Published in final edited form as:
Int J Obes (Lond). 2013 March ; 37(3): 404–409. doi:10.1038/ijo.2012.58.

Rate of weight gain predicts change in physical activity levels: a longitudinal analysis of the EPIC-Norfolk cohort

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Abstract

Objective—To investigate the relationship of body weight and its changes over time with physical activity.

Design—Population-based prospective cohort study (Norfolk cohort of the European Prospective Investigation into Cancer and Nutrition, EPIC-Norfolk, United Kingdom)

Subjects—25639 men and women aged 39-79 years at baseline. Physical activity was self-reported. Weight and height were measured by standard clinical procedures at baseline and self-reported at 18-month and 10-y follow-ups (calibrated against clinical measures). Main outcome measure was physical activity at the 10-y follow-up

Results—Body weight and physical activity were inversely associated in cross-sectional analyses. In longitudinal analyses, an increase in weight was associated with higher risk of being inactive 10 years later, after adjusting for baseline activity, 18-month activity, sex, baseline age, prevalent diseases, socioeconomic status, education, smoking, total daily energy intake, and alcohol intake. Compared with stable weight, a gain in weight of >2 kg/y during short-, medium- and long-term was consistently and significantly associated with greater likelihood of physical inactivity after 10 y, with the most pronounced effect for long-term weight gain, OR=1.89 (95% CI: 1.30-2.70) in fully adjusted analysis. Weight gain of 0.5-2 kg/y over long term was substantially associated with physical inactivity after full adjustment, OR=1.26 (95% CI: 1.11-1.41).

Conclusion—Weight gain (during short-, medium- and long-term) is a significant determinant of future physical inactivity independent of baseline weight and activity. Compared with maintaining weight, moderate (0.5-2 kg/y) and large weight gain (>2 kg/y) significantly predict future inactivity; a potentially vicious cycle including further weight gain, obesity and complications associated with a sedentary lifestyle. Based on current predictions of obesity trends, we estimate that the prevalence of inactivity in England would exceed 60% in year 2020.

Keywords

physical activity; obesity; weight gain; cohort study; epidemiology

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Conflict of interest All authors declare no conflict of interest.

Supplementary information is available at IJO's website.

The publishers final edited version of this article is available at http://www.nature.com/ijo/journal/v37/n3/full/ijo201258a.html

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INTRODUCTION

Over 400 million adults worldwide were obese in 2005 and projections for the future indicate that by 2015, this prevalence will rise to over 700 million. [1] In 2005, the estimated prevalence of obesity among men and women in England was 21.3% and 24.8%, respectively.[2] Only 35% of men and 24% of women in the UK meet the current physical activity (PA) recommendations for public health as assessed by seven-day recall[3], which points to the scale of the problem. Obesity and physical inactivity impose a high economic burden to the society. The estimated costs of physical inactivity in England are £8.2 billion annually, which does not include the contribution of inactivity to obesity which in itself has been estimated at £2.5 billion annually. [4] Although obesity is multi-factorial, involving genetic [5], environmental [6], social, cultural and psychological components [7], the current epidemic of obesity has been largely attributed to changes in behavioral patterns, including decreased PA and over-consumption of high-fat energy-dense food.[6]

It has been demonstrated that obesity and physical inactivity are closely related, but the direction of this association remains uncertain. It is, however, difficult to determine the direction of causality from observational studies. [8] Despite evidence of an inverse cross-sectional association between PA and body weight or body mass index (BMI) [9-12], results from an increasing number of prospective cohort studies appear statistically stronger for the effect of BMI on future PA, than the effect of activity on future BMI. [13-16] In most of these studies, PA was assessed by self-report[14-16], although one used individually calibrated heart rate monitoring over 4 days. [13] If obesity and increase in weight result in a decline in PA without corresponding decrease in energy intake, a process of self-promoting weight gain may take place [17], initiating a vicious cycle in which further weight gain predisposes to further reductions in PA.

Given the likely bidirectional nature of the relationship between obesity and inactivity and the increasing obesity prevalence, it is possible that population levels of inactivity would increase, although it is uncertain to what extent. In the present study, measures of weight, BMI, PA and confounding factors were available for an adult general population at three time-points; baseline (1993-1997), 18-months, and 10-years. In this longitudinal setting, we sought to: 1) examine the associations of baseline weight and changes in weight over time with 10-year PA independent of baseline activity and 2) model the prevalence of physical inactivity in England in 2020, based on observed associations within the current study combined with the predicted obesity trend nation-wide (Health Survey for England) [18].

