort on basis of infant feeding and geographical area | BMI, triceps skinfold | Solely breast-fed for first 5 mo. vs not breast-fed at all for first 5 mo. (bottle-fed) | Statistical test not stated, test to compare groups used. Men: triceps skinfold greater in breast-fed group (15.0 vs 12.0 mm, <i>P</i> = 0.06), BMI greater in breast-fed group, no summary statistics given. Women: triceps greater in breast-fed group (24.1 vs 22.5 mm) but not significantly, <i>P</i> value not given |

Table A10 Continued

Table A10 Continued Dietary papers

| <i>Author/journal/year/country</i> | <i>Ages at measurements/ no. of subjects</i> | <i>Design/participants</i> | <i>Outcome (measured unless stated otherwise)</i> | <i>Activity risk factor/ method of measurement</i> | <i>Main finding</i> |
|--|---|---|--|---|--|
| O'Callaghan <i>et al.</i> , ⁴⁷ 1997, Australia MUSP (Master University Study of Pregnancy) | 0 y, 5 y, duration 5 y, <i>n</i> = 7357 baseline, <i>n</i> = 4062 at 5 y (2133 f, 1929 m) | Prospective. Mothers (<i>n</i> = 8556) enrolled at pregnancy hospital visit (ref. 20). At birth <i>n</i> = 7357, not all of these children followed up due to insufficient funds | Obesity: severe obesity—BMI > 94th percentile; moderate obesity—BMI 85–94th percentile within cohort | Duration of breast feeding: > 6 mo.; 4–6 mo.; 7 wks–3 mo.; 3–6 wks; < 2 wks; not at all | Univariate analyses: RR, 95% CIs, χ^2 test for significance. Duration of breast feeding no influence on moderate obesity. RR for severe obesity generally higher for shorter duration of breast feeding. Only breast feeding for 7 wks–3 mo. showed significantly greater RR for severe overweight (compared with breast feeding > 6 mo.), RR = 1.6, CI 1.0–2.4, <i>P</i> < 0.05. Multivariate analyses including birth weight, gender, gestational age, feeding problems and sleeplessness, adjusted ORs calculated with 95% CIs. No influence of duration of breast feeding on moderate or severe obesity |
| Poskitt, ²⁰⁶ 1977, UK | 0–1 y, 5 y; duration 5 y, <i>n</i> = 300 baseline, <i>n</i> = 203 at 5 y | Prospective. Recruitment from welfare clinics, birth weight \geq 2.5 kg | % expected relative weight (Tanner 1996 used as reference data) | Energy intake, age of weaning | No significant correlations between age of weaning or infancy energy expenditure and % expected weight at 5 y |
| Sveger, ²⁰⁷ 1978, Sweden | 0–1 y, 4 y; duration = 4 y, <i>n</i> = 243 baseline, <i>n</i> = 226 f-up | Prospective. For recruitment details refer to refs. 5, 7 | Relative weight | Overfeeding during first year, defined as \geq 2 s.d. mean daily calorie intake of infants same age and sex | 26 of 243 children overfed at \geq 1 of four measurements during first year. 24 of these 26 were followed up at 4 y, none was obese or overweight. In children not overfed during first year, 2% of 202 followed up were obese and 5% overweight. Authors conclude that overfeeding is not a predictor for overweight or obese at 4 y |

Table A10 Continued

Table A10 Continued Dietary papers

| Author/journal/year/country | Ages at measurements/ no. of subjects | Design/participants | Outcome (measured unless stated otherwise) | Activity risk factor/ method of measurement | Main finding |
|--|--|---|---|--|---|
| Vobecky et al. ²⁰⁸ 1983, Canada | 1, 2, 3, 4, 5, 6 mo, then every 3 mo. until 3y; n = 556 baseline, n = 170 selected | Prospective. Children with complete data from larger cohort (see refs. 6–9) Mostly higher social classes | Relative weight (weight for height and age), normal data from larger cohort (refs. 6–10) | Breast feeding > 2 wks vs bottle feeding | No difference in relative weight distributions between breast and bottle fed infants at 1, 2, or 3y. At 1, 2, and 3y– males: relative weight of breast fed > bottle fed; females: relative weight of bottle fed > breast fed; all differences not significant |
| Wells et al. ²⁰⁹ 1998, UK | 12 wks, 2–3.5y; duration 2– 3.5y; n = 50 baseline, n = 20 at 2–3.5y | Prospective. Subjects recruited from large maternity hospital | % fat (deuterium dilution), mean Z-score of triceps and subscapular skinfolds | Weaning status at 12 wks, milk volume intake (MVI) measured by deuterium turnover, energy intake (EI) | Student's <i>t</i> -test used to compare groups, those weaned at 12 wks had lower skinfold Z score at 2–3.5y than those unweaned, 1.20 vs –0.54, <i>P</i> < 0.05. Multiple regression analyses: after adjusting for infant skinfolds, weaned infants had lower skinfolds than unweaned, $\beta = -0.57$, <i>P</i> = 0.06. Results similar if MVI or EI added to model. MVI or EI not significant in these models. After adjusting for infant skinfolds and including weaning status and soothability, weaning not significant, soothability negatively related to skinfolds (see Behaviour section) |
| Wilson et al. ²¹⁰ 1998, UK Dundee infant feeding study | 0y, regularly until 2y, 7.2y; duration 7y, n = 674 baseline, n = 412 at 7y | Prospective. For recruitment details refer to ref. 3 | BMI (n = 412), % fat by impedance (n = 397), skinfolds at four sites (n = 405) | Exclusive breast-feeding for 15 wks vs partial breast- feeding vs exclusive bottle feeding. Solids introduced before 15 wks or 15+ wks | General linear model: after adjustment for sex, birth weight, weight at first solid feed, % body fat by impedance significantly greater in those given solids before 15 wks (18.5% vs 16.5%). Results for skinfolds similar (<i>P</i> < 0.05) BMI not significantly different in solids before or after 15 wks |

