

Why even active people get fatter—the asymmetric effects of increasing and decreasing exercise.

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Abbreviations: BMI: body mass index

Keywords: Exercise, running, aging, body mass index, regional adiposity

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Short title: Why even active people get fatter

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Background: Public health policies for preventing obesity need guidelines for active individuals who are at risk due to exercise recidivism.

Methods: Changes in adiposity were compared to the running distances at baseline and follow-up in men and women whose reported exercise increased (N= 4,632 and 1,953, respectively) or decreased (17,280 and 5,970, respectively) during 7.7 years of follow-up.

Results: Per Δ km/wk, decreases in running distance caused over four-fold greater weight gain between 0-8 km/wk (slope \pm SE, males: -0.068 ± 0.005 kg/m 2 , females: -0.080 ± 0.01 kg/m 2) than between 32-48 km/wk (-0.017 ± 0.002 and -0.010 ± 0.005 kg/m 2 , respectively).

In contrast, increases in running distance produced the smallest weight losses between 0-8 km/wk and statistically significant weight loss only above 16 km/wk in males and 32 km/wk in females. Above 32 km/wk (30 kcal/kg) in men and 16 km/wk (15 kcal/kg) in women, weight loss from increasing exercise was equal to or greater than weight gained with decreasing exercise, otherwise weight gain exceeded weight loss. Substantial weight gain occurred in runners who quit running, which would be mostly retained with resumed activity.

Conclusion: Public health recommendations should warn against the risks of irreversible weight gain with exercise cessation. Weight gained due to reductions in exercise below 30 kcal/kg in men and 15 kcal/kg in women may not be reversed by resuming prior activity. Current IOM guidelines (i.e., maintain total energy expenditure at 160% of basal) agree with the men's exercise threshold for symmetric weight change with changing exercise levels.

To prevent unhealthy weight gain, current public health guidelines recommend including sufficient physical activity to elevate total energy expenditure to 160-180% of resting metabolic rate [1,2]. One method of achieving this goal is by walking 60 to 90 minutes per day [1].

Approximately one quarter of Americans report exercising regularly [3]. However, other obligations, changing priorities, and waning motivation often interfere, causing disruptions in the amount performed [4-6]. An informed decision on whether and how to modify physical activity to accommodate these challenges requires their consequences be understood, i.e., the long-term effects of changing activity and whether they are reversible. Randomized, controlled clinical trials show increasing exercise, with or without dieting, causes weight loss [7-12], but their limited sample size, duration, and training dose usually preclude their being used to deduce the dose-response relationship to long-term weight. Moreover, prior studies have not specifically tested whether the effects of increasing and decreasing exercise levels are symmetric, i.e., produce opposite but otherwise equal changes in weight.

This report assesses the relationships of specific starting and ending levels of vigorous exercise to long-term changes in adiposity in a large cohort of men and women. These relationships are used to create dose-response curves directly analogous to those produced from cross-sectional data. The effects of increasing and decreasing activity are compared to assess whether weight gain during a hiatus in activity can be atoned for by resuming exercise, and whether their differences might explain the minimum exercise level required to maintain healthy weight.

Methods

A two-page questionnaire, distributed nationally at races and to subscribers of the nation's largest running magazine (Runners' World, Emmaus PA), solicited information on demographics (age, race, education), running history, weight history, diet (vegetarianism and the current weekly intakes of alcohol, red meat, fish, fruit; vitamin C,

and vitamin E only), current and past cigarette use, prior history of heart attacks and cancer, and medications for blood pressure, thyroid, cholesterol or diabetes. The Lawrence Berkeley Laboratory and University of California Berkeley Committees for the Protection of Human Subjects approved the study protocol and all participants provided informed consents.

Changes in body mass index (BMI) were calculated as the changes in weight in kilograms between the first and second survey divided by the square of the average height from the two surveys in meters. Changes in waist circumference reflect changes in visceral fat [9], which is preferentially reduced by negative energy balance [9]. Self-reported height and weight from this questionnaire have been found previously to correlate strongly with their clinic measurements (unpublished correlation in 110 men were $r=0.96$ for both). Self-reported waist circumferences are somewhat less precise as indicated by their correlations with self-reported circumferences on a second questionnaire ($r=0.84$) and with their clinic measurements ($r=0.68$). Self-reported weekly distance run had a test-retest correlation of $r=0.89$.

