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Asymmetric Weight Gain and Loss from Increasing and Decreasing Exercise

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Abstract

Purpose—Although increases and decreases in physical activity are known to cause weight loss and weight gain, respectively, it is not known whether the magnitudes of these changes in weight are equal. Unequal (asymmetric) weight changes could contribute to overall weight gain or loss among individuals with seasonal or irregular activity.

Methods—Changes in adiposity were compared with the running distances at baseline and follow-up in men and women whose reported exercise increased ($N = 4632$ and 1953 , respectively) or decreased ($17,280$ and 5970 , respectively) during 7.7 yr of follow-up.

Results—Per km/wk, decreases in running distance caused more than four times greater weight gain between 0 and 8 km/wk (slope $\pm$ SE, males: -0.068 ± 0.005 kg/m²; females: -0.080 ± 0.01 kg/m²) than between 32 and 48 km/wk (-0.017 ± 0.002 and -0.010 ± 0.005 kg/m², respectively). In contrast, increases in running distance produced the smallest weight losses between 0 and 8 km/wk and statistically significant weight loss only above 16 km/wk. 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 from decreasing exercise; otherwise, weight gain exceeded weight loss.

Conclusion—Weight gained because of reductions in weekly exercise below 30 kcal/kg in men and 15 kcal/kg in women may not be reversed by resuming prior activity. Current Institute of Medicine (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. Asymmetric weight changes below this threshold may contribute to weight gain among less-active subjects.

Introduction

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 [4,6]. One method of achieving this goal is by walking 60 – 90 min/d [6]. Approximately one quarter of Americans report exercising regularly [12]. However, other obligations, changing priorities, and waning motivation often interfere, causing disruptions in the amount performed [2,16,17]. An informed decision on whether and how to modify physical activity to accommodate these challenges requires that the consequences be understood - that is, the long-term effects of changing activity, and whether they are reversible.

Randomized controlled clinical trials show that increasing exercise, with or without dieting, causes modest weight loss [8,13], but their limited sample size, duration, and training dose usually preclude their being used to deduce the dose-response relationship to long-term trends in body 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 BMI and waist circumference in a large cohort of male and female runners. Despite their general leanness, increasing BMI and waist circumference within this population are associated with greater risks for hypercholesterolemia, hypertension, and diabetes [25]. Specific goals are to assess 1) whether a 1-km increment in running distance has the same effect on BMI and waist circumference at high (e.g., 40 km/wk) and low running distances (e.g., 10 km/wk), and 2) whether the weight gained by running less is equivalent to the weight lost by running more. The effects of increasing and decreasing activity are then compared, to assess whether weight gain during a hiatus in activity might be atoned for by resuming exercise.

Methods

The survey instruments and baseline characteristics of the National Runners' Health Survey are described elsewhere [23–29]. Briefly, baseline recruitment occurred between 1991 and 1993. A two-page questionnaire, distributed nationally at races and to subscribers of a popular running magazine (Runners' World, Emmaus, PA), solicited information on demographics, running history, weight history, smoking habits, prior history of heart attacks and cancer, and medications for blood pressure, thyroid conditions, high cholesterol, and diabetes. Running was the predominant physical activity of the study population. Although other leisure-time physical activities were not recorded for many in this cohort, data from runners recruited after 1998 (when the question was introduced in the survey) show that running represents (mean $\pm$ SD) $91.5 \pm 19.1\%$ and $85.2 \pm 24.0\%$ of all vigorously intense activity in these men and women, respectively, and $73.5 \pm 23.7\%$ and $69.4 \pm 25.7\%$ of total leisure-time physical activity. Follow-up surveys took place 7 yr later. Eighty percent of the 54,956 participants of the National Runners' Health Study provided follow-up information or were deceased. The study protocol was approved by the University of California committee for the protection of human subjects, and all participants signed committee-approved informed consents.

Changes in body mass index (BMI) were calculated as the changes in weight (kg) between the first and second survey, divided by the square of the average height (m) from the two surveys. Self-reported waist circumferences were elicited by the question, "Please provide, to the best of your ability, your body circumference in inches," without further instruction. Elsewhere, we have reported the strong correlations between repeated questionnaires for self-reported running distance ($r = 0.89$) [23], between self-reported and clinically measured height ($r = 0.96$) and weight ($r = 0.96$), and for self-reported running distance versus self-reported BMI and waist circumference in cross-sectional analyses [26,27]. Self-reported and clinically measure waist circumferences were moderately correlated ($r = 0.68$) [23].