Table A10 Continued

Table A10 Continued Dietary papers

| <i>Author/journal/year/country</i> | <i>Ages at measurements/ no. of subjects</i> | <i>Design/participants</i> | <i>Outcome (measured unless stated otherwise)</i> | <i>Activity risk factor/ method of measurement</i> | <i>Main finding</i> |
|--|--|--|---|--|---|
| Zive <i>et al.</i> ¹³⁰ 1992, USA SCAN study | 4 y, <i>n</i> = 270, ~50% m, 50% f | Retrospective. Recruitment via state funded pre-schools, children's centres. Low SES families targeted for study. Anglo- and Mexican Americans. 331 respondents, complete data for 270 | BMI, sum of triceps + subscapular skinfold | Duration of breast-feeding, age of introduction to formula, age when cow's milk or formula fed more than breast milk, age solid food introduced | Correlations: no significant associations between nutrition variables and BMI or skinfolds. Multiple regression: in model which appeared to include all dietary risk factors, dietary risk factors did not contribute significantly to BMI or skinfolds |
| Childhood dietary intake (i) Childhood to adulthood Twisk <i>et al.</i> ¹⁷² 1997, The Netherlands Amsterdam Growth and Health Study (AGHS) | 13, 14, 15, 16, 21, 27, 29 y; duration 16 y; <i>n</i> = 233 baseline, <i>n</i> = 181 at 29 y (98 f, 83 m) | Prospective. Participants complete 1st and 2nd form of Amsterdam school, no refusals to participate. SES above population average | Sum of biceps, triceps, subscapular, suprailliac skinfolds | Dietary intake by cross-check dietary history for past month. Fat, protein, carbohydrate expressed as % energy intake | Multiple regression analysis: after adjusting for gender, % fat intake during adolescence (13–16 y) was negatively related to skinfolds $\beta = -0.15$, $P = 0.04$. After including variables based on significance in univariate analyses, no relationships between diet and fatness |
| Post <i>et al.</i> ²¹³ 1997, The Netherlands AGHS | 13, 14, 15, 16, 21, 27 y; duration 14 y, <i>n</i> = 233 baseline, <i>n</i> = 182 at 27 y (98 f, 84 m) | As above | Δ sum of biceps, triceps, subscapular, suprailliac skinfolds | Method as above. Animal protein, saturated fat, poly- unsaturated fat, and cholesterol expressed as % of energy intake | Longitudinal regression analysis (correction only for gender) by 'general estimating equations' showed negative relationship between change in energy intake and Δ sum of skinfolds ($P = 0.005$) and no relationship between % energy of animal protein, saturated fat, polyunsaturated fat or cholesterol and Δ sum of skinfolds |

Table A10 Continued

Table A10 Continued Dietary papers

| Author/journal/year/country | Ages at measurements/ no. of subjects | Design/participants | Outcome (measured unless stated otherwise) | Activity risk factor/ method of measurement | Main finding |
|---|--|---------------------|---|--|--|
| Twisk et al., ¹⁷³ 1997, The Netherlands AGHS | 13, 14, 15, 16, 21, 27 y; duration 14 y, n = 233 baseline, n = 181 at 27 y (98 f, 83 m) | As above | Sum of biceps, triceps, subscapular, suprailiac skinfolds | As above | Statistical model incorporating parameters calculated using generalized estimating equations. After adjusting for biological age, sex, sum of skinfolds, VO_{2max} , high risk for sum skinfolds ($\geq 20\%$ body fat for males, 30% for females), was positively related to protein intake, OR = 1.5, $P < 0.01$. No relationship between risk of skinfolds and fat or carbohydrate intake |
| van Lenthe et al., ¹⁷⁴ 1998, The Netherlands AGHS | 13, 14, 15, 16, 21, 27 y; duration 14 y, n = 233 baseline, n = 182 at 27 y (98 f, 84 m) | As above | Δ subscapular skinfold | Method as above. Fat, protein, carbohydrate expressed as % energy intake | Using 'general estimating equations' (longitudinal regression analysis), adjusting for sum skinfolds between 13 and 27 y: in males, no influence of dietary intake on Δ subscapular skinfold. In females, energy intake negatively related to Δ subscapular skinfold, $\beta = -0.25$, CI -0.31 , -0.19 , $P < 0.001$, % energy from carbohydrate positively related to Δ subscapular skinfold, $\beta = 0.09$, CI 0.02, 0.16, $P < 0.001$. Using multiple regression analysis with subscapular skinfold at 27 y as dependent variable and initially including activity, diet, smoking and alcohol intake as covariates (non-significant covariates removed by backwards elimination procedure) no influence of dietary factors on skinfold in males or females |

Table A10 Continued

Table A10 Continued Dietary papers

| <i>Author/journal/year/country</i> | <i>Ages at measurements/ no. of subjects</i> | <i>Design/participants</i> | <i>Outcome (measured unless stated otherwise)</i> | <i>Activity risk factor/ method of measurement</i> | <i>Main finding</i> |
|---|--|---|---|---|--|
| (ii) Within childhood Deheeger <i>et al.</i> ²¹⁴ 1996, France | 10 mo., 2, 4, 6, 8 y, duration 7 y; <i>n</i> = 278 baseline, <i>n</i> = 112 at 8 y | Prospective. Subjects examined in two public health centres in Paris. No further information on subject group | BMI | Energy intake by diet history | ANOVA: increase in energy intake between 4 and 6 y was significantly greater in the group who were fat (highest tertile of BMI) at 8 y compared to groups who were medium or lean (mid and lower tertile of BMI) at 8 y (<i>P</i> = 0.01). Energy intakes before 4 y or after 6 y not predictive of BMI tertile at 8 y |
| Rolland-Cachera <i>et al.</i> ²¹⁵ 1995, France | 2 y, 8 y; duration 6 y; <i>n</i> = 278 baseline, <i>n</i> = 112 at 8 y | As above | BMI, triceps skinfold, subscapular skinfold | Dietary intake by dietary history: energy intake, % energy from protein, fat, carbohydrate | Energy intake at 2 y positively correlated with BMI at 8 y <i>r</i> = 0.20, <i>P</i> = 0.049, not significantly with skinfolds. After adjusting for energy intake or BMI at 2 y, no association between % energy from fat, carbohydrate or protein and BMI or skinfolds at 8 y. After adjustment for parental BMI, % energy from protein related to BMI, <i>r</i> = 0.22, <i>P</i> = 0.03, and subscapular skinfold <i>r</i> = 0.20, <i>P</i> = 0.04, not triceps skinfold. No relationships between % energy from fat or carbohydrate and BMI or skinfolds. After adjustment for parental BMI, energy intake and BMI at 2 y, % energy from protein related to BMI <i>r</i> = 0.28, <i>P</i> = 0.008. After adjustment for SES, both energy intake and % energy from protein were positively related to BMI, <i>P</i> < 0.05 |