MATERIALS AND METHODS

Study population

The data source was baseline assessment and 3 follow-ups of the Norfolk cohort of the European Prospective Investigation into Cancer and Nutrition (EPIC). EPIC was initiated in 1989 as a European multi-centre prospective cohort study to assess the association between diet and the risk of incident cancer. [19] The cohort was recruited between 1993 and 1997 from general practices. Altogether 77630 participants were invited and 30445 completed a detailed Health and Lifestyle Questionnaire. Of these, 25639 participants (11607 men and 14032 women) resident in Norfolk, UK, aged 39-79 years, agreed to attend a health examination at baseline and provided data on anthropometric characteristics and PA. The population response rate was approximately 45%. Since we requested participation of individuals willing to provide detailed information and participate in the follow-up, the identified cohort was not a random population sample. However, it was comparable to the UK national population with respect to many characteristics, such as age, sex, anthropometry, lipids and blood pressure but with a lower proportion of current smokers. [2021] The population was ethnically homogeneous with 99.5% being white Caucasians. We conducted the first follow-up (postal questionnaire) 18 months after baseline (15081 participants responded), the second follow-up (clinical health check) approximately 3 years after baseline (15678 participants responded) and the last follow-up (postal questionnaire) 10 years after baseline (17585 participants responded). Body weight, height and PA were self-reported at the 18-month and the 10-year follow-up assessments. Clinical measures of weight and BMI obtained 3 years after baseline were used to more accurately model their changes over the period of follow-up as well as to better calibrate self-reported height and weight. However, PA at the 3-year follow-up was not analyzed herein because a different questionnaire (EPAQ2)[22] was used for the assessment of PA.

Median time duration between baseline and the last follow-up was 7.6 years, with an inter-quartile range of 7.3-7.9 years.

All participants gave written informed consents. The study was approved by the Norwich District Health Authority Ethics Committee.

Measurements

Anthropometric assessment—Height and weight were measured at baseline and 3-year follow-up by trained nurses with participants dressed in light clothing without shoes following standard clinical procedures. Participants also self-reported their weight and height at all time points. Body mass index (BMI) was calculated as weight in kg divided by height squared (kg/m^2). To increase the precision of self-reported body weight and height (at the 18-month and 10-year follow-ups), we calibrated self-reported measurements against clinical measurements using equations shown in Supplementary material.

Assessment of physical activity—Habitual PA was assessed by the Short EPIC Physical Activity Questionnaire which consists of 4 sections regarding PA during the preceding 12 months. The first section is about PA at work, classified as 4 mutually exclusive categories. The second section is about the amount of time spent in hours per week for summer and winter separately in each of the following activities: walking, cycling, gardening, do-it-yourself, physical exercise and housework. The third section asks whether any of the activities in section 2 were engaged in such a way that it caused sweating or faster heartbeat and, if so, for how many hours during a typical week. The fourth section is about stair climbing. A simple PA-index was devised to allocate participants to 4 ordered categories of overall activity [23]: inactive (sedentary job and no recreational activity); moderately inactive (sedentary job with <0.5 h recreational activity per day or standing job with no recreational activity); moderately active (sedentary job with 0.5–1 h recreational activity per day, or standing job with <0.5 h recreational activity per day, or physical job with no recreational activity); and active (sedentary job with >1 h recreational activity per day, or standing job with >0.5 h recreational activity per day, or physical job with at least some recreational activity, or heavy manual job). The index was validated against a combined heart rate and movement sensor (with individual calibration) in 2000 participants from 10 European countries (representative of the EPIC-population with respect to age and sex) and pooled estimate of the correlation between self-reported and objectively assessed energy expenditure was r=0.33. [24] We also showed a high repeatability of the index (weighted kappa = 0.6, p<0.001). [23]

Assessment of other covariates—At baseline, data on personal medical history, socioeconomic status, smoking history, educational level, alcohol intake and daily energy intake were collected by self-report. Personal medical history was assessed at baseline using the question: ‘Has a doctor ever told you that you have any of the following?’ followed by a list of diseases including myocardial infarction, stroke, diabetes mellitus type 2 and cancer. Smoking history was recorded as never-smoker, former smoker, and current smoker. Social class and educational status were classified as described previously. [2526] Alcohol intake was calculated from questions on amount and type of usual alcohol intake for each day of the week (g/week). Daily energy intake (kJ /day) was estimated using Food Frequency Questionnaire (FFQ). [27]