Statistics Our approach is to estimate the predicted weight change when running distance is increased from the lower to the upper bounds of predetermined intervals (in this report 6 intervals), and separately, when running distance is decreased from their upper to their lower bounds (Figure 1). We postulate that an individual's total expected Δ BMI is the sum of the predicted Δ BMIs for intervals that lie between their baseline and follow-up surveys, including their fractional portions (Figure 1). Multiple regression analyses were used to calculate the predicted Δ BMI for the six intervals, and included adjustments for

age and length of follow-up. These analyses use the runners' reported Δ BMI as the dependent variable, and their ages, years between surveys, and the total and fractional portions of the six distance intervals as the dependent variables. Specifically, let a_i and b_i designate the baseline and follow-up running distances respectively for individual "i" and c_j and d_j are the lower and upper bounds of interval "j", $j=1,2,\dots,6$ (Figure 1). The portion of interval j that includes the runners change in running distance is 0 if $\max(a_i, b_i) < c_j$ or $\min(a_i, b_i) > d_j$. Otherwise, it is given by the formula:

$$((\min(d_j - \min(a_i, b_i), \max(a_i, b_i) - \min(a_i, b_i)) - \max(c_j - \min(a_i, b_i), 0)) / (d_j - c_j) * \text{sign}(b_j - a_j)),$$

where min and max refer to the minimum and maximum of the arguments, and sign is whether the argument's value is positive or negative. The fitted regression coefficients are divided by the km/wk width of its corresponding interval to estimate the Δ BMI for a one-km/wk increase or decrease in running distance. Smaller intervals were chosen below 16 km/wk than above because prior cross-sectional analyses suggested greater nonlinearity there [13,14].

Results

Eighty percent of the 54,956 participants of the National Runners' Health Study provided follow-up information or were deceased. These included 4,632 men and 1,953 women whose running distance increased two or more km/wk, and 17,280 men and 5,970 women whose running distance decreased two or more km/wk, who were age eligible, nonvegetarian, nonsmokers who did not take medications for diabetes or thyroid conditions. Runners who increased and decreased their running distance had similar baseline BMI (mean \pm SD, males: 23.89 ± 2.71 vs. 23.83 ± 2.63 kg/m 2 , females: 21.17 ± 2.41 vs. 21.25 ± 2.41 kg/m 2) and waist circumferences (males: 84.25 ± 6.15 vs. 84.35 ± 6.09 cm, females: 68.47 ± 6.82 vs. 68.59 ± 6.59 cm), but differed slightly by age (males: 43.63 ± 10.05 vs. 44.78 ± 10.47 y, females: 37.79 ± 9.09 vs. 38.15 ± 9.90 y), and duration of follow-up (males: 7.37 ± 1.82 vs. 7.94 ± 1.76 y, females: 7.26 ± 2.08 vs. 7.66 ± 2.00 y).

Figure 2 presents the estimated increase in BMI and waist circumference corresponding to each 1-km/wk change in running distance when going from 0 to 7.9, 8 to 15.9, 16 to 31.9, 32 to 47.9, 48 to 63.9, and 64 to 80 km/wk. A one km/wk decrease in running distance (detraining) produced a 0.068 kg/m^2 increase in BMI (the slope being negative) at 4 km/wk, a 0.034 kg/m^2 increase at 12 km/wk, and a 0.028 kg/m^2 increase at 24 km/wk. Decreases in men's running distance between 0 and 64 km/wk all significantly increased BMI. The reductions in BMI were nonlinear, e.g., decreasing running distance produced a four-fold greater increase in BMI between 0 and 8 km/wk than between 32 and 48 km/wk. Increasing weekly running distance (training) also affected weight loss nonlinearly, but the functional relationship differs significantly from that of detraining. The greatest weight gain occurred when distance was reduced from 8 to 0 km/wk, whereas increasing running distance from 0 to 8 km/wk produced the smallest reductions in weight (actually weight gain). Increases in running distances were not associated with statistically significant weight loss unless they exceeded 16 km/wk. The slopes for ΔBMI vs $\Delta\text{running distance}$ in men that increased their running distance differed significantly from those that decreased their distance for 0-8 ($P<0.0001$), 48-64 ($P=0.0009$), and 64-80 ($P=0.009$). Thus there was an asymmetric relationship between changing activity dose and weight depending upon whether exercise was increased or decreased—i.e., it appears to require a substantially greater activity difference to lose weight by becoming active than to gain weight by becoming inactive.