Table A10 Continued

Table A10 Continued Dietary papers

| Author/journal/year/country | Ages at measurements, no. of subjects | Design/participants | Outcome (measured unless stated otherwise) | Activity risk factor/ method of measurement | Main finding |
|---|--|---|---|---|--|
| Griffiths <i>et al.</i> , ²¹⁶ Lancet 1990 | 3–4 y, ~15 y; duration ~11 y; n = 37 baseline; n = 25 at 15 y (15 m, 10 f) | Prospective. Subjects selected from obese and normal weight parents | BMI, BFMI (fat mass/hgt ²) | Method for energy intake in childhood measurement not given—see refs. 3, 4 | In girls, energy intake/kg body weight at 3–4 y significantly negatively correlated to BMI and BFMI at 15 y ($r = -0.73$, $P < 0.0118$, $r = -0.77$, $P < 0.009$ respectively). No association in boys |
| Klesges <i>et al.</i> , ⁴⁵ 1995, USA | 4.4, 5.4 y, 6.4 y, duration 2 y, n = 203 baseline, n = 146 at 6.4 y | Prospective. Participants recruited through paediatricians, day-care centres, churches. Obese oversampled | Δ BMI | % kcal fat, % kcal carbohydrate measured by food frequency questionnaire | Multiple regression analysis including adjustment for baseline BMI, gender, age, parental fatness and interactions between gender and parental fatness. In model including these covariates and also activity, baseline % kcal from fat positively related to 2 y increase in BMI ($\beta = 0.03$, $P = 0.052$). Change in kcal fat <i>negatively</i> related to increase in BMI ($\beta = -0.04$, $P = 0.011$), but text does not agree with tables and suggests positive relationship |
| Maffei <i>et al.</i> , ⁴⁶ 1998, Italy | 8.6 y, 12.6 y; duration 4 y, n = 298 baseline, n = 112 at 12.6 y | Prospective. Participants recruited through private and public schools. All Caucasian | BMI. Obesity: relative BMI > 120% (Rolland-Cachera BMI tables as reference) | Energy intake, protein, carbohydrate, fat intake as % energy measured by dietary history method—dietician interviewed mothers and children, school menus reviewed | Three different types of multivariate analysis, with age, gender, dietary variables, parental BMI, TV viewing time and vigorous activity included as covariates. No significant effects of any dietary variable is any analysis |

Table A10 Continued

Table A10 Continued Dietary papers

| <i>Author/journal/year/country</i> | <i>Ages at measurements/ no. of subjects</i> | <i>Design/participants</i> | <i>Outcome (measured unless stated otherwise)</i> | <i>Activity risk factor/ method of measurement</i> | <i>Main finding</i> |
|---|--|--|--|---|--|
| Nicklas <i>et al.</i> ²¹⁷ 1988, USA | 6 mo., 1, 2, 3, 4, 7 y, duration 6.5 y, <i>n</i> = 440 baseline, <i>n</i> = 50 at 7 y with complete data | Prospective. Original sample an 18 month birth cohort from semirural, biracial population. <i>n</i> = 50 participated in all six screenings | Subscapular skinfold. Rohrer index (wgt/hgt ²) | % energy from protein, carbohydrate, fat measured by 24 h recall | Change in total protein, fat, starch and energy from 6 mo. to 4 y significantly positively correlated with change in subscapular skinfold (same period), <i>P</i> < 0.05. After adjustment for energy or body weight, relationships not significant. Using statistical test to compare groups (undefined), those with consistently high intakes compared with those with consistently lower intakes of % energy from animal fat, total fat and protein had similar Rohrer indexes at 7 y, <i>P</i> > 0.05 |
| Shapiro <i>et al.</i> ¹⁷⁹ 1984, USA | 6 mo., 1, 2, 3, 4, 6 y, 9 y; duration 8.5 y, <i>n</i> = 450 baseline, <i>n</i> = 149 at 9 y with complete data | Prospective. Sample selected from 1193 birth records filed in 1969. Parents who could be located at 6 mo. were invited to participate if going to be in area for 3 y. Initial participants 71% White, 19% Black, 10% others | Sum of triceps, subscapular, suprailiac and chest skinfolds | Energy intake assessed at each time point by 3 day dietary records, completed by parents at 6 mo. -6 y and children at 9 y | Sum energy intake over study period negatively correlated to sum skinfolds at 9 y, <i>r</i> = -0.16, <i>P</i> < 0.05 |
| Shea <i>et al.</i> ²¹⁸ 1993, USA | 3-4 y, ≥ several follow-up measures, 4-6 y; duration 25 mo., <i>n</i> = 238 baseline, <i>n</i> = 215 at 5 y with complete data | Prospective. Participants recruited through paediatric practice. Only one child per family eligible. Predominantly Hispanic, low-income district | Rate of change in BMI (kg/m ² per y) | Dietary intake by semi- quantitative questionnaire (average of three during first year) and 24 h recall (average of four in first year) | ANOVA: no significant (<i>P</i> < 0.05) differences in rate of change in BMI across quintiles of total fat density, saturated fat density or cholesterol (mg per 1000 kcal). No difference in rate of change in BMI between children consuming less than 30% or calories from total fat compared to those consuming 30% or more |