Statistical analysis

All analyses were conducted using STATA version 11.2 (STATA Corp, College Station, Texas). A p-value <0.05 was considered statistically significant. Possible effect modification by sex was investigated by introducing an interaction term to the regression models. Since we found no significant effect modification by sex, the analyses were not stratified by sex. All associations were also analyzed after exclusion of participants with chronic diseases (myocardial infarction, stroke, diabetes mellitus type 2 and cancer). As neither the magnitude, nor significance of the examined associations were materially altered in these sensitivity analyses, we present the results including all participants, but with adjustment for the presence of these diseases.

Cross-sectional associations—Logistic regression was conducted to calculate ORs with 95% CI for being inactive with weight as predictor. We collapsed the PA outcome variable into Inactive (levels 1 and 2) and Active (levels 3 and 4). All cross-sectional models were reanalyzed with BMI instead of weight. Mixed logistic regression models with random effects were used to allow for repeated measurements.

Two models with distinct levels of adjustment were chosen according to the availability of information on specific covariates and biological importance to the associations of interest. Model 1 was adjusted for sex and age. Model 2 was additionally adjusted for smoking, socioeconomic status, educational level, personal medical history of the following diseases: myocardial infarction, stroke, diabetes mellitus type 2 and cancer, estimated daily energy intake and alcohol intake.

Longitudinal associations—Longitudinal changes in weight and BMI during the follow-up period were calculated in three overlapping time intervals (short-, medium-, and long-term) using generalized linear models thereby taking into account changes in those variables over time for every participant. We dichotomized the PA outcome variable into Inactive (levels 1 and 2) and Active (levels 3 and 4) and used logistic regression to analyze its association with weight and BMI. We modeled activity at the 10-year follow-up as outcome and weight change as ordered exposure variable with 4 categories in separate models: weight loss (>0.5 kg/y), maintained weight (±0.5 kg/y, reference), small weight gain (0.5-2 kg/y) and large weight gain (>2 kg/y). All models were additionally adjusted for baseline activity and activity at the 18-month follow-up to allow for the effect of weight change to be separated from activity at these time points. A non-parametric test for trend (an extension of Wilcoxon rank-sum test) was performed to examine the trend of continuous variables across the categories of activity.

We repeated the analyses with BMI and BMI-change as exposure. We used those estimates and BMI-trend data from the Health Survey for England [18] to predict the prevalence of physical inactivity in 2020 by applying Monte Carlo simulations.

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RESULTS

Descriptive characteristics

Supplementary table 1 summarizes baseline characteristics of participants by PA category for men and women. The proportion of active participants was 21.6% and 15.3% among men and women, respectively. Across 4 categories of PA, there was a strong linear trend in weight, BMI, age, energy intake and alcohol intake. Active participants were less often smokers and were less likely to report presence of chronic diseases.

Weight, BMI and the proportion of obese participants substantially increased over time (Table 1). The proportion of inactive participants rose at each follow-up in men (from 30.9% at baseline to 36.0% at the 10-year follow-up) and women (from 30.5% at baseline to 37.2% at the 10-year follow-up). The proportions of moderately inactive, moderately active and active participants were lower at the last follow-up, compared with baseline in both sexes.

Cross-sectional associations

We observed an inverse cross-sectional relationship between weight and activity at baseline and both follow-ups. Every 1-kg greater weight at baseline was associated with 1.6% (OR=1.016, 95% CI: 1.014-1.019) increased likelihood of physical inactivity (Table 2; Model 1- adjusted for age and sex). This association did not materially change after adjusting for additional confounders (Model 2 -additionally adjusted for smoking, socioeconomic status, educational level, personal medical history of the following diseases: myocardial infarction, stroke, diabetes mellitus type 2 and cancer, estimated daily energy intake, and alcohol intake). Same pattern was present for the 18-month and the 10-y follow-up. Using mixed logistic regression models with random effects to analyze the cross-sectional relationship for all available data across all assessment points, we observed somewhat higher odds of inactivity for every 1-kg difference in weight (OR=1.020, 95% CI: 1.017-1.023, in fully adjusted model).