The results for men's waist circumference parallel those observed for their BMI: 1) exercise reduction significantly increased waist circumference for all distances under 48 km/wk; 2) the relationship was nonlinear, with detraining between 0 and 8 km/wk producing nearly a four fold greater increase in waist circumference per km/wk as detraining between 32 and 48 km/wk; 3) increasing exercise also had a nonlinear effect on waist circumference, with significant waist reduction only at longer training distances; 4) training and detraining affected waist circumferences asymmetrically, i.e., the slopes for increasing and decreasing running distance differed significantly between 0-8 km/wk ($P=0.003$).

As revealed by these analyses, the effects of training and detraining on women's BMI and waist circumference were similar to those observed in men. The women's results were less significant because the smaller sample size provided less statistical power. Women who increased and women who decreased their running distance had significantly different slopes for ΔBMI vs $\Delta\text{km/wk}$ between 0-8 ($P=0.0003$), 8-16 ($P=0.03$), and 48-64 km/wk ($P=0.04$). There was a significant sex difference in the functional relationship between ΔBMI vs. $\Delta\text{km/wk}$ for runners who decreased their running distance ($P=0.0004$), i.e., with women having smaller increases in BMI between 16-32 and 32-48 km/wk ($P=0.01$ for both).

Our approach specifies that the total expected change in adiposity is the sum of the slopes of each of the one-km/wk intervals between the baseline and follow-up distance. For example, a man who began the study running 10 km/wk and ended running 4 km/wk would be expected to gain 0.034 kg/m^2 for going from 10 to 9 km/wk, 0.034 kg/m^2 for going from 9 to 8 km/wk, and 0.068 kg/m^2 for each of 4 one-km/wk decrements between 8 and 4 km/wk, or a total of 0.408 kg/m^2 ($2*0.034+4*0.068$). Figure 3, which displays the cumulative effects of increasing and decreasing running distance, shows: 1) decreasing exercise causes significant weight gain at all exercise levels, but that the weight gain becomes progressively greater as men and women approach sedentariness, with the greatest gain when going from 8 km (5 mi) per week to none at all; 2) achieving weight loss by increasing vigorous exercise requires substantial effort, and is unexpected until running distance is greater than 25 km/wk in men or 48 km/wk in women; 3) there is a pronounced asymmetry in the effects of increasing and decreasing vigorous exercise. Above 32 km/wk in men and 16 km/wk in women, the effects of training and detraining are comparable, such that weight gains and losses associated with changes in exercise levels are probably reversible. However, below these levels an interruption in vigorous exercise is expected to produce weight gain that is not lost simply by resuming the same exercise level.

Comparison with traditional analyses. Most other papers compare changes in adiposity to physical activity without regard to the starting and follow-up levels. Our data show that simply comparing Δ BMI or Δ waist circumference to changes in running dose obscures the nonlinearity of the relationships, and the dissimilarities between training and detraining. In men, the traditional linear regression analyses suggest that each one-km/wk change in running distance was inversely related to a (slope \pm SE) -0.021 ± 0.001 kg/m 2 change in BMI and -0.044 ± 0.002 cm change in waist circumference (both highly significant) when adjusted for age and the years between baseline and follow-up. Fitting a quadratic term, (km/wk) 2 , provided limited evidence that the relationships were nonlinear. Specifically, the quadratic term was not significant for waist circumference, and although it was significant for BMI ($P=0.0001$) the effect was minor, i.e., a one km/wk decrement was predicted to produce a 16% greater increase in BMI at 40 km/wk than at 4 km/wk when adjusted for age and duration of follow-up. The traditional regression slope for Δ BMI vs Δ km/wk in women was significantly less negative than in men (-0.017 ± 0.001 kg/m 2 per km/wk, $P<<0.001$ for the significance of both the slope and its difference from men). The quadratic terms for female runners were nonsignificant for both Δ BMI ($P=0.71$) and Δ waist circumference ($P=0.33$). Moreover, the reduction in BMI from increasing running distance was equivalent to the increase in BMI from decreasing running distance (difference in slope: -0.0009 ± 0.0016 kg/m 2 in men and -0.0017 ± 0.0030 kg/m 2 in women, $P>0.58$ for both). The traditional approach also suggested that the slope for Δ waist circumference versus Δ running distance was the same for both training and detraining (difference in slope $P=0.35$ in men and $P=0.99$ in women).

