Symposium on ‘Prevention of obesity’

Physical activity and obesity prevention: a review of the current evidence

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Ecological data on temporal trends suggest that the rising prevalence of obesity is, at least in part, attributable to declining population energy expenditure. However, population-level data on trends in physical activity are scarce. In longitudinal cohort studies individuals who report higher levels of leisure-time physical activity tend to be less likely to gain weight, but studies vary in their conclusions because of issues of confounding, reverse causality and measurement error. The majority of studies suggest that low levels of activity are only weakly associated with future weight gain. Questions about dose–response can only be properly addressed by studies including objective measures of activity with known measurement error. The observational studies leave uncertainties about the direction of causality, as individuals who are overweight are less likely to stay active. Adjustment for confounding can diminish the impact of known confounders, but only randomisation can deal with issues of unmeasured confounding. Although there are a large number of clinical trials on the treatment of individuals with obesity or the prevention of weight regain among weight losers, the updated review of trials to prevent weight gain de novo only reveals six trials published since 2000 in adults and eleven in children. Not only are these trials relatively few in number but, for various methodological reasons, they are uncertain in their conclusions about whether increasing activity will be effective in preventing obesity. Whilst efforts should continue to enhance the evidence base it is wise, in the meantime, to stick to the consensus public health advice of advocating 45–60 min moderate intensity activity daily to prevent obesity.

Physical activity: Weight gain: Critical review: Obesity

Three features characterise the current epidemic of obesity. Its rapidity is striking, as is the magnitude of the increase in prevalence. It is also a global phenomenon, as similar changes are seen in all Western countries and many non-Western countries as well. In the USA, for example, the prevalence of obesity increased by 74% between 1991 and 2001 (Mokdad et al. 2003). In the UK the proportionate increase between 1993 and 2003 was 43% for women and 74% for men (Department of Health, 2004b). The rising prevalence of obesity has been attributed in part to population-level changes in physical activity. The present paper aims to review the strength of evidence underlying the assertion that there is a secular decline in physical activity levels and that low levels of physical activity are associated with weight gain in observational studies. Finally, evidence of the effectiveness of interventions aimed at the prevention of obesity is discussed.

Ecological data

The causes of these rapid population shifts are uncertain, but data suggest that this transition has occurred simultaneously with changes in physical activity patterns (Department of...
Health, 2004b; Ham et al. 2004). This evidence is ecological, and as such does not provide as high a level of causal inference as individual-level data. The evidence is also rather limited. One of the main reasons is that physical activity, in contrast to obesity, is not simple to assess because it is a complex multi-dimensional behaviour (Wareham & Rennie, 1998). Physical activity takes place in a variety of different domains, i.e. in transportation, domestic life, occupation and recreation. Each domain probably needs to be assessed separately, not only because this approach allows the information to be more specific, but also because it is more likely to be valid. In addition to the different domains, physical activity assessment needs to consider intensity, frequency, duration and the type of activity undertaken. Many historical physical activity instruments, however, are rather simple and often reduce this complex behaviour to a global self-report index (Rennie & Wareham, 1998). Even those questionnaires that ask about specific activities sometimes group them across domains, creating questions that are difficult to answer. How many individuals can accurately report how far they walk each day? It is more likely that self-report will be accurate when questions address activities in the domain in which they take place. Thus, questions might separately address walking to and from work or school, walking at work and walking for pleasure. Even when questionnaires are logically constructed with attention to the different domains of activity, they are still relatively imprecise as a measure of total energy expenditure (Wareham et al. 2002). It may be that the inherent limitations of self-report measures of activity for population surveillance of energy expenditure are so great that alternative strategies, including the use of objective monitoring, are required.

In the UK population-level surveillance data on overall physical activity are extremely sparse. The Health Survey for England (Department of Health, 2004b) included questions originally developed for the Allied Dunbar National Fitness Survey (Sports Council and Health Education Authority, 1992), which have been reconfigured to allow comparison with contemporary definitions of desirable activity levels. As yet, insufficient time has elapsed for temporal trends to become apparent, but the current Health Survey for England data (Department of Health, 2004b) suggest that between 1997 and 2003 an increasing number of individuals achieved a physical activity target of a minimum of ≥30 min moderate-intensity activity on a minimum of 5 d/week, which is perhaps a paradox given the rising prevalence of obesity. There is no published validation study demonstrating the accuracy of these questions in assessing true activity levels. Thus, UK information on population levels of physical activity mostly stems from proxy domain-specific measures. For example, since the 1960s there has been a large increase in second car ownership (Department for Transport, 2004), an increase in the use of labour-saving devices in the house (e.g. dishwashers, tumble driers; Rickards et al. 2004), an increase in the time spent viewing television (TV) per week (Rickards et al. 2004), a decline in the distance children walk per year (Department for Transport, 2004) and a massive change in the proportion of the workforce employed in manufacturing, farming and other physically-demanding occupations. Given that the aetiological effects of activity are likely to be related to the totality of physical activity rather than the domain-specific components, it is a major deficiency that there are no population-level data on temporal trends in total activity. It could be that the apparent temporal changes in activity in domestic life, work and travel are compensated for by an increase in recreational activity. However, this information is not available for the UK. For the USA there is some suggestion from the Behaviour Risk Factor Surveillance System of a secular decrease in the proportion of the population that report total inactivity during recreational time (Ham et al. 2004). Whether this finding can be taken to imply an increase in sufficient physical activity to prevent weight gain is uncertain.

In summary, the available data seem to indicate a secular decline in overall physical activity that has occurred at the same time, or possibly before, the temporal increase in obesity. In order to have a clearer knowledge of temporal trends of physical activity, it is essential that the UK and other countries establish consistent security-funded collections of long-term population surveillance data both on sub-components of physical activity and total physical activity-related energy expenditure (PAEE). As the latter is difficult to assess by questionnaire, it would be preferable to use objective methods. This proposition requires a long-term commitment to surveillance, which in turn implies investment in the development and use of inexpensive surveillance methods. Population-level surveillance data not only provides important descriptive epidemiological information about temporal trends, but it also acts as an outcome measure for interventions aimed at changing population activity, a theme that will discussed later in the present review.

**Observational studies of physical activity and change in weight**

The contribution made by population-level data to the determination of the contribution that activity or inactivity makes to the causation of weight gain will always be limited by ecological fallacy. A higher level of causal inference comes from studies in which activity and weight change are measured for individuals rather than populations. The observational data relating activity and weight change have previously been systematically reviewed in 2000 both in adults and children (Fogelholm & Kukkonen-Harjula, 2000; Molnar & Livingstone, 2000).

The purpose of the present review is to update the two previous reviews by summarising the literature published since 2000 on observational studies studying the longitudinal association between physical activity and weight change in adults and children. For this purpose the databases Pubmed and Psyclit were searched using text words aimed at retrieving longitudinal or prospective cohort studies that include data on baseline physical activity or change in physical activity, and use change in body composition as an outcome measure, excluding cross-sectional studies. This search strategy has revealed...
a total of thirty additional papers, fourteen in adults and sixteen in children.

Cohort studies in adults

Fogelholm & Kukkonen-Harjula (2000) have systematically reviewed the literature describing data from observational cohort studies on physical activity and weight gain in adults, concluding that there is inconsistent evidence of the predictive effect of baseline physical activity on subsequent weight gain. However, they observed that the association between weight gain and change in activity or activity at follow-up is stronger, although still modest. These results may be interpreted in three different ways: (a) physical activity is an important factor in preventing weight gain, but the true association is not detectable because of measurement error; (b) less weight gain leads to better exercise adherence; a reverse causality argument; (c) the self-reported physical activity may be a proxy for a general healthy lifestyle; a confounding argument. In the current review an additional fourteen observational studies on physical activity and weight gain in adults have been identified, of which only two have included an objective measure to assess physical activity (Tataranni et al. 2003; Ekelund et al. 2005), whereas the other twelve studies have assessed physical activity by means of self-report (Rainwater et al. 2000; Schmitz et al. 2000; Sherwood et al. 2000; Bell et al. 2001; Wagner et al. 2001; Ball et al. 2002; Hu et al. 2003; Koh-Banerjee et al. 2003; Macdonald et al. 2003; Droyvold et al. 2004; Petersen et al. 2004; Wenche et al. 2004).