Table A11 Behavioural and psychological factors papers

| Author/journal/year/country | Age at predictor and outcome measurements/no. of subjects | Design/participants | Outcome measure | Risk factor | Main finding |
|---|--|---|--|---|--|
| i) Childhood to adulthood van Lenthe <i>et al.</i> , ²⁵³ 1998, The Netherlands Amsterdam Growth and Health Study (AGHS) | 13 y (14, 15, 16, 21 y), 27 y; duration 14 y; <i>n</i> = 233 baseline, <i>n</i> = 181 at 27 y (83 m, 98f) | Prospective. Participants complete 1st and 2nd form of Amsterdam school, no refusals to participate. SES above population average | Sum of biceps, triceps, subscapular and suprailiac skinfolds | Personality traits assessed by questionnaire (at all six time-points during study). Six traits: inadequacy, social inadequacy, dominance, rigidity, achievement motivation, debilitating anxiety | Generalized estimating equations (taking into account data from all time- points): inadequacy at 13– 16 y related to increase in sum skinfolds, $\beta = 0.08$, 95% CI 0.02, 0.14, $P < 0.01$. No relationship between inadequacy at 13–21 y or 13–27 y or any other trait at any period and sum skinfolds After controlling for BMI in childhood and gender, odds ratios for being overweight in young adulthood were 2.6 ($P = 0.02$) for mothers acceptance of sweet eating habits, 2.0 ($P = 0.04$) for providing more sweet money, 2.6 ($P = 0.04$) for mother's lacking knowledge about offspring's sweet- eating habits. After controlling for social factors, only mother's lacking knowledge remained significant, OR = 4.5, $P = 0.003$ |
| Lissau <i>et al.</i> , ¹³¹ 1993, Denmark | 9–10 y, 20–21 y; duration 10 y; <i>n</i> = 1258 at baseline, <i>n</i> = 552 at 20–21 y | Prospective. Participants a random sample, 25% of 3rd graders in Copenhagen | BMI (reported) overweight = 90th percentile BMI, obesity = 95th percentile | Variables relating to sweet eating habits and mother's knowledge of such habits | |
| ii) Within childhood Agras <i>et al.</i> , ⁹⁴ 1987, USA | 2, 4 wks, 2 y; duration 2 y, <i>n</i> = 99 baseline, <i>n</i> = 79 at 2 y | Prospective. Children recruited from hospital in first week of life | Triceps skinfold, log BMI | Sucking behaviour (measured in laboratory) — caloric intake during sucking, active feeding time, sucking pressure, interburst interval, no. feeds/day | Stepwise multiple regression models using several factors: higher sucking pressure ($P < 0.006$) and fewer feeds per day ($P < 0.01$) were associated with greater triceps skinfold at 2 y. No significant predictors for (log) BMI at 2 y. 'The more adipose infant at 1 and 2 y sucked more rapidly, at higher pressure, with longer suck and burst duration and shorter burst interval.' This feeding pattern was associated with higher caloric intake (refer to dietary section of review) |

Table A11 Continued

Table A11 Continued Behavioural and psychological factors papers

| <i>Author/journal/year/country</i> | <i>Age at predictor and outcome measurements/no. of subjects</i> | <i>Design/participants</i> | <i>Outcome measure</i> | <i>Risk factor</i> | <i>Main finding</i> |
|---|--|---|--|---|---|
| Agras <i>et al.</i> ⁷³ 1990, USA | 2 and 4 wks, 3y, 6y; duration 6y; $n=99$ baseline, $n=54$ at 6y | As above | As above | As above | Correlations: at 3y, sucking pressure related to log BMI, $r=0.33$, $P \leq 0.05$, no. feeds/day not related to log BMI. At 6y, sucking pressure, no. feeds/day unrelated to log BMI. Stepwise multiple regression models using several factors: at 3y, sucking pressure related to log BMI, partial $r^2=0.26$, $P=0.002$. At 6y, no aspect of feeding behaviour related to log BMI. No significant relationships between feeding behaviour and triceps skinfold |
| Carey <i>et al.</i> ²⁵⁴ 1988, USA | 3–7y, 8–9y; duration unspecified (1–5y); $n=?$ baseline, $n=138$ at 8–9y (67m, 71f) | Prospective. Private practice setting, predominantly middle class | Weight-for-height percentile from United States Public Health Service data | Behaviour as assessed by behaviour style questionnaire (BSQ). BSQ consists of 100 items which condense to nine categories of temperament: activity, adaptability, distractibility, intensity, mood, persistence/attention span, rhythmicity/predictability, threshold, withdrawal | Correlations: decreased adaptability related to increase in wgt-for-hgt, $r=0.18$, $P=0.02$. Increased intensity related to increase in wgt-for-hgt, $r=0.19$, $P=0.01$. Increased withdrawal related to increase in wgt-for-hgt, $r=0.24$, $P=0.002$ |
| French <i>et al.</i> ²⁵⁵ 1996, USA | 12–15y, 15–18y; duration 3y; $n \geq 1522$ baseline, $n=1278$ at 15–18y (656f, 622m) | Prospective. All 7–9th graders in suburban school district invited to participate. Of students in year 1 who remained in district for 3y, 83% females and 85% males measured at follow-up. No mention of students who moved out of district | BMI | Self-esteem: global, physical appearance, athletic, scholastic, social acceptance, job competence, romantic appeal, behavioural conduct, close friendship | Partial correlations: girls—physical appearance self-esteem at baseline negatively related to BMI at follow-up, $r=-0.11$, $P<0.05$; social acceptance self-esteem at baseline negatively related to BMI at follow-up, $r=-0.13$, $P<0.05$. Boys—no relationships between self-esteem and BMI |

