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TITLE PAGE

Physical activity in the prevention of weight gain: the impact of measurement and interpretation of associations

Dr Paul Remy Jones[1], MBBS, BSc (Hons); Prof Ulf Ekelund[1], PhD.

[1]Department of Sports Medicine, Norwegian School of Sport Sciences, Oslo, Norway.

Corresponding author: Dr Paul Remy Jones

Email address: paul.jones@nih.no

ORCID: Dr Paul Remy Jones 0000-0002-9770-3938

Prof Ulf Ekelund 0000-0003-2115-9267

Postal address: Department of Sports Medicine,
Norwegian School of Sport Sciences,
Ullevål Stadion,
PO Box 4014,
Oslo 0806,
Norway.

Structured abstract

Purpose of review

To what extent do different methods of physical activity measurement and statistical analysis influence the reported associations between physical activity and weight gain?

Recent findings

The obesity epidemic has led to a focus on lifestyle approaches to the prevention of weight gain. Physical activity is one such approach. A number of studies have reported beneficial associations between higher levels of physical activity and weight gain at the population level. However, limitations of physical activity measurement and analytical models in some of these studies is likely to have resulted in overestimation of the strength of association.

Summary

Understanding the limitations of assessment methods and analytical models used in epidemiological research should facilitate more realistic appraisal for physical activity to prevent weight gain at the population level and inform approaches to future research.

Key words

Measurement; methods; modelling; obesity; physical activity; weight gain.

Introduction

Physical activity is known to be beneficial for a range of health outcomes, leading some policymakers, researchers and health professionals to label it a “wonder drug” and “miracle cure” (1,2). Given the burgeoning rates of obesity, physical inactivity and associated health complications seen in recent decades, there has been substantial interest in the potential for physical activity to mitigate increasing body weight for individuals and populations. Maintenance of body weight requires balanced energy intake and expenditure, hence the expectation that increasing energy expenditure through higher levels of physical activity should reduce body weight (3).

Weight regulation in humans is complex, involving genetic, physiological, and behavioural mechanisms (4). The optimum study design with which to infer causation between physical activity and weight regulation independent of caloric intake is a randomised controlled trial (RCT). These tend to be of short duration and require intense researcher supervision (5). Given that body weight increases with age (6), and the high intra- and inter-individual variability in physical activity and energy intake behaviours over time (7,8), long-term trials would arguably provide more robust information. Unfortunately, such trials are not feasible, and we therefore rely on evidence from observational studies. Typically, such studies have a number of limitations, including recall and social desirability bias of participants (9), difficulties accurately measuring both physical activity and energy intake in large populations (10,11••), and a variety of approaches used to analyse the data.

Physical activity may promote weight loss and weight maintenance after weight loss in individuals (12), but do higher levels of physical activity prevent weight gain at a population level? The recent US Physical Activity Guidelines Advisory Committee (PAGAC) Scientific Report systematically reviewed the literature on this topic and concluded that there is a "significant relationship between greater amounts of physical activity and attenuated weight gain in adults" (13•). Here we aim to critically discuss some of the studies included in the PAGAC Scientific Report, focussing on the analytical models used to assess the prospective associations between physical activity and weight gain as well as the impact of different exposure assessment methods.

Analytical models for examining whether physical activity prevents weight gain

Modelling weight as the outcome variable

Current body weight is one of the strongest predictors of future weight. Thus, adjusting for baseline body weight in statistical models is crucial to determine the *independent* association of physical activity with weight gain. Neither an unadjusted prospective model nor unadjusted weight change model (follow-up weight minus baseline weight) can estimate the independent association and likely overestimate the magnitude of association between physical activity and weight gain. Of the 33 studies included in the PAGAC Scientific Report, 14 modelled either follow-up weight or weight change as the outcome independent of baseline weight by including the latter as a covariate in their model (14–27) (see Table). In contrast, 11 studies modelled either follow-up weight or weight change as the outcome, without adjusting for baseline weight as a covariate (28–38). Many studies used either multiple linear regression or logistic regression, and explicitly described the modelling strategy employed and whether baseline weight was included as a covariate. Others used alternative modelling strategies, such as generalised estimating equations and mixed models (39–46). Though it is possible to adjust for baseline weight in these models, the majority of those included in the PAGAC Scientific Report did not provide sufficient detail to determine whether such an adjustment was performed.

