

## 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, 2004*b*). 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

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**Abbreviations:** PAEE, physical activity energy expenditure; TV, television.

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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  $\geq 30$  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 securely-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 be 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 future low 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 1. Self-reported physical activity (PA) and weight gain in adults (&gt; 18 years)

Study	n	Baseline age, gender	Selection	Method	PA assessment	Outcome	Size of effect	Confounding
Hu <i>et al.</i> (2003)	50 277	46–71 years, female	Healthy, BMI <30 kg/m <sup>2</sup> in previous 16 years (excluding cancer, CVD and diabetes), USA	Prospective cohort, 6 years follow-up (Nurse's Health Study)	Self-report of PA and sedentary behaviour	Onset of obesity (BMI > 30 kg/m <sup>2</sup> )	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	Age, smoking, alcohol consumption, EI, total fat, glycaemic load, cereal fibre
Koh-Banerjee <i>et al.</i> (2003)	16 587	40–75 years, male	Healthy (excluding CVD, cancer, diabetes), USA	Longitudinal, 9 years follow-up (Health professionals' study)	Self-report, leisure time, last year (biennially)	ΔWC	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	Age, BMI, WC, total energy, alcohol consumption, total PA and change in smoking and in BMI
Macdonald <i>et al.</i> (2003)	898	45–54 years, female	Random sample from an osteoporosis screening programme, around menopause, no HRT, UK	Prospective 6.3 years follow-up	Self-report time in rest, light, moderate and vigorous intensity, PAL calculated	ΔBW	Increase in TV viewing by 20 h/week associated with an increase in waist of 3.0 (95% CI 0.6, 5.4) mm	Age, wt and height, EI or EI difference, smoking
Ball <i>et al.</i> (2002)	8726	18–23 years, female	Apparently healthy (excluding pregnancy, serious medical condition), Australia	Prospective, 4 years follow-up	Self-report, two items (periods of moderate + vigorous ≥20 min at a time per week). Time spent sitting	BMI maintainers (ΔBMI < 5%) or gainers (ΔBMI ≥ 5%)	Change in PAL influenced change in BW explaining 4.4%. Over time, PAL decreased with increasing wt gain ( <i>P</i> < 0.001)	Sociodemographics (education, marital status, occupation, parity, new mothers)
Bell <i>et al.</i> (2001)	2487	20–45 years, male and female	Healthy (excluding pregnancy, BMI <12 kg/m <sup>2</sup> or >60 kg/m <sup>2</sup> , wt change >20 kg/m <sup>2</sup> ), China	Prospective, 8 years follow-up	Interview, work-related PA	ΔBW	Males and females who gained >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	Wt, wt status, height, age, residence, income and education

Wagner <i>et al.</i> (2001)	8865	50–59 years, male	Healthy (excluding history of CHD, cancer or died during follow-up), France and Northern Ireland	Prospective, 5 years follow-up	Self-report last year PA at work, transportation to work and leisure time	$\Delta$ BMI	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 <sup>2</sup> ) $\Delta$ PA not correlated with $\Delta$ BW	Centre, age, marital status, education, work, socio-occupational class, dieting, alcohol, smoking, work PA Age and gender
Rainwater <i>et al.</i> (2000)	539	Mean age 37 years, male and female	Healthy Mexican-Americans (excluding anti-lipids and anti-hypertensive drugs, and diabetes), USA	Prospective, 5 years follow-up	Interview, Stanford 7 d recall	$\Delta$ BW		
Sherwood <i>et al.</i> (2000)	1044	20–45 years, male and female (79%)	Community volunteers participating in a community wt-gain-prevention project, USA	Longitudinal, annual measurements for 3 years	PA history, thirteen items, leisure and occupational activity $\geq 20$ min at a time; four categories; intensity and frequency in each category used	$\Delta$ BW	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	Age, smoking status, education, income and marital status
Schmitz <i>et al.</i> (2000)	2770	18–30 years, male and female	Healthy (excluding pregnant; CARDIA study), USA	Longitudinal, five assessments over a 10-year period	Interview based self-report; frequency and participation in thirteen sport and exercises, no assessment of duration, Outcome exercise units (CARDIA PA history)	$\Delta$ BW	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	Age, education, alcohol intake, parity, smoking, % total EI from dietary fat at baseline and year 7

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.