Table A11 Continued

Table A11 Continued Behavioural and psychological factors papers

| Author/journal/year/country | Age at predictor and outcome measurements/ no. of subjects | Design/participants | Outcome measure | Risk factor | Main finding |
|---|--|--|---|---|--|
| Klesges et al, ²⁵⁶ 1992, USA | 3–6 y, 6–9 y; duration 3 y; <i>n</i> = 203 baseline (assumed)—information from ref. 45) <i>n</i> = 132 at 6–9 y (77 m, 55 f) | Prospective. Families participating in larger study of cardiovascular risk factors in children. White, upper-middle class. Obese children oversampled | BMI, triceps skinfold; obesity: $\geq 120\%$ ideal body fat based on triceps skinfold (normal data from US Department of Health, Education and Welfare) | Perceived Competence Scale for Children (PCPS) assessing four types of self-esteem: cognitive ability, physical ability, peer relations, general self-esteem. Family Relationship Index (FRI) assessing family function: cohesion, expressiveness, conflict, reported separately by mother and father | Multiple regression analyses. After adjusting for initial body fat and gender at 1 y follow-up, physical self-esteem ($\beta = -1.03$), father's FRI ($\beta = -0.35$), physical self-esteem * gender ($\beta = 0.65$) and father's FRI * gender ($\beta = 0.23$) were significantly related to body fat (triceps skinfold), $P < 0.05$. Thus higher physical esteem and more positive family situations as viewed by the father, were related to greater increases in body fat in girls, smaller increases in boys. At year 2, relationships were weaker, only physical self-esteem was related to gain in body fat ($\beta = -0.83$, $P = 0.036$), and at year 3, none of the psychosocial variables significantly predicted body fat based on triceps skinfold. At year 3, mother's FRI * gender significantly related to BMI, as family functioning increased, BMI increased in girls, decreased in boys |
| Kramer et al, ^{7,4,96} 1986 and 1985, Canada | 1–3 days for IBH + MFA; 2 wks for ITQ, 1 y, 2 y; duration 2 y; <i>n</i> = 462 baseline, <i>n</i> = 347 at 2 y | Prospective. Participants recruited from large university teaching hospital serving socially diverse population. <i>n</i> = 553 eligible, 462 enrolled | BMI, sum of triceps + subscapular + suprailiac skinfolds | ITQ—infant temperament questionnaire; IBH—maternal preconception of ideal infant body habitus; MFA—maternal feeding attitudes, all assessed by questionnaire | Multiple regression models, stepwise procedure, constructed for BMI and skinfolds at 1 and 2 y. Maternal preconception of infant body habitus was positively associated with BMI at 12 mo. ($\beta = 0.10$, $P < 0.045$) in model including birth weight, duration of breast feeding, and gender. No associations at 2 y. No associations between maternal feeding attitude or infant temperament and BMI or skinfolds at 1 or 2 y |

Table A11 Continued

Table A11 Continued Behavioural and psychological factors papers

| <i>Author/journal/year/country</i> | <i>Age at predictor and outcome measurements/no. of subjects</i> | <i>Design/participants</i> | <i>Outcome measure</i> | <i>Risk factor</i> | <i>Main finding</i> |
|---|--|---|---|---|--|
| Morris <i>et al.</i> ²⁵⁷ 1984, UK | 10 y, 11 y; duration 1 y; <i>n</i> = 282 at baseline, <i>n</i> = 230 at 11 y | Children recruited from 10 schools in one London area. Total eligible <i>n</i> = 408 | Δ weight–height index (based on log transformation), Δ skinfold index (based on log transformation of combined triceps and subscapular skinfolds) | Attitudes towards nutrition, knowledge of nutrition | Better knowledge of nutrition was associated with greater decrease in skinfold (– 0.6 mm, <i>P</i> < 0.05), in boys, not girls. Identifying nutritious meals was associated with greater decrease in weight for height index (– 0.3 kg, <i>P</i> < 0.05) in girls not boys. No associations between positive attitude to good nutrition, preferring or identifying fattening puddings, or preferring nutritious meals and obesity indexes |
| O’Callaghan <i>et al.</i> ⁴⁷ 1997, Australia MUSP (Mater University Study of Pregnancy) | 0 y, 5 y; duration 5 y; <i>n</i> = 7357 at baseline, <i>n</i> = 4062 at 5 y (2133 f, 1929 m) | Prospective. Mothers (<i>n</i> = 8556) enrolled at pregnancy hospital visit (ref. 20). At birth <i>n</i> = 7357, not all of these children followed up due to insufficient funds | BMI, severe obesity: BMI > 94th percentile; moderate obesity: BMI 85–94th percentile within cohort | Feeding problems (undefined), sleeplessness | Univariate analyses: RR, 95% CIs. Feeding problems—no influence on moderate or severe obesity. Sleeplessness—almost significant influence of sleeplessness few times/week (not more or less) on moderate obesity only. RR = 1.2, CI 0.9–1.7. Multivariate analyses including several factors, adjusted ORs calculated with 95% CIs. Feeding problems—protective effect on severe obesity only, OR = 0.3, CI 0.1–0.8. Sleeplessness—significant influence of sleeplessness few times/week (not more or less) on moderate obesity only. OR = 1.7, CI 1.1–2.6 |

Table A11 Continued

Table A11 Continued Behavioural and psychological factors papers

| <i>Author /journal/year/country</i> | <i>Age at predictor and outcome measurements/no. of subjects</i> | <i>Design /participants</i> | <i>Outcome measure</i> | <i>Risk factor</i> | <i>Main finding</i> |
|--|---|---|---|--|---|
| Wells <i>et al.</i> ²⁴⁶ 1997, UK | 12 wks, 2–3.5 y; duration 2–3.5 y; baseline $n = 50$, f-up $n = 25–30$ (depending on outcome) | Prospective. Subjects recruited from large maternity hospital | Sum of triceps + subscapular skinfolds; % body fat by TBW method | Infant temperament by questionnaire to mother; fussiness; crying; distress to limitations; soothability | Correlation: infant distress to limitations was positively correlated to % body fat at 2– 3.5 y ($r = 0.4$, $P < 0.05$) but no association with skinfolds. Infant soothability was negatively correlated to sum of skinfolds ($r = -0.66$, $P < 0.001$), but no association with % body fat. Multiple regression: after adjustment for infant % fat and childhood age, infant distress to limitations was positively associated with childhood % body fat ($\beta = 1.96$, $P = 0.059$, borderline significance). After adjustment for infant sum of skinfolds and 'awake and active' in childhood, infant soothability was negatively associated with childhood sum of skinfolds ($\beta = -1.65$, $P = 0.012$) |
| Wells <i>et al.</i> ²⁰⁹ 1998, UK | 12 wks, 2–3.5 y; duration 2–3.5 y; $n = 50$ baseline, $n = 20$ at 2–3.5 y | As above | % fat (deuterium dilution); mean Z-score of triceps and subscapular skinfolds | Soothability | Using multiple regression analyses: after adjusting for infant skinfolds and including weaning status and soothability, weaning not significant, soothability negatively related to skinfolds $\beta = -0.41$, $P = 0.01$ |