Of the 14 studies that examined prospective associations and explicitly reported adjusting for a baseline weight variable in their models, 12 reported at least one statistically significant result with some measure of physical activity (14,16,17,19–27). For example, one study followed 34 079 women for 15 years (22). The authors categorised the participants according to baseline levels of physical activity measured in metabolic equivalent of task hours per week (MET h/wk) (<7.5; 7.5 - <21; ≥21 MET h/wk). They used repeated measures regression to determine prospective associations with change in weight over the subsequent 3 years, updating the baseline and follow-up measurements for every 3-year period. Using the most active group as the referent, the authors reported significantly greater mean differences in weight change for every three-year period for both the least active (+0.12 kg) and middle active groups (+0.11 kg). Repeated measures logistic regression analyses showed increased odds of ≥2.3 kg weight gain over a three-year interval in both the middle active (OR = 1.07) and least active (OR = 1.11) groups compared to the referent. While statistically significant, the clinical relevance of these results appear modest.

The analytical approaches used by the reviewed studies were highly heterogeneous, limiting direct comparison and summarizing the results by a formal meta-analysis, but the strong influence of current weight on future weight is demonstrated by the results of Brien et al. (15). The authors investigated whether baseline physical activity, cardiorespiratory fitness (CRF), and body mass index (BMI) predicted being overweight, obese, or a weight gain of ≥10 kg at 20-year follow-up in a sample of 459 Canadian adults. They found that higher baseline BMI predicted both overweight or obesity at follow-up, independent of baseline physical activity and CRF. In contrast, neither physical activity nor CRF were found to predict either overweight or obesity independent of baseline BMI. Further evidence of the potential mediating effect of current body weight on the association between physical activity and weight gain can be seen in a study examining the risk of becoming overweight or obese in middle-aged and older women (47). The authors found a graded decrease in risk with both crude and multivariable-adjusted models. However, inclusion of baseline BMI as a covariate diminished the hazard ratios for both total leisure-time physical activity and vigorous-intensity physical activity (VPA), including a loss of statistical significance in the VPA model.

Some of the studies included in the PAGAC Scientific Report investigated whether higher levels of physical activity reduced the likelihood of transitioning to being either overweight or obese at follow-up (15,24,30,31,33,43). Though it appears practical to examine the associations between physical activity and well-recognised clinical criteria, there are a number of drawbacks to categorising continuous outcome measures into broad diagnostic categories (48). Also, there is substantial loss of information due to the exclusion of individuals classified as overweight or obese at baseline, which prevents analysis of this important clinical group. Further, the typical statistical approach of logistic regression provides no indication of the magnitude of weight gain. By contrast, those studies that investigate the likelihood of gaining weight of a specific value (e.g., >2 kg) can utilise the information for every individual in the sample population.

Modelling physical activity as the exposure variable

Most commonly, physical activity is only measured at baseline in observational studies that examine associations with health outcomes, including body weight. However, such a model cannot account for the dynamic, reciprocal relationship between changing weight and physical activity levels over time. Data for 4880 United Kingdom Civil Service employees were examined for associations between physical activity and weight change over 10 years (44). The authors categorised the participants into three groups dependent on baseline levels of physical activity. In a cross-sectional model at baseline, they reported differences in BMI between groups. Yet in the prospective model, they found no association between baseline physical activity and BMI change over 10 years. The authors interpreted this finding as the differences reported in the cross-sectional model persisting over time but not increasing. Additional analyses modelled for consistency of physical activity over two-year follow-up, determined by the proportion of assessments at which the participants reported physical activity levels that met national recommendations. These analyses found statistically significant associations with weight change that differed depending on consistency of meeting physical activity recommendations over time.