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

Study	<i>n</i>	Baseline age, gender	Selection	Method	PA assessment	Outcome	Size of effect	Confounding
Ekelund <i>et al.</i> (2005)	739	Median age 53.8 years, male and female	Population-based sample, healthy Caucasians, UK	Prospective, 5.6 years follow-up	PAEE from individually calibrated HR monitoring	ΔBW and ΔFFM (by bio-impedance)	Interaction PAEE × age, PAEE predicted increase in FM in younger subjects who on average gained wt. PAEE positively associated with FM and FFM in older subjects who on average were wt stable	Gender, age, wt or body composition, smoking, dietary fat intake and duration of follow-up
Tataranni <i>et al.</i> (2003)	92	19–70 years, male and female	Healthy non-diabetic Pima Indians, USA	Prospective, ≥4 months follow-up (4 (sd 3) years)	PAEE and PAL from DLW	ΔBW	PAEE and PAL not associated with change in BW	Gender, age, wt or body composition and duration of follow-up

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

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.

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

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.

### *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  $\leq 2$  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 an increase of 1 h in daily recreational physical activity is associated with a  $0.06 \text{ kg/m}^2$  decrease in BMI in girls at 1-year follow-up. Furthermore, they have also shown that in both boys and girls a 1 h difference in sedentary activity or physical activity is associated with a  $0.026\text{--}0.038 \text{ kg/m}^2$  higher BMI at 1-year follow-up (Berkey *et al.* 2000). In a study on the association between TV viewing and subsequent change in body composition, Proctor *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  $0.54 \text{ kg/m}^2$  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  $4.2 \text{ kJ}$  ( $1 \text{ kcal}$ )/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  $\text{O}_2$  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 3.** Self-reported physical activity (PA) and weight gain in children (age  $\leq 18$  years)

Study	<i>n</i>	Baseline age, gender	Selection	Method	PA assessment	Outcome	Size of effect	Confounding
Hancox <i>et al.</i> (2004)	1037	3 years, male and female	Birth cohort in Otago, New Zealand	Longitudinal study, 23 years follow-up	TV viewing (5–11 years: parental report; 13–26 years: self-report)	BMI at 26 years, PAR for $\geq 2$ h TV/d	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/m <sup>2</sup> per h increase in TV viewing), PAR at 26 years indicate that 17% of the overweight can be attributed to watching >2h TV between 4 and 15 years	Gender, childhood socio-economic status, BMI at 5 years and parental BMI
Tammelin <i>et al.</i> (2004)	5706	14 years, male and female	Birth cohort, Finland	Prospective cohort, 17 years follow-up	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	Obesity (abdominal) at 31 years	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))	Maternal BMI, BMI at 14 years, education, occupational PA at 31 years, alcohol consumption, smoking, parity
Berkey <i>et al.</i> (2000, 2003)	11 887	9–14 years, male and female	Nationwide sample, USA	Longitudinal study, 1 year follow-up	Self-reported sedentary activity (TV, video, games), no. of gym classes and recreational activities	Change in BMI  BMI at follow-up	Change in BMI in girls (0.06 (95% CI -0.11, -0.01) kg/m <sup>2</sup> per h increase in daily activity) and in overweight boys (-0.22 (95% CI -0.33, -0.10) kg/m <sup>2</sup> ) BMI: in girls (0.037 (95% CI 0.016, 0.058) kg/m <sup>2</sup> increase per h watching TV per d and 0.028 (95% CI -0.056, -0.00) kg/m <sup>2</sup> decrease per h recreational activity per d); in boys (0.038 (95% CI 0.019, 0.058) kg/m <sup>2</sup> increase per h watching TV per d and 0.026 (95% CI -0.057, 0.004) kg/m <sup>2</sup> decrease per h recreational activity per d)	Race or ethnicity, EI, height growth, Tanner* and menarche history  Race, height growth, Tanner* and menarche history and BMI
Francis <i>et al.</i> (2003)	173	5 years, female	Sample of families (non-representative), USA	Longitudinal, 4 years follow-up	Self-report of tendency towards participating in PA, parental report of TV viewing (only at age 7–9 years)	Change in BMI	Tendency towards PA not related to change in BMI, TV viewing predicted change in BMI in girls from non-overweight families	Parental BMI, snacking, fat intake, BMI at age 5 years and family income
Bogaert <i>et al.</i> (2003)	59	6–9 years, male and female	Volunteer families, Australia	Prospective cohort, 1 year follow-up	Bouchard PA record (parental report)	Change in BMI z-score	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	