Discussion

National survey data suggest that slightly more than a quarter of all adults perform some physical activity ≥30 min/day for five or more days per week or vigorous activity ≥20 min for three or more days per week [3]. Our results suggest that their continued adherence to regular activity is pivotal impeding the rise of obesity. The activity levels of most exercisers fall within the range where the weight gain from reducing or stopping

exercise is greater than the weight loss from increasing exercise. These asymmetric weight changes mean that usual activity levels may be insufficient for prescribing exercise as an obesity prophylaxis, or for predicting the efficacy of exercise prescription for controlling obesity. Usual activity that meets the targeted goals for preventing obesity may fall short of its anticipated benefits if the activity is irregular, seasonal, or often interrupted. If the asymmetry shown here extends to finer variation in activity over time, then erratic week-to-week exercise participation may be less effective in preventing weight gain than one adhered to religiously.

Although our analyses focuses exclusively on running, we expect the same principles apply other vigorous activities, and possibly to all exercise both moderate and vigorous. Running expends approximately 0.94 kcal/kg/km [15]. Figures 2 and 3 show that the greatest asymmetry in weight change occurs below running 16 km/wk, or 14.5 kcal/kg. This energy expenditure corresponds to walking briskly less than 26 km/wk [15]. Weight stability may require the maintenance of running levels above 32 km/wk in men (30 kcal/kg) and 45 km/wk in women (42.3 kcal/kg), i.e., the level above which variations in weekly exercise dose appear to affect weight symmetrically. Their energy equivalences are 54 and 76 km/wk of walking briskly, respectively. Whereas prior studies have been criticized for the low prevalence of higher intensity physical activity, the measurement error associated with low-intensity activity, and the inappropriate time frame of the assessment [16,17] our analyses are based on a well-quantified activity that had been sustained over many years.

Our findings provide a possible explanation for the Institute of Medicine (IOM) guidelines for preventing weight gain, namely the prescription of sufficient activity to elevate total energy expenditure to 160% of basal expenditure (or 170% of basal expenditure should we require that their recommendations be consistent with their empirical evidence)[1]. The IOM guidelines correspond to walking sixty minutes per day, which is slightly greater than the minimum weekly distance we have identified in men to produce symmetric weight changes from decreases and increases in activity. The threshold for symmetry in men is also consistent with the exercise levels recommended

by others to achieve long-term weight loss or prevent regaining weight (2000 kcal/wk [18, 19], 65 min/day of moderate intensity physical activity [20], 60–90 min of moderate intensity exercise or 35 min of vigorous activity per day [21]). The threshold is higher than the 18 km/wk reported by Slentz et al to prevent significant accumulation of visceral fat [22]. The healthy weight subjects identified in the IOM report may simply represent those individuals whose physical activity is sufficient to ensure their usual variations in activity produces symmetric weight gains and losses and thus no net weight gain. Below this level of activity, even if total average activity remains high, weight gains from small reductions in exercise are greater than the weight loses from increasing exercise, leading to a net weight gain.

Our findings support the current consensus that substantial exercise is required to produce weight loss [2,23,24,25]. Weight loss did not begin to occur unless running distance was increased above 25 km/wk in men and 48 km/wk in women. At lower activity levels, an increase in running distance was associated with weight gain, which may represent a shift in body composition from fat to muscle or reflect behavioral changes such as overcompensating energy intake.

Whereas this report has focused exclusively in the consequence of increasing and decreasing activity, elsewhere we have demonstrated that being consistently more active attenuates age-related weight gain [26]. The tendency to gain weight with age occurs even among fit, vigorously active men and women [27,28,29], and it is necessary to increase the exercise performed each year if age-related weight gain is to be prevented all together [26,27,29].