Unless an appropriate modelling strategy is used that accounts for consistency (or change) in physical activity behaviour over time (22,39), the results may not reflect the reality of a complex, varying behaviour. Whether there is a threshold duration of follow-up above which modelling strategies that fail to adjust for physical activity levels over time are inappropriate, and what that threshold should be, is open to debate. Further, the opportunity to perform analyses that account for temporal changes in physical activity is limited by the availability of the requisite data; many cohorts having measured physical activity at only one, or few, time point(s). Nonetheless, the assumption that physical activity behaviour will remain constant during follow-up of mid- to long-term prospective analyses may be erroneous (49), and the results should be interpreted with caution. We therefore advocate a study design that includes, and an analytical model that accounts for, repeated measures of the exposure and covariates, preferably over an extended period of time.

Modelling change in both the exposure and outcome variables

Some studies, perhaps trying to account for the acknowledged volatility of physical activity behaviour, model change (follow-up measure minus baseline measure) in physical activity as the exposure, and weight change as the outcome. However, whilst these models are likely less prone to confounding, without adjusting for previous physical activity or weight status such an analysis represents a cross-sectional association, rather than examining the temporal association between the exposure and the outcome.

Instead of modelling change in physical activity in units of measurement, such as time spent in moderate- to vigorous-intensity physical activity (MVPA) or MET h/wk, some studies categorised the direction of change in volume of physical activity accumulated between two or more time points. One study grouped participants into tertiles of weekly leisure-time physical activity reported at baseline and allocated each participant an additional category determined by whether participants reported having increased, decreased, or maintained their baseline volume of physical activity at two-year follow-up interview (28). The follow-up interview did not solicit the volume of physical activity that participants had performed, which was instead estimated by the researchers. In their analysis, the authors examined whether the odds of gaining ≥5 kg weight differed dependent on the category of change in physical activity at follow-up, stratified by baseline tertile of physical activity. Those in the highest physical activity tertile at baseline that subsequently increased their physical activity were designated the reference group. The authors reported the highest odds (OR 3.76) for weight gain in men that decreased their physical activity but were in the highest tertile at baseline. Those men in the lowest tertile who subsequently increased their weekly physical activity were more than twice as likely to gain ≥5 kg compared to the referent (OR 2.19). Without quantifying the *change in volume* of physical activity in the analysis, it is difficult to interpret these results as either evidence for higher levels of physical activity preventing weight gain, or that increasing levels of physical activity in those that are less active than average at baseline prevents weight gain. Further, given that there is an absolute limit to which an individual can reduce their volume of physical activity (i.e., 0 minutes/day (min/d)), the magnitude of reduction possible in the most active group is far greater than that in the least active, usually referred to as *regression to the mean*. It is therefore erroneous to assume an equal reduction in physical activity between each group that reported decreasing physical activity at follow-up, and additional insight that could have been gained by contrasting the groups is lost.