Longitudinal associations

Participants on average gained 0.21±1.83 kg per year in weight, corresponding to a gain of 0.23±1.32 kg/m^2 per year in BMI. We analyzed weight change, both as a continuous and as an ordered exposure variable with 4 levels (Table 3) and PA at the 10-year follow-up as the outcome. In the continuous analyses, a between-individual difference in weight gain of 1kg/y over 10y was associated with significantly higher odds of being inactive, OR= 1.107 (95%CI: 1.037-1.183), which corresponds to OR=1.022 (95%CI: 1.008-1.036) for every 0.21kg/y difference (the population average). Those who gained 0.5-2 kg/y over 10 y (moderate weight gain) had 30.5 % greater odds of being inactive compared with their counterparts with stable weight (±0.5kg/y), OR= 1.255 (95% CI: 1.113-1.415) after full adjustment. Large weight gain over 10 y (>2 kg/y) was more strongly associated with future inactivity, OR=1.878 (95% CI: 1.304-2.704) in fully adjusted model. When weight change between baseline and the 3-year and between baseline and the 18-month follow-up were modeled as exposures in separate models, similar patterns of associations with inactivity were observed. Large increase in weight (>2 kg/y) was consistently and significantly related with higher odds of inactivity. The magnitude of the association increased with the duration of weight gain. The association for moderate weight gain (0.5-2 kg/y) over 3y became non-significant after adjustment in Model 2. Sensitivity analyses were performed with additional adjustment for baseline weight and the associations did not change.

The parallel analyses using BMI and changes in BMI as exposures resulted in associations similar to those from the models with weight and weight change. Overlaying observed associations between BMI and inactivity (Supplementary table 2) on the predicted BMI distribution for England in year 2020 [18], projects the national prevalence of inactivity to be 66.9 % (95% CI: 66.6%-67.2%) and 59.5% (95% CI: 59.1%-58.9%) among women and men, respectively, as assessed by Monte Carlo simulations (Supplementary table 3) and assuming that no effective interventions would be applied.

DISCUSSION

Principal findings

The results from this prospective population-based cohort study suggest that greater gain in weight over time is a significant determinant of future physical inactivity independent of weight and activity at baseline. This association was observed for increases in weight over short- (18 months), medium- (3 years) and long-term (10 years). Statistically significant effects were consistently present for the large weight gain category (>2 kg/y), with the highest magnitude during long-term weight gain. Moderate weight gain (0.5-2 kg/y) over long term was materially associated with future inactivity after full adjustment. Given the current predictions of obesity trends in England[18] and our observations of the relationship between BMI and inactivity, the prevalence of inactivity will exceed 60% by 2020.

Comparison with other studies

Results from several studies have indicated that greater weight and BMI at baseline are significantly associated with lower levels of PA [14-1628] and with more time spent sedentary [13] at follow-up. Our findings corroborate these observations suggesting that weight and weight gain predict lower levels of future PA. Nevertheless, the available evidence of the direction of the relationship between weight gain and activity is heterogeneous. Many studies have demonstrated that low PA is weakly associated with future weight gain. [29] A recent review [30] of 18 cohort studies examining the association between PA and gain in weight or BMI concluded that PA is not associated with subsequent weight gain. In addition, the results from several studies suggested that maintaining high level of activity over a long period was associated with smaller concurrent gains in weight, BMI and waist circumference. [3132]

Possible explanations and biological plausibility of the findings

Although the finding of greater weight gain predicting future inactivity may seem counterintuitive, it is biologically plausible as various symptoms resulting from overweight or obesity (musculoskeletal problems, exhaustion, sweating and dyspnea) become more pronounced with greater overweight which in turn may hinder PA. Since this study was observational, causality is difficult to infer. Yet, several studies have demonstrated an increase in PA after bariatric surgery in obese patients which supports a possible causal relationship.[33-36] Nevertheless, PA in these studies was self-reported and only one study confirmed that objectively measured changes in PA are much smaller than self-reported ones.[37] Given these limitations and small sample sizes in these studies, the causal nature of the association remains unclear. Our findings are supported by the results of the experimental study by Levine *et al.* [38] which showed a decrease in PA after 8 weeks of overfeeding by 1000 kcal/day. Moreover, we demonstrated that small to moderate increase in weight of 0.5-2 kg/y over, medium- and long-term significantly determines future inactivity which is in line with the findings of Christiansen *et al.*[17] showing that even small changes in activity which occur as a feedback after weight gain, may destabilize the weight-regulation system, thereby leading to self-promoting weight gain and vicious cycle of inactivity and obesity.