Appendix II

Search strategy: Medline (Ovid software interface),

1966–April 1998

1. exp Child/
2. exp child nutrition/
3. obes\$.tw.
4. adipos\$.tw.
5. ponderos\$.tw.
6. bodyfat\$.tw.
7. body fat\$.tw.
8. (fat adj3 mass).tw.
9. fatness\$.tw.
10. subcutaneous fat.tw.
11. body mass.tw.
12. body masses.tw.
13. bmi.tw.
14. underweight.tw.
15. overweight.tw.
16. body size\$.tw.
17. body composition.tw.
18. quetelet.tw.
19. ponderal.tw.
20. anthropomet\$.tw.
21. skinfold\$.tw.
22. skin fold\$.tw.
23. lean\$.tw.
24. thinness.tw.
25. waist\$.tw.
26. exp obesity/
27. exp skinfold thickness/
28. exp body mass index/
29. exp body weight/
30. exp body weight changes/
31. exp body composition/
32. prospectiv\$.tw.
33. longitudinal\$.tw.
34. followup\$.tw.
35. (follow adj up\$).tw.
36. long term.tw.
37. longterm.tw.
38. track\$.tw.
39. serial\$.tw.
40. cohort\$.tw.
41. reexamin\$.tw.
42. re examin\$.tw.
43. reassess\$.tw.
44. re assess\$.tw.
45. restud\$.tw.
46. re stud\$.tw.
47. reevaluat\$.tw.
48. re evaluat\$.tw.
49. reinvestigat\$.tw.
50. re investigat\$.tw.
51. rereview\$.tw.
52. re review\$.tw.
53. remeasure\$.tw.
54. re measure\$.tw.
55. retrospectiv\$.tw.

56. growth study.tw.
57. case control\$.tw.
58. case?control\$.tw.
59. exp case control studies/
60. exp cohort studies/
61. epidemiologic methods/
62. 1 or 2
63. or/3-31
64. or/57-59
65. or/26-31
66. 64 and 65
67. or/32-56,60-61,66
68. 62 and 63 and 67
69. Amsterdam Growth.tw,in.
70. atherosclerosis risk in communities.tw.
71. baltimore longitudinal study.tw.
72. belgian luxembourg child.tw.
73. birth cohort.tw.
74. bogalusa heart study.tw.
75. california child health.tw.
76. cardia study.tw.
77. cardiovascular health study.tw,in.
78. (christchurch adj3 development).tw,in.
79. darling.tw.
80. dormont high.tw.
81. dunedin multidisciplinary.tw,in.
82. dunedin multi disciplinary.tw,in.
83. fels.tw.
84. framingham.tw.
85. hhanes\$.tw.
86. kabi pharmacia.tw.
87. leuven growth.tw.
88. minneapolis children\$ blood.tw.
89. muscatine.tw.
90. (national health adj3 examination adj3 survey\$).tw,in.
91. nhanes\$.tw.
92. nhlbi.tw.
93. nurses health study.tw.
94. princeton school district.tw.
95. procam.tw.
96. stanford five city.tw.
97. tecumseh community.tw.
98. Wroclaw growth study.tw,in.
99. Wroclaw longitudinal twin study.tw,in.
100. young finns.tw.
101. youth health survey.tw,in.
102. zurich growth.tw.
103. or/69-102
104. 103 and 63
105. 68 or 104

Search strategy: Embase (Ovid software interface),

1980–May 1998

1. child/
2. exp child/
3. exp adolescent/
4. exp newborn/
5. exp adolescence/

6. exp childhood/
 7. exp child nutrition/
 8. obes\$.tw.
 9. adipos\$.tw.
 10. ponderos\$.tw.
 11. bodyfat\$.tw.
 12. body fat\$.tw.
 13. (fat adj3 mass).tw.
 14. fatness\$.tw.
 15. subcutaneous fat.tw.
 16. body mass.tw.
 17. body masses.tw.
 18. bmi.tw.
 19. underweight.tw.
 20. overweight.tw.
 21. body size\$.tw.
 22. body composition.tw.
 23. quetelet.tw.
 24. ponderal.tw.
 25. anthropomet\$.tw.
 26. skinfold\$.tw.
 27. skin fold\$.tw.
 28. lean\$.tw.
 29. thinness.tw.
 30. waist\$.tw.
 31. exp obesity/
 32. exp skinfold thickness/
 33. exp body mass/
 34. exp body size/
 35. exp body weight/
 36. exp body composition/
 37. body fat/
 38. subcutaneous fat/
 39. prospectiv\$.tw.
 40. longitudinal\$.tw.
 41. followup\$.tw.
 42. (follow adj up\$).tw.
 43. long term.tw.
 44. longterm.tw.
 45. track\$.tw.
 46. serial\$.tw.
 47. cohort\$.tw.
 48. reexamin\$.tw.
 49. re examin\$.tw.
 50. reassess.tw.
 51. re assess.tw.
 52. restud\$.tw.
 53. re stud\$.tw.
 54. reevaluat\$.tw.
 55. re evaluat\$.tw.
 56. reinvestigat\$.tw.
 57. re investigat\$.tw.
 58. rereview\$.tw.
 59. re review\$.tw.
 60. remeasure\$.tw.
 61. re measure\$.tw.
 62. retrospectiv\$.tw.
 63. growth study.tw.
 64. exp cohort analysis/
 65. exp clinical study/
 66. exp epidemiology/
 67. case control\$.tw.
 68. case?control\$.tw.
 69. or/1-7
 70. or/8-38
 71. or/39-68
 72. 69 and 70 and 71
 73. Amsterdam Growth.tw,in.
 74. baltimore longitudinal study.tw.
 75. belgian luxembourg child.tw.
 76. birth cohort.tw.
 77. bogalusa heart study.tw.
 78. california child health.tw.
 79. cardia study.tw.
 80. (christchurch adj3 development).tw,in.
 81. darling.tw.
 82. dormont high.tw.
 83. dunedin multidisciplinary.tw,in.
 84. dunedin multi disciplinary.tw,in.
 85. fels.tw.
 86. framingham.tw.
 87. hhanes\$.tw.
 88. kabi pharmacia.tw.
 89. leuven growth.tw.
 90. minneapolis children\$ blood.tw.
 91. muscatine.tw.
 92. (national health adj3 examination adj3 survey\$).tw,in.
 93. nhanes\$.tw.
 94. nhlbi.tw.
 95. nurses health study.tw.
 96. princeton school district.tw.
 97. procam.tw.
 98. stanford five city.tw.
 99. tecumseh community.tw.
 100. young finns.tw.
 101. zurich growth.tw.
 102. atherosclerosis risk in communities.tw.
 103. cardiovascular health study.tw,in.
 104. youth health survey.tw,in.
 105. Wroclaw growth study.tw,in.
 106. Wroclaw longitudinal twin study.tw,in.
 107. or/73-106
 108. 107 and 70
 109. 72 or 108
- Search strategy: CAB Abstracts (Ovid software interface), 1973–May 1998**
1. exp adolescents/ or exp children/ or exp daughters/ or exp infants/ or exp sons/
 2. obes\$.ab,do,ti.
 3. adipos\$.ab,do,ti.
 4. ponderos\$.ab,do,ti.
 5. bodyfat\$.ab,do,ti.
 6. body fat\$.ab,do,ti.
 7. (fat adj3 mass).ab,do,ti.
 8. fatness\$.ab,do,ti.
 9. subcutaneous fat.ab,do,ti.
 10. body mass.ab,do,ti.