Categorising the exposure variable

Implausible or conflicting associations

In a study of postmenopausal women in the US, the authors defined categories of recreational physical activity in MET h/wk, estimating quartiles and creating a fifth category for those who reported 0 MET h/wk (29). They investigated whether baseline physical activity was associated with either 5 - 9 lb weight gain, or ≥10 lb weight gain, over the seven-year follow-up period, and stratified their analyses on whether the participating women were "non-overweight" (<25 kg/m^2) or "overweight" (≥25 kg/m^2) at baseline. The authors reported a 12% lower odds (OR 0.88) of ≥10 kg weight gain for non-overweight women in the highest quartile of physical activity at baseline when compared to the referent (>0 to <4.0 MET h/wk) in the multi-adjusted model. However, using the more conservative weight gain of 5 - 9 lb as the outcome, there was no evidence that those women performing either comparatively more or high absolute amounts of physical activity had a lower odds of weight gain compared with the referent. This suggests that even high levels of physical activity may not fully prevent weight gain. Interestingly, overweight women that reported performing 0 MET h/wk of recreational physical activity had a 29% lower odds (OR 0.71) of 5 - 9 lb weight gain compared to the referent. This result is unexpected, given the typical assumption of a monotonic, inverse relationship between volume of physical activity and a variety of health-related outcomes, including weight gain. There are a number of methodological reasons that may explain these results, including lack of adjustment for multiple comparisons or spurious interactions when categorising two continuous exposure variables that are correlated (physical activity and non-overweight/overweight) (50••). Equally, the lack of association seen for the more conservative 5 - 9 lb weight gain outcome in comparison to ≥10 lb could be due to known issues with cut-point selection when categorising continuous outcomes (48,51).

Dose-response associations

The *biological gradient* (or "dose-response trend"), refers to a change in effect, or outcome, for different levels of exposure (52). With regard to physical activity as the exposure, the PAGAC define *dose-response* as:

> "The relationship between the dose or volume of physical activity and the magnitude of change, if any, in the health outcome or physiologic change."

Delineating whether there is a minimum effective dose of physical activity, a threshold level above which causes harm, or, perhaps more usefully, a granular understanding of the magnitude of response per increment in amount of physical activity performed, is integral to the development of guidelines that are evidence-based, and therefore can constructively inform policy and clinical practice.

It is common for studies investigating dose-response associations between physical activity and health outcomes to categorise the physical activity exposure. Potential reasons for categorising an exposure are numerous (50••), and can be performed in different ways, such as dividing the sample into groups based on volume of physical activity performed in a given time period (16), assigning physical activity scores (23), or defining directions of change in activity levels

between time points (28). Whilst categorisation may be adequate in certain circumstances to provide evidence of a dose-response relationship, there are a number of inherent limitations when categorising physical activity that bias the analysis and should be considered when interpreting the results of such studies.

One potentially inappropriate assumption of categorisation is that of homogeneity of risk within exposure categories (53). For example, one study categorised total weekly MVPA into three groups and examined the prospective associations with odds of ≥2 kg weight gain compared to the referent (0 - 149 min/wk) (19). They found that the group accumulating ≥300 min/wk of MVPA had a 10% reduced odds (OR 0.90) of weight gain, but no difference in odds for the group that accumulated 150 - 299 min/wk. It is assumed that the odds for weight gain in those adults that achieve 149 min/wk of MVPA will be the same as those adults performing no MVPA. Or one could interpret the lack of association between the lower two categories of MVPA exposure as evidence that the odds of weight gain are equal between adults who performed 10 min/wk of MVPA and those that accumulated 295 min/wk, for example. Using few categories of the physical activity exposure, each of which encompasses a broad spectrum of physical activity volumes, results in substantial loss of information, and can obscure variation in risk within categories, and also non-linear or monotonic associations (50••).

Another limitation when categorising a continuous exposure such as physical activity, is that it assumes a step function between groups, and therefore a discontinuity of response in outcome (53,54). The assumption of discontinuity of response can reduce a study's power to detect an association (55). For example, in one study, a discontinuity of response is assumed between the effects of 299 min/wk MVPA on weight gain, and the effects of 300 min/wk. Unless there is a threshold effect of MVPA at 300 min/wk (56), as opposed to a continuous dose-response relationship, this is improbable, and is one potential explanation for the lack of association reported for the group achieving 150 - 299 min/wk MVPA volume as compared to the referent. However, the large sample size casts some doubt on this being the case. Further, given the lack of association between the two lower categories of MVPA volume, and without an overall test of significance (trend test), there is neither evidence for a dose-response association - contrary to the interpretation of the PAGAC - nor evidence against a threshold effect of ≥300 min/wk MVPA.