Proctor <i>et al.</i> (2003)	106	3–5 years, male and female	Framingham offspring, USA	Longitudinal, 7 years follow-up	Parental report of TV (and video) viewing	SSF, BMI	Children who watched $\geq 3.0$ 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 $< 1.75$ h TV per d ( $P=0.007$ )	BMI or SSF. PA by activity monitor, fat intake, total EI, parental BMI or education
Horn <i>et al.</i> (2001)	198	7.5 years male and female (Grades 1–4)	Sample from two Mohawk communities, Canada	Longitudinal, 2 years follow-up	Self-report of PA (weekly checklist) and TV viewing	SSF	Excessive TV viewing and higher relative PA associated with higher SSF at follow-up in girls. No effect of PA in boys	SSF, community, age,
Kimm <i>et al.</i> (2001)	2379	9–10 years, female	Sample from California and Cincinnati, OH, USA	Longitudinal, 10 years follow-up	Self-reported PA: 3 d diary (including one weekend day)	Change in SSF	PA not related to change in SSF for both races	Age, age at menarche, dietary intake
Davison <i>et al.</i> (2001)	197	5 years, female	Sample (non-Hispanic white) Pennsylvania, USA	Cohort study, 2 years follow-up	Parental report (5 years), self-reported tendency towards PA (at 7 years)	Change in BMI	PA not related to change in BMI	BMI at 5 years, parents' education, family income, and parents' wt status and dietary intake
O'Loughlin <i>et al.</i> (2000)	2318 (1 year sample), 633 (2 years sample)	9–12 years, male and female	Montreal Heart Health Program, Canada	Longitudinal, 1 and 2 years follow-up	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 $> 90$ th percentile	1-year predictors of $> 90$ th percentile of change in BMI: in boys included baseline BMI of $\geq 90$ th percentile (OR 2.66 (95% CI 1.80, 3.94)); in girls included baseline BMI of $\geq 90$ th 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 $> 90$ th percentile of change in BMI: in boys included baseline BMI of $\geq 90$ th percentile (OR 3.26, (95% CI 1.52, 7.01)), no sports outside school (OR 2.14, (95% CI 0.96, 4.77)) and least active (OR 2.18 (95% CI 1.01, 4.71)); in girls only baseline BMI of $\geq 90$ th percentile (OR 2.22 (95% CI 1.02, 4.81))	Age, grade
Mamalakis <i>et al.</i> (2000)	1046	6 years, male and female	Representative sample, Greece	Prospective cohort study, 6 years follow-up	Parental report of three typical days, total time spent on strenuous PA	BMI, SSF, WHR at 9 and 12 years	PA did 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

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  $\leq 18$  years)