Self-report physical activity and weight gain: studies in adults

Table 1 provides a summary of the twelve studies reporting on the association between self-reported physical activity and weight gain. Most studies include a large number of participants (sample size ranging from 539 to 50,277) and have relatively long follow-up (ranging from 3 to 11 years). The age of the participants ranges from 18 to 78 years at inclusion and only five studies include both males and females (Rainwater et al. 2000; Schmitz et al. 2000; Sherwood et al. 2000; Bell et al. 2001; Petersen et al. 2004). Most studies report on longitudinal associations between physical activity and BMI or body weight. Only two of the included studies do not find an association (Rainwater et al. 2000; Ball et al. 2002), whereas nine report a negative association between physical activity and weight gain, i.e. that lower physical activity predicts higher subsequent weight gain (Schmitz et al. 2000; Sherwood et al. 2000; Bell et al. 2001; Wagner et al. 2001; Hu et al. 2003; Koh-Banerjee et al. 2003; Macdonald et al. 2003; Droyvold et al. 2004; Wenche et al. 2004). One study reports a reverse association, suggesting that higher baseline levels of BMI predict lower levels of physical activity (Petersen et al. 2004). Comparing these results with the previous review (Fogelholm & Kukkonen-Harjula, 2000) it seems that the more-recent studies more often report associations in the expected direction. This observation could have several possible explanations. First, the more-recent studies could be larger and, therefore, could have the power to detect small associations. This explanation certainly seems to be a possibility as five of the sixteen studies reported by Fogelholm & Kukkonen-Harjula (2000) include <500 participants, whereas the twelve more-recent papers all had >500 participants. Alternatively, improvements could have been made to study design, reducing the effects of confounding by adjustment for a greater range of factors or limiting measurement error by using more-valid self-report measures of physical activity. However, neither of these explanations is very likely because the confounding factors considered are similar in all studies, as are the measures of activity. Finally, it is possible that the predominance of recent studies in the expected direction could be a manifestation of publication bias. Given the plausibility of an association between inactivity and weight gain, and the widespread public view that lack of activity is driving the current obesity epidemic, publication bias is certainly possible. Few authors are likely to challenge current orthodoxies.

One group who have attempted to consider alternative explanations are Petersen et al. (2004): their longitudinal Copenhagen Study is well placed to investigate relationships between activity and weight as they have repeated measures over time. Their data suggest that physical activity at baseline is not related to weight gain, but that the converse is true, as a higher BMI at baseline is related to an increased risk of later physical inactivity. This study raises important questions about reverse causality. However, it is difficult to determine the direction of causality for this type of data because of the marked difference in the measurement precision of physical activity and obesity. When the more imprecise variable is used as the outcome, the measure of effect is estimated accurately, but with error. When it is used as the exposure, the measure of effect is attenuated towards the null. Since activity measurement is much less precise than quantification of obesity, it is not surprising that baseline weight predicts follow-up activity, whereas the reverse is not demonstrable because of measurement error. Longitudinal studies that use more accurate measurement of both activity and weight change might be able to more accurately estimate the true relationship between changes in these measures. However, ultimately there remains a ‘chicken and egg’ argument, which may not be resolvable using observational data.

Although all studies adjust for other lifestyle factors that are associated with activity and may be confounders in the relationship with weight gain, it is not possible to remove the effect of confounding, which can take two forms. Residual confounding can exist when the measurement of a confounding factor is less than perfect, so that adjustment does not fully remove the effect of the confounder. Unmeasured confounding is an issue for factors that have not been thought about at all and are therefore not adjusted for. Given the diversity and complexity of the potential confounding factors for the association between activity and weight gain, it is highly likely that both forms of confounding exist. No observational study can ever resolve issues of confounding. An attempt can be made to reduce residual confounding by measuring plausible factors as
<table>
<thead>
<tr>
<th>Study</th>
<th>n</th>
<th>Baseline age, gender</th>
<th>Selection</th>
<th>Method</th>
<th>PA assessment</th>
<th>Outcome</th>
<th>Size of effect</th>
<th>Confounding</th>
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<tr>
<td>Hu et al. (2003)</td>
<td>50277</td>
<td>46–71 years, female</td>
<td>Healthy, BMI &lt;30 kg/m² in previous 16 years (excluding cancer, CVD and diabetes), USA</td>
<td>Prospective cohort, 6 years follow-up (Nurse’s Health Study)</td>
<td>Self-report of PA and sedentary behaviour</td>
<td>Onset of obesity (BMI &gt;30 kg/m²)</td>
<td>2 h/d increase in TV viewing associated with a 23 (95% CI 17, 30)% increase in obesity; each 2 h/d increase in sitting at work associated with a 5 (95% CI 0, 10) % increase in obesity; standing or walking around at home (2 h/d) associated with a 9 (95% CI 6, 12) % reduction in obesity; 1 h brisk walking/d associated with a 24 (95% CI 19, 29) % reduction in obesity</td>
<td>Age, smoking, alcohol consumption, EI, total fat, glycaemic load, cereal fibre</td>
</tr>
<tr>
<td>Koh-Banerjee et al. (2003)</td>
<td>16567</td>
<td>40–75 years, male</td>
<td>Healthy (excluding CVD, cancer, diabetes), USA</td>
<td>Longitudinal, 9 years follow-up (Health professionals’ study)</td>
<td>Self-report, leisure time, last year (biennially)</td>
<td>ΔWC</td>
<td>Increase in vigorous PA (by 25 MET-h/week; approx 4 h vigorous activity) associated with a reduction in waist of 1.9 (95% CI -3.5, -0.3) mm Increase in TV viewing by 20 h/week associated with an increase in waist of 3.0 (95% CI 0.6, 5.4) mm</td>
<td>Age, BMI, WC, total energy, alcohol consumption, total PA and change in smoking and in BMI</td>
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<tr>
<td>Macdonald et al. (2003)</td>
<td>898</td>
<td>45–54 years, female</td>
<td>Random sample from an osteoporosis screening programme, around menopause, no HRT, UK</td>
<td>Prospective 6.3 years follow-up</td>
<td>Self-report time in rest, light, moderate and vigorous intensity, PAL calculated</td>
<td>ΔBW</td>
<td>Change in PAL influenced change in BW explaining 4.4%. Over time, PAL decreased with increasing wt gain (P &lt; 0.001)</td>
<td>Age, wt and height, EI or EI difference, smoking</td>
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<tr>
<td>Bell et al. (2002)</td>
<td>8726</td>
<td>18–23 years, female</td>
<td>Apparently healthy (excluding pregnancy, serious medical condition), Australia</td>
<td>Prospective, 4 years follow-up</td>
<td>Self-report, two items (periods of moderate + vigorous ≥20 min at a time per week); Time spent sitting</td>
<td>BMI maintainers (ΔBMI &lt; 5%) or gainers (ΔBMI ≥ 5%)</td>
<td>No association between PA and wt maintenance status</td>
<td>Sociodemographics (education, marital status, occupation, parity, new mothers)</td>
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<tr>
<td>Bell et al. (2001)</td>
<td>2487</td>
<td>20–45 years, male and female</td>
<td>Healthy (excluding pregnancy, BMI &lt;12 kg/m² or &gt;60 kg/m², wt change &gt;20 kg/m²), China</td>
<td>Prospective, 8 years follow-up</td>
<td>Interview, work-related PA</td>
<td>ΔBW</td>
<td>Males and females who gained &gt;5 kg over 8 years were 3.1 (95% CI 1.7, 5.6) and 1.8 (95% CI 1.1, 3.1) times more likely to engage in light rather than heavy work-related PA</td>
<td>Wt, wt status, height, age, residence, income and education</td>
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<tr>
<td>Study</td>
<td>Study Population</td>
<td>Study Design</td>
<td>Measured PA at Work, Transportation to Work, Leisure Time</td>
<td>Measured BMI Change</td>
<td>Measured BW Change</td>
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<td>Wagner et al. (2001)</td>
<td>Healthy (excluding history of CHD, cancer or died during follow-up), France and Northern Ireland</td>
<td>Prospective, 5 years follow-up</td>
<td>Self-report last year PA at work, transportation to work and leisure time</td>
<td>Regular walking or cycling to work inversely related to BMI change ($\beta = -0.006; (95% CI = -0.011, 0.000). Subjects who performed high-intensity leisure time activity gained less in BMI than those who did not (beta: 0.10 (95% CI: 0.03, 0.17) kg/m^2)</td>
<td>Centre, age, marital status, education, work, socio-occupational class, dieting, alcohol, smoking, work PA</td>
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<tr>
<td>Rainwater et al. (2000)</td>
<td>Healthy Mexican-Americans (excluding anti-lipids and anti-hypertensive drugs, and diabetes), USA</td>
<td>Prospective, 5 years follow-up</td>
<td>Interview, Stanford 7 d recall</td>
<td>$\Delta$PA not correlated with $\Delta$BW</td>
<td>Age and gender</td>
<td></td>
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<td>Sherwood et al. (2000)</td>
<td>Community volunteers participating in a community wt-gain-prevention project, USA</td>
<td>Longitudinal, annual measurements for 3 years</td>
<td>PA history, thirteen items, leisure and occupational activity ≥20 min at a time; four categories; intensity and frequency in each category used</td>
<td>Men: increase in one high-intensity exercise session/week associated with decrease in wt of 0.53 (95% CI 0.33, 0.74) kg. Women: increase in one high-intensity exercise session/week associated with decrease in wt of 0.15 (95% CI 0.04, 0.25) kg. Increase in one moderate-intensity exercise session/week associated with decrease in wt of 0.11 (95% CI 0.05, 0.16) kg and an increase in one vigorous activity session/week associated with a decrease of 0.21 (95% CI 0.06, 0.36) kg.</td>
<td>Age, smoking status, education, income and marital status</td>
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<td>Schmitz et al. (2000)</td>
<td>Healthy (excluding pregnant; CARDIA study), USA</td>
<td>Longitudinal, five assessments over a 10-year period</td>
<td>Interview based self-report; frequency and participation in thirteen sport and exercises, no assessment of duration, Outcome exercise units (CARDIA PA history)</td>
<td>An increase in 200 'exercise units' (equal to regular exercise at 6 MET 2h/week for 11 months) expected to decrease BW by 0.38–1.12 kg/year</td>
<td>Age, education, alcohol intake, parity, smoking, % total EI from dietary fat at baseline and year 7</td>
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OR, odds ratio; LTPA, leisure-time PA; BW, body weight; $\Delta$, mean change; MI, myocardial infarction; HRT, hormone-replacement therapy; WC, waist circumference; EI, energy intake; MET, metabolic equivalent task; CARDIA, Coronary Artery Risk Development in Young Adults; TV, television.
accurately as possible. Similarly, unmeasured confounding can be reduced by including a greater range of possible factors. However, there are always factors that have not been considered and the only solution that really reduces confounding is randomisation within a trial.