One approach to categorising physical activity that could lessen the detrimental effects of the homogeneity of risk and discontinuity of response assumptions would be to create a greater number of ordinal categories. However, determining the optimum number of analytical groups would be a challenge given the broad range of time people spend physically active, and also whether or not the questionnaires used to collect the physical activity information are valid to discriminate smaller time intervals. In addition, the direction of association can be highly dependent on the number of categories and interval boundaries chosen, such that the direction of association can be reversed and, hence, easily manipulated (intentionally or unintentionally) (57).

Another approach is to divide the participants into quantiles based on the measure of physical activity, as has been done in some of the studies included in the PAGAC Scientific Report. Though this seems an objective approach, the

categories created are heavily dependent on the distribution of the data, and therefore specific to each sample. This makes it difficult to compare results across studies.

It seems practical to categorise physical activity using cut-points that correspond to those of published physical activity guidelines. However, these categories are predicated on a number of assumptions. Recommended physical activity levels are non-specific for any one health outcome, having been developed as general recommendations to address a number of non-communicable diseases (NCDs) and risk factors through change in one behaviour. Therefore, it is highly unlikely that these broad categories are optimal to investigate associations with singular health outcomes, such as weight gain, and that potentially important insights remain undetected. Further, the recommended levels of physical activity result in categories so broad that significant information loss is inevitable and hence high participant numbers are required.

Given the marked limitations of categorising physical activity and thereby determining dose-response associations with health outcomes, including weight change, perhaps the over-reliance on guideline recommendations to categorise physical activity should be discouraged. There are alternative approaches available that incorporate far more of the original data, such as cubic spline modelling (58). Further discussion of the limitations of categorising continuous exposure variables and suggestions for alternative analytical approaches have been discussed elsewhere (50••,53,55).

Physical activity assessment methods

Of the 33 original studies included in the PAGAC Scientific Report, 30 measured physical activity using self-report methods, including questionnaires (23), activity logs (14), in-person (39) and telephone interviews (41), and used recall lengths that ranged from 24 hours (41) to one year (29). Physical activity was measured in different contexts, for example occupational (42) or leisure-time physical activity (43), and quantified differently, such as time spent at certain intensities (19), time spent walking (18), or weekly running distance (35–37). Even the comprehensiveness of information solicited regarding physical activity varied markedly between included studies, from those that used validated multi-component physical activity questionnaires (44), to those that asked one question only (34).

The limitations of various self-report measures, including proneness to various biases, have been comprehensively discussed in existing reviews (11••,58). Measuring physical activity using devices such as accelerometers can overcome some of the biases inherent in self-report methods, yet comparatively few studies have used objective measures to investigate associations between physical activity and weight gain. Included in the PAGAC Scientific Report, 3 studies measured physical activity objectively using either arm-worn accelerometers (16,17) or pedometers (27). All three studies reported favourable associations between more physical activity and weight gain.

One study examined the associations between change in light-, moderate-, and vigorous-intensity physical activity (min/d) measured over a 12-month period (LPA, MPA, and VPA, respectively), and body weight at 12-month follow-up in 195 young adults (20 - 35 years old) (16). They stratified the physical activity change variables between weekdays

and weekends, and analyses were adjusted for baseline body weight and physical activity measure. The authors reported inverse associations for MPA increased on both weekends and weekdays, and VPA increased on weekends. In their follow-up study, the authors explored associations between two-year change in LPA, MVPA, and MVPA bouts, and body weight at two-year follow-up (17). Inverse associations with body weight were reported for both increased MVPA and MVPA bouts.

Another investigated whether achieving 10 000 steps/d or not was associated with body weight in 1155 adults (26 - 36 years old) over five-year follow-up (27). The participants were grouped into four categories dependent on whether they achieved 10 000 steps/d at baseline or follow-up. Using the group that met the recommended step count at both baseline and follow-up as the referent, the two groups that did not meet the threshold step count at follow-up had greater weight gain.