Our findings could be interpreted in the light of energy balance theory. It has been shown that most people maintain their weight in a narrow range over time despite varying levels of

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activity and energy intake. [17] However, this regulation fails if an energy imbalance of only 1% occurs. [17] Energy imbalance leading to self-promoting weight gain may result from increased energy intake, decreased activity or both. Weight gain leads to increased basal metabolic rate and has feedback effects on activity (decrease) and energy intake (increase, to compensate for increased amount of tissue). Christiansen *et al.* described various statistical models which allow estimation of the conditions under which weight gain becomes self-promoting and concluded that the small effects cannot be measured by currently available methods. [17] Therefore, our observation that higher weight is associated with lower future PA may be explained by counteracting feedback on PA. If such feedback is not accompanied by a reduction in energy intake, a self-promoting process leading to weight gain occurs. However, the factors which determine whether weight gain will result in a feedback process that disrupts the stability of weight regulation remain elusive.

Strengths and limitations of the study

The present study has several strengths. First, it has a longitudinal design with a prospective data collection and long-term follow-up with repeated assessments of PA, weight and other variables. Second, sample size is large, which increases generalizability of the findings to the middle-aged and older Caucasians and further lowers the possibility that the observed associations have occurred by chance. Third, we used an estimate of PA which has been validated against an objective criterion. [22] Fourth, where self-reported weight and height were used, these were calibrated against clinical measures.

However, the following limitations must be considered. Error associated with PA assessment is substantial when self-report methods are used. Further, true activity level is of a continuous nature but was expressed here in categories and it is likely that there is variation in activity within each category and that some participants have been misclassified. The misclassification of PA, which was analyzed as outcome in this study, may increase the standard error of the estimates thus making the results statistically non-significant. [8] However, the large sample size and repeated measurements of both weight and activity compensate for some of the precision lost due to random measurement error. In addition, we adjusted the analysis for several potential confounders, including total energy intake although residual confounding by dietary factors may still persist.

Non-response and loss to follow-up could have introduced selection bias. Around half of the participants reported their PA at the 18-month follow-up and 61.0% of the baseline cohort reported PA at the 10-year follow-up, but only 9310 participants (36.3%) did so at baseline and both follow-ups. In addition, those who did not attend the 10-year follow-up were older, less active, less educated, more often current smokers and had slightly higher weight at baseline than 10-year attendants. However, such differences were not observed with respect to response at the 18-month follow-up (data not shown). It is also possible that attrition for a certain category of baseline PA was different for participants who gained weight and those who did not, but the consistent inverse cross-sectional association between PA and weight status at all time points, as well as the consistent cross-sectional associations between baseline PA and weight and BMI analyzed with respect to attendance status at follow-ups indicate that such selection bias is unlikely. No difference in short-term weight gain was found between participants who attended and those who did not attend the 10-year follow-up.

Predictions of future inactivity

Being obese throughout the entire follow-up (OR=1.59, 95% CI: 1.34-1.88) or becoming obese between baseline and the 10-year follow-up (OR=1.41, 95% CI: 1.17-1.70) was associated with greater likelihood of being inactive compared to remaining in the non-obese

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category (data not shown). According to the report by the National Health Forum [18] , which was based on the data from the Health Survey for England 2007, the trend of obesity prevalence in the period between 2007 and 2020 is expected to increase in adults aged 20-65 years, from 26% to 37% in men and from 23% to 30% in women, which does not include the predicted substantial surge in the proportion of morbidly obese people (BMI> 40 kg/m^2). These estimates are modeled based on the increase in the prevalence of obesity from 1993 to 2007. Correspondingly, the proportion of people with normal BMI is expected to have a downward trend, whereas the proportion of overweight people is predicted to remain stable.[18] The report also predicts a considerable rise in the incidence of coronary heart disease, stroke and hypertension as well as a dramatic 4-fold elevation in the incidence of type 2 diabetes between 2007 and 2046 in both sexes aged 40-60 years.[18] According to the Health Survey for England 2004, there was an increasing trend in the proportion of adults meeting PA-recommendations for public health between 1997 and 2004, with the percentage rising from 32% to 37% in men and from 21% to 25% in women. [3] However, given the limitations of subjective assessment of PA, the observed trend can reflect an increase in social desirability bias rather than a true increase in activity at the population level. Our projections suggest an increase in the population prevalence of inactivity, based on the expected increase in obesity rates. Overlaying the association between BMI and inactivity on the predicted BMI distribution for England in year 2020 [18], we predict the national prevalence of inactivity to be 66.9 % (95% CI: 66.6%-67.2%) and 59.5% (95% CI: 59.1%-58.9%) among women and men, respectively, as assessed by Monte Carlo simulations. Consequently, the epidemic of obesity will pose a huge challenge to the National Healthcare Service and enormous financial costs to the society if adequate lifestyle interventions are not implemented at the population level urgently.