Overall, the magnitude of the effect is small in the studies that report the expected inverse association between physical activity and subsequent weight gain. For example, Koh-Banerjee et al. (2003) have estimated that an increase in vigorous physical activity with a 25 metabolic equivalent task-h/week is associated with a reduction in waist circumference of 19 mm in men at 9-year follow-up. This increase in activity equates to approximately 4 h vigorous physical activity at 6 metabolic equivalent task-h/week. Sherwood et al. (2000) have shown that in men an increase of one high-intensity exercise session per week is associated with a decrease in weight of 0.53 kg at 3-year follow-up. In a large study in women with a 6-year follow-up Hu et al. (2003) have shown that for each 2 h increase in time spent watching TV there is a 23% increase in obesity risk. An increase in brisk walking for 1 h/d is associated with a 24% decrease in obesity risk. They have estimated that a relatively-active lifestyle, consisting of <10 h TV watching per week and ≥30 min brisk walking/d, would prevent 30% of the observed cases of obesity in this female cohort (Hu et al. 2003). However, all these studies have employed subjective measures of reported activity, which are known to estimate true PAEE with a high degree of error. It would be preferable to estimate the true magnitude of the relationship between activity and weight gain with objective measures of the PAEE.

Only two studies have been identified that describe the association between PAEE and weight gain using an objective measure of physical activity (Table 2). Tataranni et al. (2003) have used a doubly-labelled-water technique to assess PAEE and physical activity level in ninety-two Pima Indians aged 19–70 years. Physical activity level is calculated as total energy expenditure divided by RMR. Neither PAEE nor physical activity level is related to change in body weight during follow-up after 4 years. However, there is a positive association between total energy intake at baseline and change in body weight over the 4-year follow-up period. Ekelund et al. (2005) have examined whether PAEE assessed by heart-rate monitoring using individual calibration predicts weight gain and change in body composition (fat mass and fat free mass) in a population-based cohort of UK adults (n 739) over a period of 5 years. The association between PAEE and gain in body weight is modified by age. PAEE predicts change in fat mass in younger adults who as a group gained weight over the follow-up period, although the magnitude of the effect is small. In contrast, in older adults who are on average weight stable PAEE is associated with a gain in body weight, possibly as a result of preservation of fat free mass.

Table 2. Objectively-measured physical activity (PA) and weight gain in adults (>18 years)

<table>
<thead>
<tr>
<th>Study</th>
<th>Baseline age, gender</th>
<th>Selection</th>
<th>PA assessment</th>
<th>Method</th>
<th>Size of effect</th>
<th>Outcome</th>
<th>Size of effect</th>
<th>Confounding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ekelund et al. (2005)</td>
<td>Median age 53.8 years, male and female</td>
<td>Population-based sample, healthy Caucasians, UK</td>
<td>PAEE and PAL from DLW</td>
<td>Prospective, 4.6 months follow-up</td>
<td>PAEE predicted increase in FM in younger subjects who on average were at stable weight and FFM in older subjects who were at stable weight</td>
<td>ΔBW</td>
<td>PAEE and PAL not associated with change in BW</td>
<td>Gender, age, wt or body composition and duration of follow-up</td>
</tr>
<tr>
<td>Tataranni et al. (2003)</td>
<td>19–70 years, male and female</td>
<td>Healthy non-diabetic Pima Indians, USA</td>
<td>PAEE and PAL from DLW</td>
<td>Prospective, ≥4 months follow-up</td>
<td>PAEE and PAL not associated with change in BW</td>
<td>ΔBW</td>
<td>PAEE and PAL not associated with change in BW</td>
<td>Gender, age, wt or body composition and duration of follow-up</td>
</tr>
</tbody>
</table>

BW, body weight; Δ, mean change; DLW, doubly-labelled water; PAEE, PA-related energy expenditure; HR, heart rate; PAL, PA level; ΔFM, Δ fat mass; ΔFFM, Δ fat-free mass.

Objectively-measured physical activity and weight gain: studies in adults

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**Cohort studies in children and adolescents**

Molnar & Livingstone (2000) have reviewed published papers reporting observational studies of the association between physical activity and weight gain in children. As was the case in the adult studies (Fogelholm & Kukkonen-Harjula, 2000), they conclude that the results are inconsistent. Of the seven studies included in the 2000 review, four report that physical activity is associated with less weight gain in children, whereas the other studies do not observe an association. The present search has identified a further sixteen papers that have been published since the previous review. The major difference from the adult studies is that a greater number of these studies include objective measurement of activity or PAEE (five of sixteen; Figueroa-Colon et al. 2000; Johnson et al. 2000; Wells & Ritz, 2001; Bogaert et al. 2003; Moore et al. 2003; Treuth et al. 2003; Tammelin et al. 2004), with the remaining eleven studies using a self- or parental-report of physical activity (Berkey et al. 2000; Mamalakis et al. 2000; O’Loughlin et al. 2000; Davison & Birch, 2001; Horn et al. 2001; Kimm et al. 2001; Berkey et al. 2003; Bogaert et al. 2003; Francis et al. 2003; Proctor et al. 2003; Hancox et al. 2004; Tammelin et al. 2004).

**Self-reported physical activity and weight gain: studies in children and adolescents**

Table 3 gives an overview of the studies reporting on the longitudinal association between self-reported physical activity and weight gain in children, with follow-up ranging from 1 to 23 years. Five of the studies have a follow-up time of ≤2 years. The studies include between 59 and 11 887 children, with four studies including >1000 children. The ages of the children included range from 3 to 14 years, with most studies including children aged <10 years. Three studies include girls only (Davison & Birch, 2001; Horn et al. 2001; Francis et al. 2003). With only one exception (Tammelin et al. 2004), all the studies use reported change in BMI or sum of skinfolds as the outcome. Overall, the results are mixed. Five studies do not find an association between physical activity or sedentary behaviour and weight gain (Mamalakis et al. 2000; Davison & Birch, 2001; Kimm et al. 2001; Bogaert et al. 2003; Francis et al. 2003). The other six studies show an inverse association between higher levels of physical activity and weight gain, or a positive association with time spent on sedentary activities (Berkey et al. 2000; O’Loughlin et al. 2000; Horn et al. 2001; Berkey et al. 2003; Proctor et al. 2003; Hancox et al. 2004; Tammelin et al. 2004). However, as in the studies in adults, the measures of association tend to be small. Berkey et al. (2003) have shown that children who watch TV >3 h/d have a higher sum of skinfolds than the children watching <1.75 h/d, irrespective of physical activity level during the study period. Two studies initially included children or adolescents in their study, showing that physical activity in childhood and adolescence have an association with body composition in adulthood (Hancox et al. 2004; Tammelin et al. 2004). One of these studies followed >1000 children from 3 years of age until adulthood (26 years of age). This 23-year follow-up study has shown that one extra hour TV viewing daily between 5 and 15 years of age predicts a 0.54 kg/m² increase in BMI at the age of 26 years (Hancox et al. 2004). Approximately 17% of the overweight observed in adulthood could be attributed to TV viewing of >2 h/d during childhood (5–15 years).