Our analytic approach was motivated by the apparent discrepancy between the nonlinear cross-sectional relationships we have reported [13,14] and the linear fits we obtained when Δ adiposity was regressed against Δ running. The cross-sectional data suggest differences in running distance are associated with greater differences in adiposity at shorter distances than longer distance (i.e., convex relationship). Figures 2 and 3 are entirely consistent with these cross-sectional relationships. Although the current report

exclusively involves vigorous exercise, cross-sectionally we have also reported there is a convex relationship between walking distance and adiposity [30], and asymmetric changes in adiposity might also apply to walkers who increase and decrease their activity.

Other large, long-term prospective studies have compared changes in activity to changes in weight [29,31-33], however their analyses will also not reveal the dose-response relationship unless the relationship is linear. This is because change in activity is a mixture of different starting and ending levels, e.g. a 10 km/wk increase in walking distance includes those who initially walked five, ten, or twenty km/wk at baseline who then walk fifteen, twenty, or thirty km/wk at follow-up. Other prospective studies that related a single measure of physical activity to prior [31, 34] or subsequent weight change [32, 35] also provide limited evidence for causality because they do not involve changes in activity. Forsooth, the prospective studies by Williams and Wood [26] and DiPietro et al [28, 29] considered weight change in relation to physical activity or fitness at baseline and follow-up, but not in a form analogous to the dose-response curves produced from cross-sectional data.

Our findings suggest that an effective public health policy for preventing weight gain must include a strategy to keep physically active men and women active. They also suggest that it may also be important to minimize exercise variation. Prior guidelines have focused almost exclusively on promoting physical activity among the sedentary. However, the benefits of such advocacy may be transitory unless activity is maintained consistently without extended interruption. The weight consequences of becoming inactive are not reversible simply by resuming prior activity. When the priority of regular exercise change due to obligations of family and work, the temptation to forgo activity must be countered by the knowledge that benefits gained by being active are not readily reclaimed. The leanness dividend from investing in exercise may be forever lost, or only reclaimed at considerably greater effort than simply sustaining a minimum level of vigorous exercise equivalent to running 16 km week.

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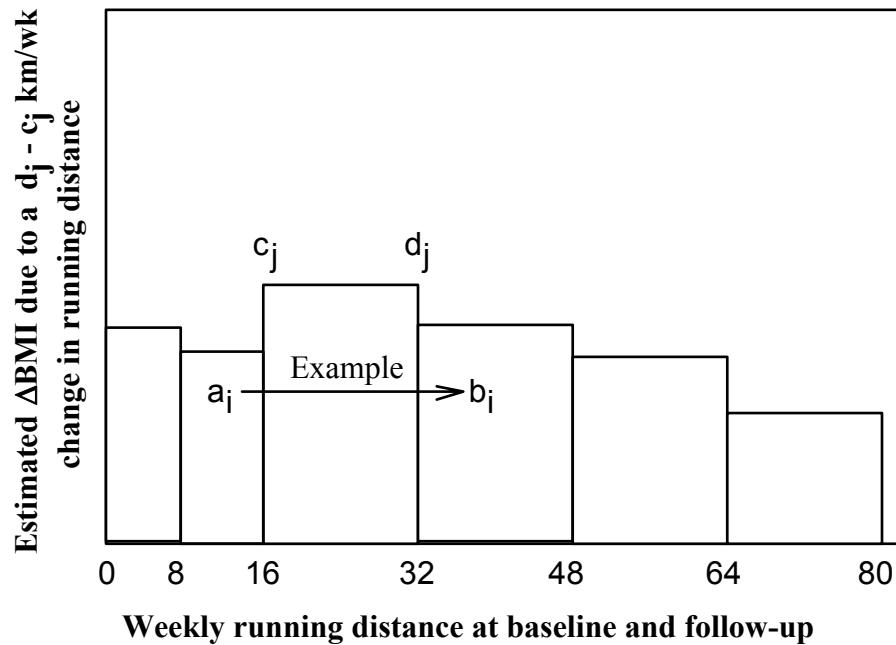


Figure 1. The additive contributions of the changes in weight for six predetermined intervals of running distance to an individual's total weight change. Specifically, if an individual ran 14 km/wk at baseline and 36 km/wk at follow-up, their Δ BMI is represented by two-eights of the predicted Δ BMI for going from 8 to 16 km/wk, all of the predicted Δ BMI in going from 16 to 32 km/wk, and four-sixteenths of the predicted Δ BMI in going from 32 to 48 km/wk.