11. body masses.ab,do,ti.
12. bmi.ab,do,ti.
13. underweight.ab,do,ti.
14. overweight.ab,do,ti.
15. body size\$.ab,do,ti.
16. body composition.ab,do,ti.
17. quetelet.ab,do,ti.
18. ponderal.ab,do,ti.
19. anthropomet\$.ab,do,ti.
20. skinfold\$.ab,do,ti.
21. skin fold\$.ab,do,ti.
22. lean\$.ab,do,ti.
23. thinness.ab,do,ti.
24. waist\$.ab,do,ti.
25. weight?for?height.ab,do,ti.
26. weight?height.ab,do,ti.
27. exp body fat/ or exp obesity/ or exp overeating/
or exp overfeeding/ or exp overweight/ or exp
thinness/ or exp weight reduction/
28. exp body density/ or exp body weight/
29. exp body composition/
30. prospectiv\$.ab,do,ti.
31. longitudinal\$.ab,do,ti.
32. followup\$.ab,do,ti.
33. (follow adj up\$).ab,do,ti.
34. long term.ab,do,ti.
35. longterm.ab,do,ti.
36. track\$.ab,do,ti.
37. serial\$.ab,do,ti.
38. cohort\$.ab,do,ti.
39. reexamin\$.ab,do,ti.
40. re examin\$.ab,do,ti.
41. reassess\$.ab,do,ti.
42. re assess\$.ab,do,ti.
43. restud\$.ab,do,ti.
44. re stud\$.ab,do,ti.
45. reevaluat\$.ab,do,ti.
46. re evaluat\$.ab,do,ti.
47. reinvestigat\$.ab,do,ti.
48. re investigat\$.ab,do,ti.
49. rereview\$.ab,do,ti.
50. re review\$.ab,do,ti.
51. remeasur\$.ab,do,ti.
52. re measur\$.ab,do,ti.
53. retrospectiv\$.ab,do,ti.
54. growth study.ab,ti,do.
55. exp aetiology/ or exp disease surveys/ or exp
epidemiological surveys/ or epidemiology/
case control\$.ab,do,ti.
56. 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9 or 10 or 11 or
12 or 13 or 14 or 15 or 16 or 17 or 18 or 19 or 20
or 21 or 22 or 23 or 24 or 26 or 27 or 28 or 29
58. 30 or 31 or 32 or 33 or 34 or 35 or 36 or 37 or 38
or 39 or 40 or 41 or 42 or 43 or 44 or 45 or 46 or
47 or 48 or 50 or 51 or 52 or 53 or 54 or 55 or 56
59. 1 and 57 and 58

Search strategy: PsycLIT (Silverplatter software interface), 1966–March 1998

1. explode “Children”
2. “Childhood-” in DE
3. “Adolescence-” in DE
4. explode “Adolescents”
5. explode “College-Students”
6. explode “Infants”
7. “Preadolescents-” in DE
8. explode “Students”
9. explode “High-School-Students”
10. explode “College-Students”
11. explode “Preschool-Students”
12. explode “Elementary-School-Students”
13. obes*
14. adipos*
15. ponderos*
16. bodyfat*
17. body fat*
18. (fat near3 mass)
19. fatness*
20. subcutaneous fat
21. body mass
22. body masses
23. bmi
24. underweight
25. under weight
26. overweight
27. over weight
28. body size*
29. body composition
30. quetelet
31. ponderal
32. anthropomet*
33. skinfold*
34. skin fold*
35. lean*
36. thinness
37. waist*
38. weight?for?height
39. weight?height
40. explode “Body-Weight”
41. “Weight-Control” in DE
42. prospectiv*
43. longitudinal*
44. followup*
45. follow up*
46. long term*
47. longterm*
48. track*
49. serial*
50. cohort*
51. reexamin*
52. re examin*
53. reassess*
54. re assess*
55. restud*
56. re stud*
57. reevaluat*
58. re evaluat*