**Objectively-measured physical activity and weight gain: studies in children and adolescents**

Five studies have reported on the longitudinal association between objectively-measured physical activity or PAEE and weight gain in children (Table 4). The children included in these studies are mostly younger than 10 years and the duration of follow-up ranges from 2 to 8 years. Physical activity or PAEE is measured using accelerometry (Moore et al. 2003) or the doubly-labelled-water technique (Figueroa-Colon et al. 2000; Johnson et al. 2000; Wells & Ritz, 2001; Treuth et al. 2003). The reported results are mixed. Three studies report on an association between physical activity and change in BMI (Figueroa-Colon et al. 2000; Moore et al. 2003; Treuth et al. 2003), and two of these studies are restricted to girls (Figueroa-Colon et al. 2000; Treuth et al. 2003). Treuth et al. (2003) report that total energy expenditure, measured by the doubly-labelled-water method, predicts change in percentage body fat in girls but in the opposite direction to that expected. The effect size, however, is small; an increase of 4.2kJ (1 kcal)/d results in a 0.002 increase in percentage body fat at follow-up. However, activity energy expenditure and physical activity level are not related to change in fat mass or percentage body fat. In a similar study in young girls Figueroa-Colon et al. (2000) have observed a prospective association between PAEE adjusted for fat-free mass and percentage body fat over a follow-up period of 1-6 years. However, this association is attenuated and is not significant after 2-7 years of follow-up. Johnson et al. (2000) have studied the longitudinal associations between energy expenditure and change in fat mass in prepubertal children over a period of 3–5 years. None of the energy expenditure measures predicts increasing adiposity. However, peak VO₂ uptake is a predictor of increasing adiposity in that study. The interpretation of this finding is different in children than in adults, since there is only a weak association between physical fitness and level of physical activity in children (Ekelund et al. 2001).

**Trials to prevent weight gain**

The observational studies of activity and weight gain in adults and children are affected by issues of measurement
<table>
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<tr>
<th>Study</th>
<th>n</th>
<th>Baseline age, gender</th>
<th>Selection</th>
<th>Method</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Hancox et al. (2004)</td>
<td>1037</td>
<td>3 years, male and female</td>
<td>Birth cohort in Otago, New Zealand</td>
<td>Longitudinal study, 23 years follow-up</td>
<td>TV viewing (5–11 years: parental report; 13–26 years: self-report)</td>
<td>BMI at 26 years, PAR for ≥2 h TV/d</td>
<td>Mean period (h) spent TV viewing from 5 to 15 years predicted BMI at 26 years (0.54 (95% CI 0.19, 0.87) kg/m² per h increase in TV viewing), PAR at 26 years indicate that 17% of the overweight can be attributed to watching ≥2 h TV between 4 and 15 years</td>
<td>Gender, childhood socio-economic status, BMI at 5 years and parental BMI</td>
</tr>
<tr>
<td>Tammelin et al. (2004)</td>
<td>5706</td>
<td>14 years, male and female</td>
<td>Birth cohort, Finland</td>
<td>Prospective cohort, 17 years follow-up</td>
<td>Change in PA assessed by: frequency of sport participation in leisure time at 14 years (self-report) and frequency of brisk PA at 31 years</td>
<td>Obesity (abdominal) at 31 years</td>
<td>Compared with being persistently active, becoming inactive associated with overweight in males (OR 1.49; (95% CI 1.18, 1.89)), obesity in males (OR 1.51 (95% CI 0.99, 2.37)), and severe abdominal obesity in females (OR 1.80 (95% CI 1.13, 2.95)). Being persistently inactive associated with mild abdominal obesity in males (OR 1.83 (95% CI 1.13, 2.95)).</td>
<td>Maternal BMI, BMI at 14 years, education, occupational PA at 31 years, alcohol consumption, smoking, parity</td>
</tr>
<tr>
<td>Berkey et al. (2000, 2003)</td>
<td>11 887</td>
<td>9–14 years, male and female</td>
<td>Nationwide sample, USA</td>
<td>Longitudinal study, 1 year follow-up</td>
<td>Self-reported sedentary activity (TV, video, games), no. of gym classes and recreational activities</td>
<td>Change in BMI</td>
<td>Change in BMI in girls (0.06 (95% CI –0.11, –0.01) kg/m² per h increase in daily activity) and in overweight boys (–0.22 (95% CI –0.33, –0.10) kg/m²) BMI; in girls (0.037 (95% CI 0.016, 0.058) kg/m² increase per h watching TV per d and 0.028 (95% CI –0.056, –0.00) kg/m² decrease per h recreational activity per d); in boys (0.038 (95% CI 0.019, 0.058) kg/m² increase per h watching TV per d and 0.026 (95% CI –0.057, 0.004) kg/m² decrease per h recreational activity per d)</td>
<td>Race or ethnicity, EI, height growth, Tanner* and menarche history</td>
</tr>
<tr>
<td>Francis et al. (2003)</td>
<td>173</td>
<td>5 years, female</td>
<td>Sample of families (non-representative), USA</td>
<td>Longitudinal, 4 years follow-up</td>
<td>Self-report of tendency towards participating in PA, parental report of TV viewing (only at age 7–9 years)</td>
<td>Change in BMI</td>
<td>Tendency towards PA not related to change in BMI, TV viewing predicted change in BMI in girls from non-overweight families</td>
<td>Parental BMI, snacking, fat intake, BMI at age 5 years and family income</td>
</tr>
<tr>
<td>Bogaert et al. (2003)</td>
<td>59</td>
<td>6–9 years, male and female</td>
<td>Volunteer families, Australia</td>
<td>Prospective cohort, 1 year follow-up</td>
<td>Bouchard PA record (parental report)</td>
<td>Change in BMI z-score</td>
<td>No correlation between BMI z-score change and amount (h) of planned exercise, amount (h) of TV viewing, and either percentage time in low-intensity activity, in moderate intensity activity, or in moderate-to-high intensity activity</td>
<td></td>
</tr>
<tr>
<td>Study</td>
<td>Sample Size</td>
<td>Age &amp; Gender</td>
<td>Data Collection Method</td>
<td>Outcome Measures</td>
<td>Findings</td>
<td></td>
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<tr>
<td>Proctor et al. (2003)</td>
<td>106</td>
<td>3-5 years, male and female</td>
<td>Framingham offspring, USA</td>
<td>Longitudinal, 7 years follow-up</td>
<td>Parental report of TV (and video viewing) SSF, BMI</td>
<td>Children who watched ≥3 h TV per d had a mean SSF of 106.2 mm, compared with a mean SSF of 76.5 mm for those who watched &lt;1.75 h TV per d (P=0.007)</td>
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<tr>
<td>Horn et al. (2001)</td>
<td>198</td>
<td>7-5 years, male and female (grades 1-4)</td>
<td>Sample from two Mohawk communities, Canada</td>
<td>Longitudinal, 2 years follow-up</td>
<td>Self-report of PA (weekly checklist) and TV viewing SSF</td>
<td>Excessive TV viewing and higher relative PA associated with higher SSF at follow-up in girls. No effect of PA in boys</td>
<td></td>
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<tr>
<td>Kimm et al. (2001)</td>
<td>2379</td>
<td>9-10 years, female</td>
<td>Sample from California and Cincinnati, OH, USA</td>
<td>Longitudinal, 10 years follow-up</td>
<td>Self-reported PA: 3 d diary (including one weekend day) Change in SSF</td>
<td>PA not related to change in SSF for both races</td>
<td></td>
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<tr>
<td>Davison et al. (2001)</td>
<td>197</td>
<td>5 years, female</td>
<td>Sample (non-Hispanic white) Pennsylvania, USA</td>
<td>Cohort study, 2 years follow-up</td>
<td>Parental report (5 years), self-reported tendency towards PA (at 7 years) Change in BMI</td>
<td>BMI not related to change in SSF for both races</td>
<td></td>
<td></td>
</tr>
<tr>
<td>O'Loughlin et al. (2000)</td>
<td>2318 (1 year sample), 633 (2 years sample)</td>
<td>9-12 years, male and female</td>
<td>Montreal Heart Health Program, Canada</td>
<td>Longitudinal, 1 and 2 years follow-up</td>
<td>Self-report, 7 d recall of total PA, school sports and organised sport outside school; TV and video games as sedentary BMI, excessive wt gain BMI &gt;90th percentile 1-year predictors of &gt;90th percentile of change in BMI: in boys included baseline BMI of ≥90th percentile (OR 2.66 (95% CI 1.80, 3.94)); in girls included baseline BMI of ≥90th percentile (OR 2.34 (95% CI 1.46, 3.76)), no sports outside school (OR 1.90 (95% CI 1.18, 3.06)) and playing video games everyday (OR 2.48 (95% CI 1.04, 5.92)) 2-year predictors of &gt;90th percentile of change in BMI: in boys included baseline BMI of ≥90th percentile (OR 3.26 (95% CI 1.52, 7.01)), no sports outside school (OR 2.14, (95% CI 1.01, 4.71)) and least active (OR 2.18 (95% CI 1.01, 4.71)); in girls only baseline BMI of ≥90th percentile (OR 2.22 (95% CI 1.02, 4.81))</td>
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<tr>
<td>Mamalakis et al. (2000)</td>
<td>1046</td>
<td>6 years, male and female</td>
<td>Representative sample, Greece</td>
<td>Prospective cohort study, 6 years follow-up</td>
<td>Parental report of three typical days, total time spent on strenuous PA BMI, SSF, WHR at 9 and 12 years</td>
<td>PA not predict BMI, SSF, or WHR at age 9 and 12 years BMI, WHR, and SSF, dietary intake, treatment, endurance run test, children’s and parents’ knowledge on health, parent-related factors, region and gender</td>
<td></td>
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</tr>
</tbody>
</table>