Study	n	Baseline age, gender	Selection	Method	PA assessment	Outcome	Size of effect	Confounding
Moore <i>et al.</i> (2003)	103	3-5 years, male and female	Framingham children's study, USA	Longitudinal, annual measurements during 8 years follow-up	Caltrac activity monitor	$\Delta$ BMI, TSF, and SSF	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/m <sup>2</sup> ) 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), 18.5 (sd 0.9), 15.1 (sd 1.1; P < 0.05); SSF (mm): 95.1 (sd 6.8), 94.5 (sd 6.0), 74.1 (sd 7.0; P < 0.05). However, no significant longitudinal associations between change and PA are reported	Gender, age, BMI, amount (h) of TV viewing per d, % energy from fat, parental BMI and education
Truth <i>et al.</i> (2003)	88	8-9 years, female	Normal-wt (<90th percentile wt-for-height and % BF 12-30), USA	Longitudinal, 1 and 2 years follow-up	TEE by DLW PAEE=TEE - RMR; PAL=TEE/RMR	$\Delta$ FM by DXA	No associations between PAEE and PAL and change in FM. TEE predicts a higher change in % BF ( $\beta$ 0.002 (95% CI 0.000, 0.0004))	Time of baseline measurement, ethnicity, Tanner*, FM, group for parental obesity
Wells & Ritz (2001)	26	9-12 months, gender not mentioned	Healthy, born at term, UK	Prospective, follow-up at age 2 years	TEE by DLW adjusted for FFM	FMI by <sup>18</sup> O, skinfold	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	Infant FFM, FMI
Johnson <i>et al.</i> (2000)	115	4-6-11.0 years, female and male	Free of any major illness since birth, USA	Longitudinal, annual follow-up for year 3-5 years	TEE by DLW AEE=0.9 $\times$ TEE-RMR	$\Delta$ FM by DXA	None of the measures of EE predicted change in FM	FM and FFM, age, ethnicity, gender and Tanner stage*
Figueroa-Colon <i>et al.</i> (2000)	47	4-8-8.9 years, female	Normal wt (10th to 85th percentile for BMI), Tanner stage 1, USA	Longitudinal, at 1.6 and 2.7 years follow-up	TEE by DLW PAEE=0.9 $\times$ TEE-RMR	$\Delta$ FM by DXA	PAEE adjusted for FFM was a predictor of FM at first follow-up but not at second follow-up	Age, FM, parental FM and FFM, sleeping EE, follow-up time

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;  $\Delta$ , 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)

Study	Subjects	Intervention	Behaviour change method	Main outcome measure	Size of effect
Burke <i>et al.</i> (2003)	137 couples (274 subjects; women 28.6 (range 18–62) years, BMI $\geq 25$ kg/m <sup>2</sup> : 28%; men 31.4 (range: 20–61) years, BMI $\geq 25$ kg/m <sup>2</sup> : 61%), Australia	<ol style="list-style-type: none"> <li>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</li> <li>Low-level intervention: six printed modules focusing on PA and nutrition (same as 1), mailed every 2 weeks</li> <li>No intervention</li> </ol>	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 <i>et al.</i> (2003)	Seventy schizophrenic patients (male and female) taking an anti-psychotic drug known to induce wt gain (mean age intervention 33.7 (sd 9.2) years, control 34.5 (sd 10.0) years), USA	<ol style="list-style-type: none"> <li>Weekly, 1 h psycho education classes for 16 weeks discussing 2 main modules: 'Nutrition, Wellness, and Living a Healthy Lifestyle' and 'Fitness and Exercise'</li> <li>Standard treatment</li> </ol>	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 <i>et al.</i> (2003)	299 employees (77% male) of municipal services (intervention 43.8 (sd 8.3) years, control 44.0 (sd 9.4) years), The Netherlands	<ol style="list-style-type: none"> <li>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)</li> <li>Written lifestyle information only</li> </ol>	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 ( $\beta$ -0.22 (95% CI -0.47, -0.03), although both groups decreased. No intervention effect on BMI. EE differed at follow-up ( $\beta$ 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 (OR 1.46 (95% CI 0.76, 2.79))
Simkin-Silverman <i>et al.</i> (2003)	535 healthy premenopausal women, aged 44–50 years USA	<ol style="list-style-type: none"> <li>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</li> <li>Assessment-only control condition</li> </ol>	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 (sd 5.2) kg compared with 26% of controls (mean change: 2.4 (sd 9) kg at 54-month follow-up ( $P<0.001$ )). WC decreased significantly more in intervention subjects than in controls (-29 (sd 53) mm v. -5 (sd 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