Future research and conclusions

With obesity reaching epidemic proportions, knowledge of modifiable risk factors for weight gain is essential for public health interventions. Physical inactivity and obesity seem to have independent effects on morbidity and mortality, but there may be dependent effects, which needs further investigation. [39,40] Irrespective of our findings, there are several well-documented health benefits from a lifestyle that includes regular PA. Thus, it is essential to identify factors that are associated with increased PA and the factors that prevent weight gain and subsequent obesity. Randomized controlled trials (RCTs) could clarify the causal association between weight gain and PA. However, several issues (e.g. required duration of follow-up, adherence to increased activity and large sample size) hamper the feasibility of such RCTs. Therefore, future research warrants large-scale observational studies with repeated measurements of objectively measured PA and body composition to address the direction of the association between PA and weight gain. Better control over confounders, especially dietary factors, and an analysis of preceding and subsequent changes in PA, weight and body composition is also necessary to characterize the direction of the association.

In conclusion, our results suggest that weight gain over time significantly predicts future physical inactivity. Reconsidering physical inactivity as a result of increased weight gain that potentially leads to a self-promoting process and gives rise to obesity could be a necessary step in formulating public health measures which target prevention of obesity. The underlying rationale in such recommendations is that small increases in activity may prevent a self-promoting process and the development of obesity.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

We are grateful to all the volunteers who devoted their valuable time to participate in the EPIC-Norfolk study. We would also like to thank Amit Bhaniani for his assistance with data management. EPIC-Norfolk is financially supported by program grants from Cancer Research Campaign and Medical Research Council with additional support from the Stroke Association, British Heart Foundation, Department of Health, Food Standards Agency and the Wellcome Trust. Rajna Golubic is financially supported by a scholarship from the Gates Cambridge Trust.

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Table 1

Body weight, BMI and physical activity categories at baseline and follow-ups; the EPIC-Norfolk study

Variable	Baseline	Follow-up 18 months	Follow-up 3 y	Follow-up 10 y
Men				
N	11607	5586	6582	6773
Weight [1], mean (SD), kg	80.38 (11.5)	80.19 (11.3)	81.38 (11.54)	81.79 (11.9) **
BMI[1], mean (SD), kg/m^2	26.52 (3.3)	26.49 (3.3)	26.88 (3.34)	27.20 (3.5) **
N (%) obese[2]	1550 (13.4)	635 (12.3)	998 (15.19)	1229 (18.9) **
Physical activity category [1]				
N (%) inactive	3586 (30.9)	1981 (35.5)		2441 (36.0)
N (%) moderately inactive	2858 (24.6)	1408 (25.2)		1565 (23.1)
N (%) moderately active	2660 (22.9)	1105 (19.8)		1397 (20.6)
N (%) active	2502 (21.6)	1092 (19.6)		1370 (20.2) *
Women				
N	14032	7252	8448	8874
Weight [1], mean (SD), kg	67.95 (11.8)	67.34 (12.5)	68.69 (11.84)	68.78 (12.0) **
BMI[1], mean (SD), kg/m^2	26.23 (4.4)	25.97 (4.6)	26.53 (4.37)	26.46 (4.4) **
N (%) obese[2]	2375 (17.0)	999 (15.0)	1564 (18.54)	1659 (19.7) **
Physical activity category [1]				
N (%) inactive	4277 (30.5)	2535 (35.0)		3304 (37.2)
N (%) moderately inactive	4493 (32.0)	2489 (34.3)		2651 (29.9)
N (%) moderately active	3117 (22.2)	1385 (19.1)		1678 (18.9)
N (%) active	2147 (15.3)	843 (11.6)		1241 (14.0) *

[1] Test for trend was conducted to determine whether body weight and BMI significantly differ by follow-up status. X^2-tests were performed to assess the differences in proportions of participants in each physical activity category according to follow-up status. Information on physical activity at the 3-year follow-up was assessed by a different questionnaire and these results are not shown in this paper.