These studies had a number of limitations. The sample sizes were small and the age ranges narrow, especially in comparison to some of the other studies included in the PAGAC Scientific Report. Two papers analysed the same 195 participants and tested similar, though not identical, hypotheses. It is arguable whether this be considered a “redundant publication” in the strictest sense, but certainly it is not necessary to include both publications with equal importance attributed to each (60). A further issue in both papers is that of potential confounding by reverse causation. Both studies showed that those who increased body weight over the follow-up period, decreased MPA or MVPA significantly. The authors of the third study dichotomised the physical activity exposure into those that did or did not meet a “guideline” amount of >10 000 steps/d, effectively discarding a third of the data and creating other limitations of categorising continuous exposure variables, which have been discussed above. They then further categorised the participants dependent on change in physical activity. Given the limitations of these three studies, the data should be interpreted cautiously.

The findings of more recent papers that used device-based measures of physical activity failed to demonstrate prevention of weight gain. Data on physical activity and body weight in 1710 Norwegian 20 - 85-year-olds, sampled from a national physical activity surveillance programme were collected at two time points, approximately six years apart (61). In prospective models of LPA, MPA, and VPA, there were no associations with either body weight or BMI, adjusted for the respective anthropomorphic variables in either crude or multivariable models.

Another study that measured physical activity using accelerometers in 71 young adults concluded that neither high sedentary time nor low levels of MVPA predicted gain in body weight or body fat over time (62). Neither did they find associations with any anthropomorphic variables in a two-year change model. Similarly, in a study of 1943 adults sampled from five countries, there were no associations between accelerometer-measured MVPA or LPA and weight gain over two-year follow-up (63). These studies challenge the general belief that low levels of physical activity are a strong predictor of weight gain.

Bidirectional association

An increasingly recognised concern when interpreting the association between physical activity and weight gain is reverse causation. Simply put, does past or present weight status, or weight gain, increase the risk of having lower levels of physical activity? Equally, the association may be bidirectional, in that a reduction in physical activity or an increase in weight, increases the risk of further detrimental changes in physical activity or weight status. If the latter is true, in which directional is the association stronger?

Of the 33 studies included in the PAGAC Scientific Report, 31 demonstrate that adults have a tendency to increase their body weight over time. The other two studies either did not provide enough information to determine average weight change, or reported only a trend to reduced weight over a short follow-up (31,41). It appears that, at best, physical activity may mitigate age-related weight gain (22), or that those who are more active at baseline gain weight at the same rate as less active individuals, but from a lower absolute weight (44). Given that almost all included studies report absolute weight gain in spite of physical activity, it is surprising that only four of these studies examine reverse causation (16,17,28,44). One study stratified baseline BMI into quintiles and examined change in leisure-time physical activity over two years as the outcome. The authors reported that men with higher baseline BMI tended to increase their leisure-time physical activity, whereas women with higher baseline BMI tended to decrease their leisure-time physical activity. These results suggest reverse causation, at least in women. In another study, the authors compared the participants that gained at least 5% BMI over five years, with those who either maintained or reduced their BMI. The group that gained weight displayed lower levels of MVPA at five-year follow-up. Similarly, in the other two studies, the group that gained weight between baseline and follow-up reduced either their MPA or MVPA.

Reverse or bidirectional causation between physical activity and weight gain is plausible. In a cohort of children from ALSPAC, Mendelian randomisation was used to assess the causal association between BMI and fat mass index (FMI), and objectively measured physical activity (64•,65). Their results suggest that increased adiposity determined future reduced total physical activity, reduced MVPA, and increased sedentary time. One study, in which physical activity was measured objectively with accelerometers found that body weight at baseline predicted lower time spent in both MVPA and VPA at follow-up, having adjusted for multiple covariates including baseline values of the respective physical activity domain (61). Similarly, in a study that measured sedentary time using heart rate (HR) monitors, the authors found that sedentary time predicted neither body weight, BMI, waist circumference, nor fat mass at follow-up, yet all four anthropometric variables predicted higher amounts of sedentary time at follow-up, having adjusted for baseline sedentary time and a number of other covariates (66). Furthermore, there is evidence to suggest that the *rate* of weight gain is also an important predictor of future physical activity levels (67), and even a threefold stronger magnitude of inverse association when body weight is modelled as the exposure compared to when modelled in the opposite direction (68).