PAR, population attributable risk; OR, odds ratio; SSF, sum of skinfolds; WHR, waist:hip ratio; TV, television; EI, energy intake.

*Classification of sexual maturation (Tanner, 1962).*
Table 4. Objectively-measured physical activity (PA) and weight gain in children (age ≤18 years)

<table>
<thead>
<tr>
<th>Study</th>
<th>n</th>
<th>Baseline age, gender</th>
<th>Selection Method</th>
<th>PA assessment</th>
<th>Outcome</th>
<th>Size of effect</th>
<th>Confounding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moore et al. (2003)</td>
<td>103</td>
<td>3–5 years, male and female</td>
<td>Framingham children’s study, USA</td>
<td>Longitudinal, annual measurements during 8 years follow-up</td>
<td>Caltrac activity monitor</td>
<td>ΔBMI, TSF, and SSF</td>
<td>Children in the highest tertile of mean activity (over follow-up period) had lower BMI, TSF, and SSF at age 11 years (for lowest, middle and highest tertile, respectively: BMI (kg/m²): 20.3 (SD 0.6), 19.8 (SD 0.5), 18.6 (SD 0.6; P= 0.05); TSF (mm): 18.6 (SD 1.0); SSF (mm): 95.1 (SD 6.8); 74.1 (SD 6.9; P&lt; 0.05)). However, no significant longitudinal associations between change and PA are reported</td>
</tr>
<tr>
<td>Treuth et al. (2003)</td>
<td>88</td>
<td>8–9 years, female</td>
<td>Normal-wt (&lt;90th percentile wt-for-height and% BF 12–30), USA</td>
<td>Longitudinal, 1 and 2 years follow-up</td>
<td>TEE by DLW, PAEE = TEE – RMR; PAL = TEE/RMR</td>
<td>ΔFM by DXA</td>
<td>No associations between PAEE and PAL and change in FM. TEE predicts a higher change in% BF (β 0.002 (95% CI 0.000, 0.0004))</td>
</tr>
<tr>
<td>Wells &amp; Ritz (2001)</td>
<td>26</td>
<td>9–12 months, gender not mentioned</td>
<td>Healthy, born at term, UK</td>
<td>Prospective, follow-up at age 2 years</td>
<td>TEE by DLW, adjusted for FFM</td>
<td>FMI by 18O, skinfold</td>
<td>No association between TEE/FFM in infancy and fatness at age 2 years. Parent-reported infant PA behaviour is positively associated with childhood skinfold thickness</td>
</tr>
<tr>
<td>Johnson et al. (2000)</td>
<td>115</td>
<td>4.6–11.0 years, female and male</td>
<td>Free of any major illness since birth, USA</td>
<td>Longitudinal, annual follow-up for year 3–5 years</td>
<td>TEE by DLW, AEE=0.9 × TEE-RMR</td>
<td>ΔFM by DXA</td>
<td>None of the measures of EE predicted change in FM</td>
</tr>
<tr>
<td>Figueroa-Colon et al. (2000)</td>
<td>47</td>
<td>4.8–8.9 years, female</td>
<td>Normal wt (10th to 85th percentile for BMI), Tanner stage 1, USA</td>
<td>Longitudinal, at 1-6 and 2-7 years follow-up</td>
<td>TEE by DLW, PAEE = 0.9 × TEE-RMR</td>
<td>ΔFM by DXA</td>
<td>PAEE adjusted for FFM was a predictor of FM at first follow-up but not at second follow-up</td>
</tr>
</tbody>
</table>

SF, skinfold; SSF, sum of SF; EE, energy expenditure; TEE, total EE; DLW, doubly-labelled water; FFM, fat-free mass; FMI, fat mass index; AEE, activity-related EE; PAEE, PA-related EE; DXA, dual-energy X-ray absorptiometry; Δ, mean change; FM, fat mass; PAL, PA level; BF, body fat; TSF, triceps SF thickness; TV, television.

*Classification of sexual maturation (Tanner, 1962).
error, residual and unmeasured confounding, and reverse causality. Although improved study design with greater emphasis on objective measures with known degree of measurement error can deal with a number of these issues, they cannot resolve problems of the direction of association or deal with unmeasured confounding, for which randomised clinical trials are required. Although there are a large number of clinical trials on the treatment of individuals with obesity or the prevention of weight regain among weight losers, a recent systematic review of trials to prevent weight gain de novo has only revealed a total of nine trials (Hardeman et al. 2000). Not only are these trials relatively few in number but also, for various methodological reasons, they are uncertain in their conclusions about whether increasing activity will be effective in preventing obesity. Campbell et al. (2001) have drawn similar conclusions from their review of the trials on obesity prevention in children. This review comprises seven trials, of which one only includes a dietary intervention. Six of the trials studied interventions aimed at preventing obesity by increasing physical activity, which is combined with changing dietary behaviour in three of these trials.

The aim of the second part of the present review is to update the two previous reviews by summarising the papers published from 2000 onwards on controlled trials studying interventions aimed at preventing weight gain in both adults and children by increasing physical activity or decreasing physical inactivity. For this purpose, the search criteria applied by Hardeman et al. (2000) have been used. The following inclusion criteria have been used: (a) physical activity promotion is a main component of the intervention; (b) the effects on changes in weight or body composition are reported; (c) the intervention is not aimed at weight reduction or at preventing weight regain. The search has revealed a total of seventeen additional studies, of which six include adults and eleven include children.

**Trials in adults**

A total of six trials aimed at increasing physical activity and preventing weight gain in adults have been identified (Table 5; Muto & Yamauchi, 2001; Polley et al. 2002; Burke et al. 2003; Litterell et al. 2003; Proper et al. 2003; Simkin-Silverman et al. 2003). The interventions are mostly aimed at populations who are either defined on the basis of their risk for weight gain or because they could be a specific group to whom an intervention might be targeted. These groups include couples in their first 2 years of living together (Burke et al. 2003), the working population (Muto & Yamauchi, 2001; Proper et al. 2003), pregnant (Polley et al. 2002) or middle-aged premenopausal (Simkin-Silverman et al. 2003) women, or patients taking drugs that induce weight gain as a side-effect (Litterell et al. 2003). In general, the interventions are of a high intensity and are spread over a relatively long period of time (ranging from 12 weeks to 5 years) with face-to-face counselling on behaviour change in either group or individual settings. Two studies have follow-up measurements >3 months after the end of the intervention (Muto & Yamauchi, 2001; Burke et al. 2003). As in the Hardeman et al. (2000) review, the description of the underlying theories supporting the interventions is limited. Only one of the interventions is based on a behaviour change theory, the Trans Theoretical Model (Proper et al. 2003). In four of the seven trials there are differences in body composition between the intervention and control group (Muto & Yamauchi, 2001; Litterell et al. 2003; Proper et al. 2003; Simkin-Silverman et al. 2003), which is also sustained at longer follow-up (Muto & Yamauchi, 2001). The dynamics of change differ between the studies, as some show an increase in body weight in the control group and weight stability in the intervention group (Litterell et al. 2003; Simkin-Silverman et al. 2003), whereas others show a decrease in the intervention group (Muto & Yamauchi, 2001), or decreases in both study groups (Proper et al. 2003).