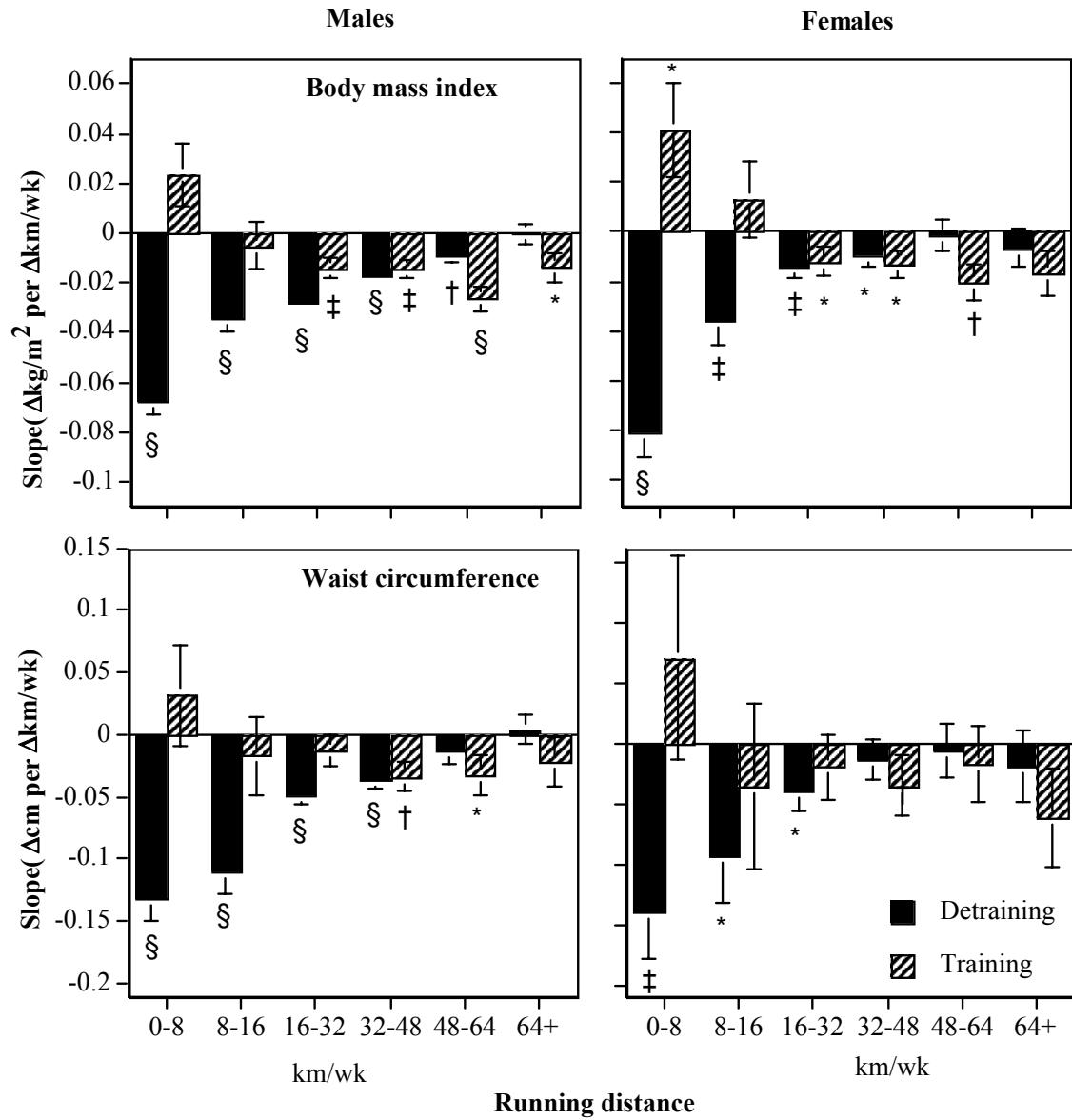


Figure 2. Effect of a one-km/wk change in running distance on Δ BMI and Δ waist circumference in 21,912 men and 7,923 women. The effects (slopes) were significantly different for training vs. detraining between 0-8 km/wk for both males' and females' changes in BMI ($P<0.0001$ and $P=0.0003$, respectively) and waist circumferences ($P=0.003$ and $P=0.05$), between 8-16 km/wk for women's BMI change ($P=0.03$), between 48-64 km/wk for men's and women's BMI change ($P=0.0009$ and $P=0.04$, respectively), and between 64-80 km/wk for men's BMI change ($P=0.009$).