59. reinvestigat*
 60. re investigat*
 61. rereview*
 62. re review*
 63. remeasure*
 64. re measure*
 65. retrospectiv*
 66. growth study
 67. "Causal-Analysis" in DE
 68. explode "Between-Groups-Design"
 69. explode "Cohort-Analysis"
 70. explode "Followup-Studies"
 71. explode "Longitudinal-Studies"
 72. explode "Repeated-Measures"
 73. "Etiology-" in DE
 74. case control*
 75. casecontrol*
 76. #1 or #2 or #3 or #4 or #5 or #6 or #7 or #8 or #9 or #10 or #11 or #12
 77. #13 or #14 or #15 or #16 or #17 or #18 or #19 or #20 or #21 or #22 or #23 or #24 or #25 or #26 or #27 or #28 or #29 or #30 or #31 or #32 or #33 or #34 or #35 or #36 or #37 or #38 or #39 or #40 or #41
 78. #42 or #43 or #44 or #45 or #46 or #47 or #48 or #49 or #50 or #51 or #52 or #53 or #54 or #55 or #56 or #57 or #58 or #59 or #60 or #61 or #62 or #63 or #64 or #65 or #66 or #67 or #68 or #69 or #70 or #71 or #72 or #73 or #74 or #75
 79. #76 and #77 and #78
 80. amsterdam growth
 81. (atherosclerosis risk) near2 (communities)
 82. baltimore longitudinal study
 83. belgian luxembourg child
 84. birth cohort
 85. bogalusa heart study
 86. california child health study
 87. christchurch near3 development
 88. darling in ti,ab,su
 89. dormont high
 90. dunedin multi disciplinary
 91. dunedin multidisciplinary
 92. fels
 93. framingham
 94. hhanes
 95. kabi pharmacia
 96. leuven growth
 97. minneapolis children* blood
 98. muscatine
 99. (national health) near3 examination near3 survey
 100. nhanes
 101. nhlbi
 102. nurses health study
 103. princeton school district
 104. procam
 105. stanford five city
 106. tecumseh community
 107. Wrowclaw growth study
 108. Wroclaw longitudinal twin study
 109. young finns
 110. youth health survey
 111. zurich growth
 112. #80 or #81 or #82 or #83 or #84 or #85 or #86 or #87 or #88 or #89 or #90 or #91 or #92 or #93 or #94 or #95 or #96 or #97 or #98 or #99 or #100 or #101 or #102 or #103 or #104 or #105 or #106 or #107 or #108 or #109 or #110 or #111 or #112
 113. #112 and #78
 114. #113 or #80
- Search strategy: Sport Discus (Silverplatter software interface), 1975–March 1998**
1. explode "CHILD"
 2. explode "INFANT"
 3. explode "ADOLESCENT"
 4. OBES* in TI, AB
 5. ADIPOS* in TI,AB
 6. PONDEROS* in TI,AB
 7. BODYFAT* in TI,AB
 8. BODY FAT* IN TI,AB
 9. (FAT near3 MASS) in TI,AB
 10. FATNESS* in TI,AB
 11. SUBCUTANEOUS FAT IN TI,AB
 12. BODY MASS IN TI,AB
 13. BODY MASSES IN TI,AB
 14. BMI in TI,AB
 15. UNDERWEIGHT in TI,AB
 16. UNDER WEIGHT IN TI,AB
 17. OVERWEIGHT in TI,AB
 18. OVER WEIGHT IN TI,AB
 19. BODY SIZE* IN TI,AB
 20. BODY COMPOSITION IN TI,AB
 21. QUETELET in TI,AB
 22. PONDERAL in TI,AB
 23. ANTHROPOMET* in TI,AB
 24. SKINFOLD* in TI,AB
 25. SKIN FOLD* IN TI,AB
 26. LEAN* in TI,AB]
 27. THINNESS in TI,AB
 28. WAIST* in TI,AB
 29. "OBESITY-" IN DE
 30. "SKINFOLD-THICKNESS" IN DE
 31. explode "BODY-WEIGHT"
 32. explode "BODY-COMPOSITION"
 33. explode "ANTHROPOMETRY"
 34. PROSPECTIV* in TI,AB
 35. LONGITUDINAL* in TI,AB
 36. FOLLOWUP* in TI,AB
 37. (FOLLOW* near1 UP*) in TI,AB
 38. LONG TERM IN TI,AB
 39. LONGTERM in TI,AB
 40. TRACK* in TI,AB
 41. SERIAL* in TI,AB
 42. COHORT* in TI,AB
 43. RE?EXAMIN* in TI,AB
 44. RE?ASSESS in TI,AB
 45. RE?STUD in TI,AB
 46. RE?EVALUAT* in TI,AB

47. RE?INVESTIGAT* in TI,AB
48. RE?REVIEW* in TI,AB
49. RE?MEASURE* in TI,AB
50. RETROSPECTIV* in TI,AB
51. GROWTH STUDY IN TI,AB
52. CASE CONTROL* IN TI,AB
53. CASE?CONTROL* in TI,AB
54. explode "CASE-STUDY"
55. explode "LONGITUDINAL-STUDY"
56. explode "PREVENTIVE-MEDICINE"
57. "EPIDEMIOLOGY-" IN DE
58. "COMPARATIVE-STUDY" IN DE
59. explode "PROSPECTIVE-STUDY"
60. explode "RETROSPECTIVE-STUDY"
61. "TREND-ANALYSIS" IN DE
62. #1 or #2 or #3
63. #4 or #5 or #6 or #7 or #8 or #9 or #10 or #11 or #12 or #13 or #14 or #15 or #16 or #17 or #18 or #19 or #20 or #21 or #22 or #23 or #24 or #25 or #26 or #27 or #28 or #29 or #30 or #31 or #32 or #33
64. #34 or #35 or #36 or #37 or #38 or #39 or #40 or #41 or #42 or #43 or #44 or #45 or #46 or #47 or #48 or #49 or #50 or #51 or #52 or #53 or #54 or #55 or #56 or #57 or #58 or #59 or #60 or #61
65. #62 and #63 and #64
66. AMSTERDAM GROWTH
67. ATHROSCLEROSIS RISK NEAR2 COMMUNITIES
68. BALTIMORE LONGITUDINAL
69. BELGIAN LUXEMBOURG CHILD
70. BIRTH COHORT
71. BOGALUSA HEART STUDY
72. CALIFORNIA CHILD HEALTH
73. CARDIA STUDY
74. CARDIOVASCULAR HEALTH STUDY
75. CHRISTCHURCH near3 DEVELOPMENT
76. DARLING STUDY
77. DORMONT HIGH
78. DUNEDIN MULTIDISCIPLINARY
79. DUNEDIN MULTI DISCIPLINARY
80. FELS
81. FRAMINGHAM
82. HHANES
83. KABI PHARMACIA
84. LEUVEN GROWTH
85. MINNEAPOLIS CHILDREN* BLOOD
86. MUSCATINE
87. NATIONAL HEALTH NEAR3 EXAMINATION NEAR3 SURVEY*
88. NHANES
89. NHLBI
90. NURSES HEALTH STUDY
91. PRINCETON SCHOOL DISTRICT
92. PROCAM
93. STANFORD FIVE CITY
94. TECUMSEH
95. WROCLAW GROWTH STUDY
96. WROCLAW LONGITUDINAL TWIN STUDY
97. YOUNG FINNS

98. YOUTH HEALTH SURVEY
99. ZURICH GROWTH
100. #66 or #67 or #68 or #69 or #70 or #71 or #72 or #73 or #74 or #75 or #76 or #77 or #78 or #79 or #80 or #81 or #82 or #83 or #84 or #85 or #86 or #87 or #88 or #89 or #90 or #91 or #92 or #93 or #94 or #95 or #96 or #97 or #98 or #99
101. #100 and #63
102. #65 or #101

Appendix III. Collaborators, steering group and experts consulted

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