**Trials in children and adolescents**

A total of eleven trials aimed at preventing unhealthy weight gain by increasing physical activity or reducing sedentary behaviour in children have been found (Table 6; Sahota et al. 2001; McMurray et al. 2002; Baranowski et al. 2003; Caballero et al. 2003; Neumark-Sztainer et al. 2003; Pangrazi et al. 2003; Robinson et al. 2003; Sallis et al. 2003; Warren et al. 2003; Dennison et al. 2004; Kain et al. 2004). Nine trials have studied the effectiveness of school-based interventions, whereas the others have studied home-based or family-orientated interventions (Baranowski et al. 2003; Robinson et al. 2003). Effectiveness is mostly assessed directly after the intervention and only one study includes a follow-up measurement at >3 months after the end of the intervention (Dennison et al. 2004). In three of the eleven trials there is a small intervention effect on body composition at follow-up (McMurray et al. 2002; Sallis et al. 2003; Kain et al. 2004), with two of them (Sallis et al. 2003; Kain et al. 2004) reporting effects for boys only. Although several of the non-effective trials report on positive changes in physical activity levels or on dietary behaviour, they do not show significant differences in body weight or body composition at follow-up between the intervention and control group. When studying the intensities and settings of the interventions, it seems that comprehensive school-based interventions aimed at increasing physical activity levels through physical education classes and behaviour change are most likely to be effective in preventing weight gain in children, whereas interventions aimed at reducing sedentary behaviour and family-based interventions seem to be less effective.

The present updated review demonstrates that there are still relatively few trials aimed at the primary prevention of weight gain and that there is, therefore, still insufficient evidence on which to base conclusions about which of the approaches are effective. It is evident, however, that understanding information from such trials would be enhanced if they are based on an explicit causal model with a clear theoretical foundation, so that even if the overall effect is non-significant, it would be possible to disentangle which aspects of the intervention are ineffective. At present, most of the interventions are black boxes, and when they are not effective it is difficult to generalise
### Table 5. (Randomised) controlled trials to prevent weight gain by increasing physical activity (PA) in adults (>18 years)

<table>
<thead>
<tr>
<th>Study</th>
<th>Subjects</th>
<th>Intervention</th>
<th>Behaviour change method</th>
<th>Main outcome measure</th>
<th>Size of effect</th>
</tr>
</thead>
</table>
| Burke et al. (2003)    | 137 couples (274 subjects; women 28–6 (range 18–62) years, men 31–4 (range: 20–61 years, BMI ≥ 25 kg/m²: 61 %), Australia) | 1. High-level intervention: six printed modules focusing on PA and nutrition. A module was received every 2 weeks alternating between mailing modules and discussing them during interactive sessions  
2. Low-level intervention: six printed modules focusing on PA and nutrition (same as 1), mailed every 2 weeks  
3. No intervention | Couple-based, no behaviour change model specified | BMI, WHR, cholesterol, BP, PA, diet at end of intervention and 1-year follow-up | No significant differences in BMI and WHR At both follow-ups, fall in total cholesterol and LDL-cholesterol in the high-level intervention group, compared with the control group. At the end of the intervention, an increase in the number of PA sessions and a decrease in saturated fat intake for both intervention groups compared with the control group, but not at 1-year follow-up |
| Litterell et al. (2003) | Seventy schizophrenic patients (male and female) taking an anti-psychotic drug known to induce wt gain (mean age intervention 33–7 (so 9–2) years, control 34–5 (so 10–0) years), USA | 1. Weekly, 1 h psycho education classes for 16 weeks discussing 2 main modules: ‘Nutrition, Wellness, and Living a Healthy Lifestyle’ and ‘Fitness and Exercise’  
2. Standard treatment | Group-based sessions, no behaviour-change model specified | Wt and BMI, at 4- and 6-month follow-up | Differences in wt changes at 4 months (control 3.3. (95% CI –4–9, 11–4) kg v. intervention 0.4 (95% CI –7–6, 8–3) kg), $P$=0.005, and at 6 months (control 4.3 (95% CI –7–2, 15–8) kg v. intervention –0.05 (95% CI –8–4, 8–3) kg, $P$=0.007). In both groups men gained more than women. No differences in change in BMI |
| Proper et al. (2003)   | 299 employees (77% male) of municipal services (intervention 43–8 (so 8–3) years, control 44–4 (so 9–4) years), The Netherlands | 1. Written lifestyle information plus seven individual 20-min PACE-counselling sessions by a physiotherapist, primarily focused on enhancing PA and secondarily on promoting healthy nutrition habits. Stage of change was assessed in the first session, whereas an individual behaviour change plan was created in the second session. This plan was discussed and evaluated in the five following sessions (intervention took 9 months)  
2. Written lifestyle information only | Individual work-based counselling, based on Trans Theoretical Model of Behaviour Change | PA, wt, BMI, and % body fat at 9-month follow-up | % Body fat significantly different at follow-up ($β$=0.22 (95% CI –0.47, –0.03), although both groups decreased. No intervention effect on BMI. EE differed at follow-up ($β$=176.2 (95% CI 60.6, 291.8), mainly as a result of a decrease in EE in the control condition. Non-significant effect on the proportion of subject meeting PA guidelines (OR1.46 (95% CI 0.76, 2.79)) |
| Simkin-Silverman et al. (2003) | 535 healthy premenopausal women, aged 44–50 years USA | 1. 5-year lifestyle intervention. First, to achieve long-term prevention of wt gain, modest wt-loss goals given, to be achieved by three goals: 25% energy from total fat, 7% energy from saturated fat, increase PA expenditure to 4184–6276 kJ/week (6 months, fifteen group meetings. Follow-up provided six-weekly for 4-5 years providing additional behavioural skills, support and motivation  
2. Assessment-only control condition | Group-based sessions, no behaviour change model specified | PA, WC, BW, BMI, % body fat, and % fat-free mass, at 30, 42, and 54 months follow-up | Wt gain prevention: 55% of intervention subjects at or below baseline wt (mean change: −0.1 (so 5–2) kg compared with 26% of controls (mean change: 2.4 (so 9) kg at 54-month follow-up ($P<0.001$). WC decreased significantly more in intervention subjects than in controls (−29 (so 53) mm v. −5 (so 56) mm, $P<0.001$). At all follow-ups, intervention subjects more physically active, and eating fewer energy and less fat than controls. Long-term adherence to PA and a low-fat eating pattern associated with better wt maintenance |
<table>
<thead>
<tr>
<th>Study</th>
<th>Participants</th>
<th>Design</th>
<th>Intervention Details</th>
<th>Outcome Measures</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polley et al. (2002)</td>
<td>120 pregnant women (BMI &gt;19.8 kg/m², age &gt;18 years, &lt;20 weeks gestation) from a hospital-based clinic serving low-income women, USA</td>
<td>1. Individual sessions aimed at healthy wt gain during pregnancy during all regular scheduled clinic visits (including: reviewing wt gain chart, assessment of current diet and exercise, review of progress toward behaviour goals, problem-solving, instruction to uses, behavioural techniques and goal setting for dietary and exercise behaviour. Extra sessions offered to women exceeding normal wt gain patterns)</td>
<td>Individual counselling, no behaviour change model specified</td>
<td>Wt gain, wt in relation to IOM recommendations, post-partum wt loss</td>
<td>Among normal-prepregnancy-wt women, 58% of the control group exceeded the IOM-recommendation, whereas 33% of the women in the intervention group (P&lt;0.05). The intervention had no effect on average wt gain from pre-pregnancy to delivery, or on postpartum wt loss</td>
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<tr>
<td>Muto &amp; Yamauchi (2001)</td>
<td>302 male employees of a building maintenance company (mean age: intervention 42.3 (sd 4.5) years, control 42.7 (sd 2.7) years, Japan</td>
<td>1. Multicomponent health promotion programme during a 4 d seminar mainly aimed at improving dietary behaviour and PA consisting of several components: lectures (6 × 60 min), practical training (8 × 60–180 min), individual counselling (1 × 120 min), group discussion (3 × 60–120 min), self-education (2 × 60 min). Four follow-up self-evaluation sessions (every 3 months)</td>
<td>Group-based, no behaviour change model specified</td>
<td>Wt, BMI, cholesterol, BP, and fasting blood glucose, at 6 and 18 months after main intervention (4 d seminar)</td>
<td>A significant intervention effect on wt change (kg) from baseline at 6 months (intervention –1.6 (95% CI –6.9, 3.7), control 0.1 (95% CI –3.6, 3.8); P&lt;0.001) and at 18 months (intervention –1.0 (95% CI –7.3, 5.3), control 0.5 (95% CI –3.8, 4.8); P&lt;0.001). BMI (kg/m²) changed significantly at 6 months (–0.5 (95% CI –2.3, 1.3), control: 0.0 (95% CI –1.4, 1.4), and at 18 months (–0.3 (95% CI –2.5, 1.9), control 0.2 (95% CI –1.4, 1.8); P&lt; 0.001). Favourable changes at both follow-ups also for systolic BP and total cholesterol. Favourable changes for diastolic BP and fasting blood glucose at 6 months only</td>
</tr>
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</table>