Polley <i>et al.</i> (2002)	120 pregnant women (BMI >19.8 kg/m <sup>2</sup> , age >18 years, <20 weeks gestation) from a hospital-based clinic serving low-income women, USA	<p>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</p> <p>2. Standard prenatal care</p>	Individual counselling, no behaviour change model specified	Wt gain, wt in relation to IOM recommendations, post-partum wt loss	Among normal-pregnancy-wt women, 58% of the control group exceeded the IOM-recommendation, whereas 33% of the women in the intervention group ( $P < 0.05$ ). The intervention had no effect on average wt gain from pre-pregnancy to delivery, or on postpartum wt loss
Muto & Yamauchi (2001)	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	<p>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)</p> <p>2. No intervention</p>	Group-based, no behaviour change model specified	Wt, BMI, cholesterol, BP, and fasting blood glucose, at 6 and 18 months after main intervention (4 d seminar)	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); $P < 0.001$ ) and at 18 months (intervention -1.0 (95% CI -7.3, 5.3), control 0.5 (95% CI -3.8, 4.8); $P < 0.001$ ). BMI (kg/m <sup>2</sup> ) 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); $P < 0.001$ ). 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

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 ( $\leq 18$  years)

Study	Subjects	Intervention	Behaviour-change method	Main outcome measure	Size of effect
Kain <i>et al.</i> (2004)	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/m <sup>2</sup> , % $\geq$ P95 CDC: 15); control: $n$ 1202 (mean age 10.6 years, BMI 19.2 kg/m <sup>2</sup> , % $\geq$ P95 CDC 11.3); Chile	1. Combined dietary and PA intervention. Diet: trained nutritionist provided educational programme to children (5–11 h 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/d), and extra activities per school 2. No intervention	School-based, no theoretical model stated	BMI, TSF, and WC at 8-month follow-up	Boys: significant effect on BMI (kg/m <sup>2</sup> ; intervention baseline 19.5 (sd 3.7), follow-up 19.5 (sd 3.5); control baseline 18.9 (sd 3.3), follow-up 19.2 (sd 3.1); $P < 0.001$ ) and on WC (mm; intervention baseline 674 (sd 109), follow-up 665 (sd 96); control baseline 646 (sd 93), follow-up 655 (sd 89); $P < 0.001$ ). Girls: no significant effects No effect reported on PA and dietary intake
Dennison <i>et al.</i> (2004)	Children from preschool or day care centre (male and female) intervention: $n$ 43 (age 3.9 (sd 0.07) years, BMI 15.9 (sd 0.3) kg/m <sup>2</sup> ); control: $n$ 34 (age 4.0 (sd 0.10) years, BMI 15.9 (sd 0.2) kg/m <sup>2</sup> ), USA	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	Parent-based, no theoretical model stated	TV viewing (parental report), wt, BMI, and TSF at 6-month follow-up	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
Pangrazi <i>et al.</i> (2003)	606 4th grade students (male and female) from Arizona schools (age 9.8 (sd 0.6) years), USA	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	School-based (teacher-facilitated), no behaviour change model stated	PA (steps per d assessed with pedometer), and BMI at 12-week follow-up (directly after intervention)	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
Caballero <i>et al.</i> (2003)	1074 2nd grade American Indian students (male and female; mean age 7.6 (sd: 0.6) years, mean BMI 18.8 (sd 3.9) kg/m <sup>2</sup> , mean% body fat 32.6 (sd 6.8), USA	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	School-based, no behaviour change model stated	% 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	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 -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