[2] Obesity was defined as BMI>30kg/m^2.

* p<0.01;

** p<0.001

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Table 2

Cross sectional associations from 4 separate analyses: odds ratios (95% CI) for being physically inactive associated with 1kg difference in weight; the EPIC-Norfolk study

	Model 1		Model 2	
	N	**OR[1] (95% CI)**	**N**	**OR[1] (95% CI)**
Baseline	25608	1.016 (1.014; 1.019)	24460	1.015 (1.012; 1.018)
Follow-up 18 months	11824	1.012 (1.009; 1.016)	11513	1.015 (1.009; 1.016)
Follow-up 10 y	12144	1.015 (1.011;1.017)	10103	1.014 (1.010;1.018)
All time points: mixed logistic regression model with random effects[2]	25627	1.019 (1.016; 1.022)	24714	1.020 (1.017; 1.023)

Model 1 was adjusted for age at baseline or at particular follow-up and sex

Model 2 was additionally adjusted for socio-economic status, education, smoking status, diagnosis of stroke, myocardial infarction, diabetes mellitus type 2 and cancer, total daily energy intake, daily alcohol intake

Outcome variable was dichotomized (active and inactive).

[1]All p-values were <0.001

[2]Estimates were obtained from mixed logistic regression model with random effects taking into account repeated measurements of exposure, outcome and covariates in the same model. Information on dietary covariates was not available for the 18-month and the 10-y follow-up. In the mixed model we considered total daily energy intake and daily alcohol intake from baseline and from the second health check which took place 3 y after baseline.

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Table 3

Categories of weight change and odds ratios (95% CI) for being physically inactive at the 10-year follow-up

	Model 1		Model 2	
Period and weight change category	**N**	**OR (95% CI)**	**N**	**OR (95% CI)**
From baseline to 18-month follow-up	8620		8364	
Continuous weight change (kg/y)	8620	1.000 (0.982; 1.019)	8364	0.996 (0.978; 1.015)
Lost > 0.5 kg/y	2613	1.099 (0.955; 1.265)	2532	1.092 (0.946;1.260)
Maintained weight (±0.5 kg/y)	2082	1.0 reference	2029	1.0 reference
Gained 0.5-2 kg/y	2671	1.056 (0.917; 1.216)	2587	1.026 (0.888; 1.185)
Gained>2 kg/y	1254	1.236 (1.042; 1.467)	1216	1.172 (0.985; 1.356)
From baseline to 3-y follow-up	7819		7597	
Continuous weight change (kg/y)	7819	1.053 (1.005; 1.103)	7597	1.042 (0.993; 1.091)
Lost > 0.5 kg/y	1272	0.982 (0.802; 1.202)	1241	1.120 (0.951; 1.319)
Maintained weight (±0.5 kg/y)	2997	1.0 reference	2874	1.0 reference
Gained 0.5-2 kg/y	3057	1.147 (1.012; 1.300)	2982	1.119 (0.985; 1.272)
Gained>2 kg/y	513	1.487 (1.184; 1.867)	500	1.406 (1.113; 1.777)
From baseline to 10-y follow-up	9267		8987	
Continuous weight change (kg/y)	9267	1.133 (1.063; 1.209)	8987	1.107 (1.037; 1.183)
Lost > 0.5 kg/y	1198	1.073 (0.923; 1.248)	1162	1.050 (0.899; 1.225)
Maintained weight (±0.5 kg/y)	5327	1.0 reference	5175	1.0 reference
Gained 0.5-2 kg/y	2564	1.305 (1.161; 1.467)	2481	1.255 (1.113; 1.415)
Gained>2 kg/y	178	2.089 (1.468; 2.967)	169	1.878 (1.304; 2.704)

Model 1 was adjusted for: age, sex, physical activity at baseline, physical activity at 18-months follow-up Model 2 was additionally adjusted for: socio-economic status, education, smoking status, diagnosis of stroke, myocardial infarction, diabetes mellitus type 2 and cancer, total daily energy intake, daily alcohol intake Each exposure was entered into a separate model.