WHR, waist:hip ratio; BP, blood pressure; OR, odds ratio; EE, energy expenditure; BW, body weight; WC, waist circumference; IOM, Institute of Medicine; PACE, Patient-centered Assessment and Counseling for Exercise.
### Table 6. (Randomised) controlled trials to prevent weight gain by increasing physical activity (PA) in children (≤18 years)

<table>
<thead>
<tr>
<th>Study</th>
<th>Subjects</th>
<th>Intervention</th>
<th>Behaviour-change method</th>
<th>Main outcome measure</th>
<th>Size of effect</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Kain et al. (2004)</strong></td>
<td>Students (male and female) from Chilean primary schools with low SES (1st–8th grade). Intervention: n 2141 (mean age 10.6 years, BMI: 19.6 kg/m², % ≥ P95 CDC: 18), control: n 1202 (mean age 10.6 years, BMI: 19.2 kg/m², % ≥ P95 CDC 11.3), Chile</td>
<td>1. Combined dietary and PA intervention. Diet: trained nutritionist provided educational programme to children (5–11h over 6 months), information meetings with owners of kiosks in schools, and parent meetings (two times). PA: Canadian Active Living Challenge applied by research PE teacher once weekly for 6 months, 90 min./week extra PA from PE teacher (sports activities), active recess (15 min/day), and extra activities per school</td>
<td>School-based, no theoretical model stated</td>
<td>BMI, TSF, and WC at 8-month follow-up</td>
<td>Boys: significant effect on BMI (kg/m²); intervention baseline 19.5 (SD 3.7), follow-up 19.2 (SD 3.5); control baseline 18.9 (SD 3.3), follow-up 19.2 (SD 3.1); P &lt; 0.001 and on WC (mm; intervention baseline 674 (SD 109), follow-up 665 (SD 96); control baseline 646 (SD 93), follow-up 655 (SD 89); P &lt; 0.001). Girls: no significant effects. No effect reported on PA and dietary intake</td>
</tr>
<tr>
<td><strong>Dennison et al. (2004)</strong></td>
<td>Children from preschool or day care centre (male and female) intervention: n 43 (age 3–9 (± 0.07) years, BMI 15.9 (± 0.3) kg/m²), control: n 34 (age 4.0 (± 0.10) years, BMI 15.9 (± 0.2) kg/m²), USA</td>
<td>1. Raising parents’ awareness of children’s TV viewing with a 1-week diary, and seven weekly 1 h sessions including a 20 min interactive, educational session for parents, children and preschool staff led by programme staff. Topics included: read stories daily, family meal time with TV off, thinking of alternatives for watching TV, festivities for not watching TV for 1 week. 2. Safety and injury prevention programme</td>
<td>Parent-based, no theoretical model stated</td>
<td>TV viewing (parental report), wt, BMI, and TSF at 6-month follow-up</td>
<td>TV viewing: (borderline) significant differences in mean change in favour of intervention group (h): weekdays –0.62 (95% CI –1.11, –0.12), Saturdays –0.63 (95% CI –1.44, 0.17), Sundays –0.99 (95% CI –1.73, –0.25). No significant differences in mean change for BMI and TSF. No report on relationship between change in TV viewing and BMI or TSF</td>
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<td><strong>Pangrazi et al. (2003)</strong></td>
<td>606 4th grade students (male and female) from Arizona schools (age 9–8 (± 0.06) years), USA</td>
<td>1. PLAY: 12-week intervention implemented during the school day and facilitated by classroom teachers. PLAY aims at daily PA and consists of three steps: promote play behaviour, introduce teacher directed activities (daily 15 min active break), and encourage self-directed activity 2. PE: Existing PE classes 3. PLAY and PE: combination of interventions 1 and 2 4. No treatment</td>
<td>School-based (teacher-facilitated), no behaviour change model stated</td>
<td>PA (steps per d assessed with pedometer), and BMI at 12-week follow-up (directly after intervention)</td>
<td>No significant differences in BMI were observed. Compared with ‘no treatment’ group, ‘PLAY and PE’ and ‘PLAY’ groups took significantly more steps per d (‘no treatment’ 11 180 (95% CI 2826, 19 533), ‘PLAY and PE’ 12 763 (95% CI 5250, 20 276; P = 0.010), ‘PLAY’ 12 598 (95% CI 4707, 20 489; P = 0.035). Stratified analyses for gender showed significant differences compared with ‘no treatment’ for the ‘PLAY and PE’ and the ‘PE only’ in girls, but not in boys</td>
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<td><strong>Caballero et al. (2003)</strong></td>
<td>1074 2nd grade American Indian students (male and female; mean age 7.6 (± 0.6) years, mean BMI 18.8 (± 3.9) kg/m², mean % body fat 32.6 (± 6.8), USA</td>
<td>1. 3-year pathways-intervention consisting of four components: classroom curriculum (45 min two times weekly), food service (reducing fat in school meals), physical education (exercise break during recess, 30 min PE sessions three times weekly), and family involvement (information and events) 2. No intervention</td>
<td>School-based, no behaviour change model stated</td>
<td>% Body fat, BMI, TSF, dietary intake (observation of school lunch and 24 h recall), PA (motion sensor and self-report) at 3-year follow-up</td>
<td>No significant differences between the study groups in % body fat, BMI or TSF were observed. Significant changes in favour of the intervention group for dietary intake (mean difference at follow-up from 24 h recall of 1109 (95% CI –437, –94) kJ/d (P = 0.003), and −2.5 (95% CI –3.9, –1.1) % energy from fat (P = 0.001); from school-lunch observation −4.2 (95% CI –7.1, –1.3) % energy from fat (P = 0.005) and for PA (mean difference at follow-up from self-report 0.04 (95% CI 0.01, 0.06; P = 0.001). No intervention effect was detected with the motion sensor</td>
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<tr>
<td>Study</td>
<td>Sample</td>
<td>Interventions</td>
<td>Design</td>
<td>Outcomes</td>
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<td>Warren et al. (2003)</td>
<td>213 1st and 2nd grade students (male and female) from Oxford primary schools (age 6-1 (so 0.6) years, BMI 15-9 (so 2-1) kg/m², 8% overweight, 2% obese), UK</td>
<td>14-month interventions: weekly session in the 1st term, bi-weekly in the 2nd–4th term 1. Eat smart: sessions aiming at increasing knowledge about food and health, and promoting eating healthy food 2. Play smart: PA group promoting PA in daily life, and reduction of TV viewing 3. Eat smart play smart: combination of intervention 1 and 2 4. Be smart: control condition with educational programme on food in a non-nutrition sense</td>
<td>School-based, (provided by external educator), based on Social Learning Theory</td>
<td>% Overweight or obese (assessed from BMI reference values), dietary intake (parental report), and PA (self-report and parental report) at 14-month follow-up</td>
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<td>Sallis et al. (2003)</td>
<td>1109 6th–8th grade students (male and female) from San Diego middle schools, USA</td>
<td>1. Three-component intervention: PA interventions to increase PA in PE classes and throughout the school day, nutrition interventions to provide and market low-fat foods at all school food sources, and school staff and students were engaged in policy change efforts. There was no classroom health education and the intervention lasted 2 years 2. No intervention</td>
<td>School-based, based on Cohen's structural, ecological model of health behaviour</td>
<td>BMI, fatty food intake and PA (observation) at 2-year follow-up</td>
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<td>Neumark-Sztainer et al. (2003)</td>
<td>201 9th–12th grade females from Minnesota high schools (age 15-4 s (1:1) year, BMI 26-7 (16-5) kg/m²), USA</td>
<td>1. New moves: girls-only alternative PE programme (instead of regular coed PE classes) consisting of 4 PA sessions per week, bi-weekly social support sessions, and bi-weekly nutritional guidance (over 16 weeks and provided by teachers and members of the research team) 2. Written information on PA and nutrition (once at baseline measurement)</td>
<td>School-based, based on Social Cognitive Theory</td>
<td>BMI, PA, sedentary activity, fruit and vegetable intake, and soda pop intake, post-intervention and at 3-month follow-up</td>
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<td>Baranowski et al. (2003)</td>
<td>Thirty-five African-American girls (8 years, BMI (kg/m²) intervention 21.1 (so 4.4), control 26.3 (so 6.5) kg/m²), USA</td>
<td>1. GEMS-FFFP project: 4-week summer day camp and additional weekly home Internet intervention (eight times; for both girls and their parents) aimed at increasing fruit and vegetable intake, water intake, and increasing PA to 60 min/d 2. Usual summer day camp and monthly Internet program (two times) for both girls and their parents (not including FFFP-features)</td>
<td>Community and family-based (pilot-project), based on Social Cognitive Theory</td>
<td>BMI, PA (CSA and self-report), and dietary intake at mean follow-up of 16 weeks</td>
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<td>Robinson et al. (2003)</td>
<td>Sixty-one African-American girls (8–10 years, BMI (kg/m²) intervention 21.3 (so 4.4), control 26.3 (so 7.9)), USA</td>
<td>1. Intervention consisting of (a) providing free 2-5h dance classes 5/week in community centres, and (b) five lessons during home visits aimed at reducing TV viewing (electronic TV time managers provided). Intervention lasted 12 weeks 2. State-of-the-art information-based health education programme consisting of monthly community health lectures and newsletters</td>
<td>Community and family-based (pilot-project), based on Social Cognitive Theory</td>
<td>BMI, WC, PA (CSA and self-report), TV use, and dietary intake at 12-week follow-up</td>
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Small changes in the percentage of children classified as overweight or obese from baseline to follow-up, but no differences between the study groups. An overall increase in the consumption of vegetables (P<0.05) and fruit (P<0.01), but no differences between the study groups. No differences in PA.
Table 6. (Continued)