The associations between physical activity and weight gain are complex, likely bidirectional, and further complicated by the difference in accuracy of the exposure and outcome variables. Physical activity is measured imprecisely

compared to weight, and therefore its association to the outcome tends to be underestimated. This is known as *regression dilution bias*. Conversely, when the exposure is measured with more precision that the outcome, the uncertainty of the estimate of the effect size increases, but the association is not underestimated. Therefore, an association is far more likely when modelling body weight as the exposure and physical activity the outcome.

Conclusion

Though physical activity is advocated as an important intervention to prevent weight gain, the considerable methodological and analytical limitations present in the current literature prohibit firm conclusions regarding the magnitude and direction of association. Despite weak associations between physical activity and weight gain, promoting physical activity remains one of the most important public health strategies. High levels of physical activity are consistently associated with reduced risks for many chronic diseases and all-cause mortality (69,70). In addition, high levels of physical activity seem to eliminate the detrimental association between high sitting time and all-cause, cardiovascular and cancer mortality (71,72). Whilst it may not confer a protective effect against weight gain, what is absolutely clear is that physical activity is beneficial for a number of health outcomes, regardless of body weight, and encouraging more active lifestyles remains integral to tackling the global rise in NCDs.

Conflict of interest

Paul Remy Jones and Ulf Ekelund declare that they have no conflict of interest.

Human and animal rights

This article does not contain any studies with human or animal subjects performed by any of the authors.

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Table 1 Characteristics of included studies

Reference	Analytical sample (n)	Age at first assessment (mean ± SD)	Physical activity measure	Outcome	Baseline outcome adjustment (statistical model)
Adair et al. (2011)	3055 100% female	27.1 ± 6.0 years	Self-report Interview	Δweight	Mixed models
Basterra-Gortari et al. (2009)	11 974 59.2% female	40.0 - 48.8 years (depending on sex and level of physical activity)	Self-report Questionnaire	ΔBMI Relative (%) ΔBMI Weight gain ≥5 kg	No
Bea et al. (2010)	122 100% female	56.3 ± 4.3 years	Self-report Questionnaire and activity log 7-day recall	Δweight	Yes
Blanck et al. (2007)	18 583 100% female	60.1 years	Self-report Questionnaire 1-year recall	Weight gain 5 - 9 lbs Weight gain ≥10 lbs	No
Botoseneanu and Liang (2012)	10 314 52.3% female	55.8 ± 3.2 years	Self-report Interview	ΔBMI	Hierarchical linear models
Brien et al. (2007)	459 51.4% female	32.8 ± 9.6 years	Self-report Questionnaire 1-year recall	Transition to overweight Transition to obesity Weight gain ≥10 kg	Yes
Brown et al. (2016)	4881 100% female	20.7 ± 1.4 years	Self-report Questionnaire Weekly	Maintain BMI <25 kg/m^2	No