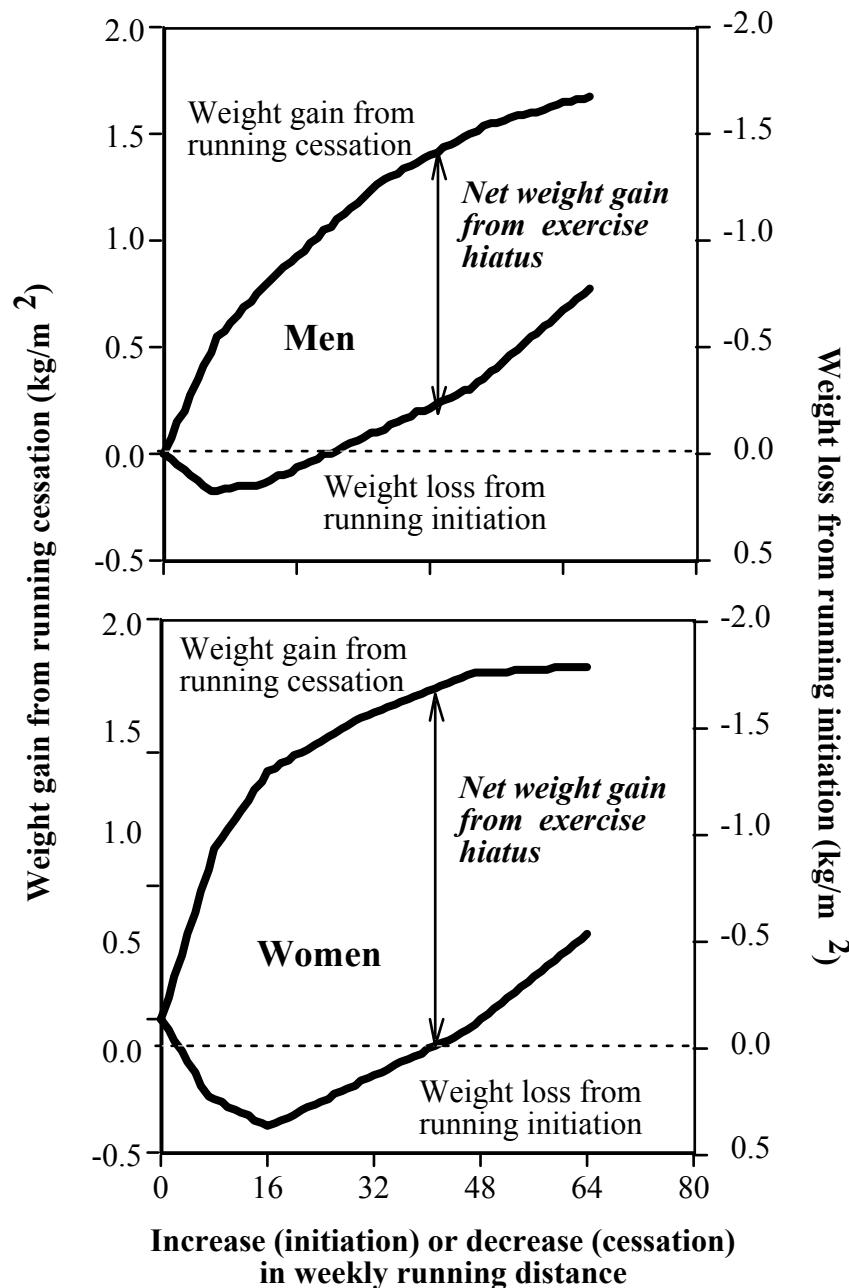


Figure 3. Cumulative effects of increasing and decreasing running distance on Δ BMI in 21,912 men and 7,923 women. The right vertical axis is the estimated effect of starting at the sedentary state at baseline and increasing running (i.e., training). Left vertical axis is the estimated effect of decreasing running to the sedentary state at follow-up (detraining). The effects of any change in distance may be calculated by subtracting the ordinate of the follow-up distance from the ordinate of the starting distance. The difference between the curves is the estimated weight gain for an extended hiatus in exercise.