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<tr>
<th>Study</th>
<th>Subjects</th>
<th>Intervention</th>
<th>Behaviour-change method</th>
<th>Main outcome measure</th>
<th>Size of effect</th>
<th>Study Subjects Intervention</th>
<th>Size of effect</th>
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<tbody>
<tr>
<td>McMurray et al. (2002)</td>
<td>1140 students (male and female) from middle schools in North Carolina 12 (SD 1) years; 28.7% had BMI‡ 30 kg/m², USA</td>
<td>1. Active programme promoting lifestyle in schools consisting of teacher training, modification of school meals, and the development and implementation of school action plans designed to promote healthy eating and PA over 1 year</td>
<td>School-based, no behaviour change model mentioned</td>
<td>BM, SSF, and BP directly after the intervention</td>
<td>No difference in BMI between the intervention and control groups (P = 0.0001), change in SSF (P = 0.0001), increase in systolic and diastolic BP in the intervention group (P = 0.0001). The changes in SSF, sum of skinfolds (SSF), and triceps skinfold thickness (TSF) measured at 1 year follow-up were significantly different (P &lt; 0.001). Further, obese children in the intervention group also showed lower fruit consumption at follow-up than controls (P &lt; 0.001).</td>
<td>No difference in change in BMI between the four study groups. Overall, a significant difference in BMI, SSF, and BP between the intervention and control group was observed. The changes in SSF, sum of skinfolds (SSF), and triceps skinfold thickness (TSF) measured at 1 year follow-up were significantly different (P &lt; 0.001). Further, obese children in the intervention group also showed lower fruit consumption at follow-up than controls (P &lt; 0.001).</td>
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<td>Sahota et al. (2001)</td>
<td>636 primary school children enrolled in two schools in the UK</td>
<td>2. Normal school curriculum + health education</td>
<td>Classroom-based, no behaviour-change model mentioned</td>
<td>BM, BMR, and PA at 12-month follow-up</td>
<td>No difference in BMI between the intervention and control group (P = 0.0001), but a significant difference in BMI, SSF, and BP between the intervention and control group was observed. The changes in SSF, sum of skinfolds (SSF), and triceps skinfold thickness (TSF) measured at 1 year follow-up were significantly different (P &lt; 0.001). Further, obese children in the intervention group also showed lower fruit consumption at follow-up than controls (P &lt; 0.001).</td>
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SSF, sum of skinfolds; TSF, triceps skinfold thickness; WC, waist circumference; BP, blood pressure; %, P95 CDC, percentage of subjects >95th percentile of CDC-F; TV, television; FFFP, Fun, Food, and Fitness Project; CSSE, Centre for School Sport, Exercise, and Health; PSES, Physical Education and School Sport Centre; BM, body mass index; BMI, body mass index; SSF, sum of skinfolds; TSF, triceps skinfold thickness; WC, waist circumference; BP, blood pressure; %, P95 CDC, percentage of subjects >95th percentile of CDC-F; TV, television; FFFP, Fun, Food, and Fitness Project; CSSE, Centre for School Sport, Exercise, and Health; PSES, Physical Education and School Sport Centre.

explanations for their ineffectiveness. In the absence of clearly successful trials, the evidence from randomised controlled trials does not contribute solutions to the issues left unresolved by observational cohort data.

Analysis of true population-level approaches to increasing physical activity

All the trials considered in the present review were aimed at changing individual behaviour. However, if a broader perspective is taken it is clear that this approach ignores important collective determinants of physical activity. These determinants, including environmental influences such as transport policy, are much less amenable to the traditional medical reductionist approach to evaluation and it is unlikely that they will ever be subject to assessment by a randomised controlled trial. However, if they are powerful influences on physical activity, and therefore strong drivers of the current obesity epidemic, it is important that opportunities are sought to assess the impact of environmental changes that are brought about by deliberate policy intention, such as the provision of bicycle pathways, or those that are the result of policies aimed at an entirely different issue, e.g. congestion-charging schemes. In either situation it is also important not only to assess specific behaviours that are likely to be directly affected by the policy change, but also other physical activity behaviours that might be altered as a consequence, and ideally to assess the totality of activity as well. A congestion-charging scheme might impact on car use, with a resultant increase in cycling and walking to work. However, its overall impact on total activity might be neutral if the increases in energy expenditure during transportation to work are compensated for by opposite trends in recreational activity. It is unlikely that policy makers will wish to invest in such detailed analyses of their decisions, particularly when the impact on activity and prevention of weight gain are secondary to the main purpose of their initiatives. It is also unlikely that researchers will be given time to accumulate sufficient pre-change data to be able to determine whether the policy change has had an effect, since most policy initiatives work to the short timetable of the political agenda. Thus, a mixed approach to evaluation is likely to be the most successful, with the incorporation of rapid local measures of specific and total activity into areas likely to be affected by policy changes, with longer-term trend data being obtained from ongoing population surveillance studies. As the introduction to the present paper has indicated, such background data are scarce in the UK and are barely sufficient to be able to describe the current trends in physical activity behaviour, let alone allow for analysis of the impact of societal-level interventions. Efforts to address these deficiencies are clearly long overdue and require as comprehensive a system as that in place for infectious diseases. As inactivity and overweight are the major public health challenges of the 21st century, it is timely to consider whether Victorian public health surveillance systems are up to the task of tracking progress in meeting these new challenges.
How much activity is enough to prevent weight gain?

Given the uncertainty from the observational studies described in the present review and the scarcity of randomised controlled trials, the short answer to the question 'how much activity is enough to prevent weight gain?' is that it is not known. A report from a consensus conference published in 2003 (Saris et al. 2003) has attempted to provide recommendations, concluding from two prospective non-randomised cohort studies among women who had successfully lost weight that there is 'compelling evidence that prevention of weight regain in formerly obese individuals requires 60–90 minutes of moderate intensity activity or lesser amounts of vigorous activity'. Such observational evidence might be considered by some researchers to be less than complete, but when Saris et al. (2003) came to discussing the amount of activity required for the primary prevention of weight gain, they acknowledge that 'definitive data are lacking'. However, on the basis of a selective review they conclude that 'moderate intensity activity of approximately 45–60 minutes per day... is required to prevent the transition to overweight or obesity.' Although the details may be disputed, the most salient points on which most researchers would agree are that there are still no definitive data and that, on the basis of the studies that are currently available, the amount of activity necessary is likely to be substantial.

Whilst further studies are required to address the deficiencies in the evidence base, public health authorities have to make responsible recommendations using whatever evidence is available now. In England the Chief Medical Officer’s recent review on the evidence of the impact of physical activity and its relationship to health (Department of Health, 2004a) very much follows the lines of the Saris et al. (2003) consensus report in respect of weight gain. While questions may be raised about the scientific basis on which the conclusions of the review are based, it may be that the overall conclusion is still sensible. The danger of overplaying the strength of evidence underlying these conclusions is that it may hinder efforts to improve the evidence base and may undermine an approach to prevention if interventions are unsuccessful. The Chief Medical Officer’s overall recommendation is that individuals should accumulate at least five 30 min episodes of moderate activity per week. Although the impact of such a recommendation on the likelihood of weight gain cannot be quantified, it is rational because any increased activity overall is likely to reduce obesity risk, a low proportion of the population currently meet this recommendation and the recommended levels are not so distant from an individual’s everyday experience to be unachievable. Whether they will be adopted remains to be seen, as does their impact on the prevalence of obesity if they are adopted.

References