Chiriboga et al. (2008)	572 48.1% female	47.8 ± 12.3 years	Self-report Telephone interview 24-hour recall	Weight	Linear mixed models
Colchero et al. (2008)	2952 100% female	26.3 ± 6.1 years	Self-report Interview	ΔBMI	Fixed effects regression
de Munter et al. (2015)	23 108 55.2% female	48.3 ± 16.0 years	Self-report Questionnaire 1-year recall	ΔBMI Transition to overweight Transition to obesity	Generalised estimating equations
Drenowatz et al. (2017)	195 48% female	27.8 ± 3.7 years	Device Accelerometer and activity log Worn for 10 days	Follow-up weight	Yes
Drenowatz et al. (2016)	338 46.6% female	27.8 ± 3.7 years	Device Accelerometer and activity log Worn for 10 days	Follow-up weight	Yes
French et al. (2012)	153 61.4% female	41.0 ± 8.8 years	Self-report Questionnaire	Follow-up BMI	Yes
Gebel et al. (2014)	32 087 53.4% female	59.5 ± 9.3 years	Self-report Questionnaire	Weight gain ≥2 kg	Yes
Gradidge et al. (2015)	428 100% female	41.1 ± 5.4 years	Self-report Questionnaire at interview	ΔBMI	Yes
Hamer et al. (2013)	4880 28.5% female	49.3 ± 5.9 years	Self-report Questionnaire	ΔBMI Follow-up BMI	Linear mixed models

Hankinson et al. (2010)	3554 52.5% female	24.5 - 25.2 years (depending on sex and level of physical activity)	Self-report Questionnaire 1-year recall	ΔBMI	Yes
Hillemeier et al. (2011)	689 100% female	32.8 years (normal BMI) 33.4 years (overweight BMI)	Self-report Interview	Transition to overweight/obesity Transition to obesity	No
Kaikonnen et al. (2015)	1715 53.8% female	32.1 years (female) 31.9 years (male)	Self-report Questionnaire	Δweight	Bivariate analysis only
Kelly and Latner (2015)	86 100% female	20.3 ± 2.9 years	Self-report Questionnaire Weekly	Δweight	No
Lee et al. (2010)	34 079 100% female	54.2 years	Self-report Questionnaire Weekly	Δweight Weight gain ≥5 lbs	Yes
MacInnis et al. (2014)	5879 63.8% female	53.6 ± 8.2 years (female) 53.5 ± 8.4 years (male)	Self-report Questionnaire 6-month recall	Follow-up weight	Yes
Moholdt et al. (2014)	19 127 56.4% female	34.9 - 39.4 years (depending on sex and level of physical activity)	Self-report Questionnaire Weekly	Δweight Weight gain ≥2.3 kg	Linear mixed models
Mortensen et al. (2006)	4595 22.4% female	40.7 ± 3.0 years	Self-report Interview question	ΔBMI	Yes
Parsons et al. (2006)	15 006 48.6% female	11.0 years	Self-report Questionnaire	ΔBMI	Multilevel model

Rosenberg et al. (2013)	20 259 100% female	30.4 ± 5.0 - 31.5 ± 5.0 years (dependent on amount walking / strenuous exercise)	Self-report Questionnaire Weekly	Transition to obesity	No
Shibata et al. (2016)	3261 56.5% female	48.3 ± 10.5 years	Self-report Questionnaire	Δwaist circumference	Yes
Sims et al. (2012)	57 735 100% female	50 - 79 years (stratified into three 10-year age groups)	Self-report Questionnaire	Δweight ΔBMI	Yes
Sjösten et al. (2012)	3812 24.7% female	56 ± 2.4 years (age at retirement)	Self-report One question	Δweight Relative (%) Δweight	No
Smith et al. (2017)	1155 57.5% female	31.3 ± 2.7 years (female) 31.7 ± 2.5 years (male)	Device Pedometer Worn for 7 days	Follow-up weight	Yes
Williams and Wood (2006)	12 568 38.2% female	39.8 ± 9.9 years (female) 46.4 ± 10.3 years (male)	Self-report Questionnaire Weekly	ΔBMI Relative (%) Δweight	No
Williams (2007)	8340 26.6% female	39.6 ± 9.7 years (female) 45.3 ± 10.1 years (male)	Self-report Questionnaire Weekly	Δweight ΔBMI	No
Williams and Thompson (2006)	6406 30.2% female	Not reported	Self-report Questionnaire Weekly	Δweight ΔBMI	No