Warren <i>et al.</i> (2003)	213 1st and 2nd grade students (male and female) from Oxford primary schools (age 6-1 (sd 0.6) years, BMI 15.9 (sd 2.1) kg/m <sup>2</sup> , 8% overweight, 2% obese), UK	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	School-based (provided by external educator), based on Social Learning Theory	% Overweight or obese (assessed from BMI reference values), dietary intake (parental report), and PA (self-report) at 14-month follow-up	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
Sallis <i>et al.</i> (2003)	1109 6th-8th grade students (male and female) from San Diego middle schools, USA	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	School-based, based on Cohen's structural, ecological model of health behaviour	BMI, fatty food intake and PA (observation) at 2-year follow-up	In boys, BMI significantly reduced in the intervention group compared with the control group ( $F = 4.60$ , $P = 0.044$ , $d = 0.83$ ). No intervention effect for girls. Intervention schools increased PA over time at a greater rate than control schools (for time $\times$ condition interaction $F = 7.53$ , $P < 0.009$ , $d = 0.93$ ). This effect was stronger for boys than for girls. No intervention effect on fatty food intake
Neumark-Sztainer <i>et al.</i> (2003)	201 9th-12th grade females from Minnesota high schools (age 15-4 s (sd 1.1) year, BMI 26.7 (sd 6.5) kg/m <sup>2</sup> , USA	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)	School-based, based on Social Cognitive Theory	BMI, PA, sedentary activity, fruit and vegetable intake, and soda pop intake, post-intervention and at 3-month follow-up	No change in BMI at either follow-up measurement. Although favourable behavioural changes, no significant difference between the intervention and control condition at either follow-up
Baranowski <i>et al.</i> (2003)	Thirty-five African-American girls (8 years, BMI (kg/m <sup>2</sup> ) intervention 21.1 (sd 4.4), control 26.3 (sd 7.9)), USA	1. GEMS-FFFFP 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)	Community and family-based (pilot-project), based on Social Cognitive Theory	BMI, PA (CSA and self-report), and dietary intake at mean follow-up of 16 weeks	No intervention effect on BMI, dietary intake or PA
Robinson <i>et al.</i> (2003)	Sixty-one African-American girls (8-10 years, BMI (kg/m <sup>2</sup> ) intervention 21.0 (sd 5.4), control 21.6 (sd 5.3)), USA	1. Intervention consisting of (a) providing free 2.5 h dance classes 5 d/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	Community and family-based (pilot-project), based on Social Cognitive Theory	BMI, WC, PA (CSA and self-report), TV use, and dietary intake at 12-week follow-up	No intervention effect of BMI, WC, PA or dietary intake. A reduction in total household TV use in the intervention group, relative to controls

Table 6. (Continued)

Study	Subjects	Intervention	Behaviour-change method	Main outcome measure	Size of effect
McMurray <i>et al.</i> (2002)	1140 students (male and female) from middle schools in North Carolina (12 (sd 1) years; 28.7% had BMI $\geq 30$ kg/m <sup>2</sup> ), USA	<ol style="list-style-type: none"> <li>Exercise only: 30 min aerobic exercise 3 d/week for 8 weeks, taught by their regular PE teacher</li> <li>Education only: information on nutrition, smoking, and exercise during two class periods per week for 8 weeks, taught by a regular classroom teacher</li> <li>Exercise and education: combination of interventions 1 and 2, lasting 8 weeks</li> <li>No intervention</li> </ol>	School-based, no behaviour change model mentioned	BMI, SSF, and systolic and diastolic BP directly after the intervention	No difference in change in BMI between the four study groups. Overall, a significant increase in change in SSF ( $P=0.0001$ ), and the increase in SSF less in the 'exercise and education' group than in the 'education only' and the control group ( $P=0.0001$ ). The changes in systolic and diastolic BP in the intervention groups (remained stable or decreased) different from the control group (increased; $P < 0.0001$ )
Sahota <i>et al.</i> (2001)	636 primary school children (male and female) from Leeds (age 8.4 (sd 0.63) years, 17% $\geq 85$ th percentile for BMI), UK	<ol style="list-style-type: none"> <li>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</li> <li>Normal school curriculum</li> </ol>	School-based, based on the Health Promoting Schools philosophy	BMI, diet and PA at 12-month follow-up	No difference in BMI between the intervention and control group. A 24 h recall showed that intervention children had higher vegetable intakes (weighted mean difference: 0.3 (95% CI 0.2, 0.4)). Further, obese children in the intervention group also showed lower fruit consumption at follow-up. Overall, PA decreased and sedentary behaviour increased over time, but no significant difference between the study groups

SSF, sum of skinfolds; TSF, triceps skinfold thickness; WC, waist circumference; BP, blood pressure; %  $\geq$  P95 CDC, percentage of subjects  $\geq 95$ th percentile of CDC-F; TV, television; FFFP, Fun, Food, and Fitness Project; CSA, Computer Science Applications accelerometer.

explanations for their ineffectiveness. In the absence of clearly successful interventions, the evidence from randomised trials does not currently 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.

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