To address the hypothesis of whether change in exercise affects weight, we restrict our analyses to runners whose follow-up weekly running distances differed from their reported baseline values by at least 2 km/wk (our analyses of runners who maintained consistent running levels have appeared elsewhere [24]). In addition, runners were excluded if they smoked, followed strict vegetarian diets, or used thyroid or diabetes medications because of their possible influence on adiposity.

Statistics

Our objective is to estimate the change in weight that occurs when running mileage is increased or decreased between specific starting (baseline) and ending (follow-up) levels (e.g. the expected weight loss from increasing weekly running distance from mileage from 14 to 36 km/

wk). In particular, we did not want to assume that an 8-km/wk decrease in running distance would produce the same effect when going from 64 to 56 km/wk as when going from 8 to 0 km/wk. Running distances were divided into six intervals: 0–8, 8–16, 16–32, 32–48, 48–64, and 64–80 km/wk (Fig. 1). Smaller intervals were chosen below 16 km/wk than above because prior cross-sectional analyses have suggested greater nonlinearity within this region [26,27]. The runners' data were then used to estimate the change in weight that occurs when running distance is increased from the lower to the upper bounds of each interval, and, separately, when running distance is decreased from the upper to the lower bounds (Fig. 1). The average Δ BMI for each of the six intervals was estimated, using multiple regression analyses and including adjustments for age and length of follow-up. Specifically, 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 independent variables. The fitted regression coefficients for each of the six intervals were then divided by the kilometer-per-week width of its corresponding interval (by 8 for the interval 8–16 km/wk; by 16 for the interval 32–48 km/wk) to estimate the Δ BMI for each 1-km/wk increase or decrease in running distance within the interval. An individual's total expected Δ BMI between baseline and follow-up was calculated as the sum of the predicted Δ BMI for intervals that lie between their baseline and follow-up surveys, including their fractional portions [24].

Results

Eighty percent of the 54,956 participants of the National Runners' Health Study provided follow-up information or were deceased. These included 4632 men and 1953 women whose running distances increased by two or more kilometers per week, and 17,280 men and 5970 women whose running distances decreased by two or more kilometers per week, all of whom were age-eligible, nonvegetarian, nonsmokers who did not take diabetes or thyroid medications. The two groups of runners (i.e., those who increased their running distance and those who decreased their running distance) had similar baseline BMI (mean $\pm$ SD, males: 23.89 ± 2.71 vs. 23.83 ± 2.63 kg/m²; females: 21.17 ± 2.41 vs. 21.25 ± 2.41 kg/m²) 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 they differed slightly by age (males: 43.63 ± 10.05 vs. 44.78 ± 10.47 yr; females: 37.79 ± 9.09 vs. 38.15 ± 9.90 yr) and duration of follow-up (males: 7.37 ± 1.82 vs. 7.94 ± 1.76 yr; females: 7.26 ± 2.08 vs. 7.66 ± 2.00 yr).

Figure 2 presents the estimated gain in BMI and waist circumference corresponding to each 1-km/wk change in running distance when going from 0 to 8, 8 to 16, 16 to 32, 32 to 48, 48 to 64, and 64 to 80 km/wk. A 1-km/wk decrease in running distance (detraining) produced a 0.068-kg/m² gain in men's BMI (the slope being negative) at 4 km/wk, a 0.034-kg/m² gain at 12 km/wk, and a 0.028-kg/m² gain at 24 km/wk. Decreases in men's running distance significantly increased BMI for all five intervals between 0 and 64 km/wk. The gain in BMI per kilometer-per-week reduction in running distance depended on the starting value; for instance, decreasing running distance produced a fourfold-greater gain in BMI per kilometer per week between 0 and 8 km/wk than between 32 and 48 km/wk. Increasing weekly running distance (training) also affected weight loss nonlinearly, but not in the same way as detraining. For men who cut back their running distance, the greatest weight gain occurred when distance was reduced from 8 to 0 km/wk, whereas among those who increased their running distance, going from 0 to 8 km/wk produced the smallest weight loss (actually weight gain). Increases in running distance were not associated with statistically significant weight loss unless they occurred above 16 km/wk. The slopes for Δ BMI vs. change in running distance in men who increased their running distance differed significantly from those who decreased their distance for 0–8 km/wk ($P < 0.0001$), 8–16 km/wk ($P = 0.009$), 16–32 km/wk ($P = 0.003$), and 48–64 km/wk ($P = 0.002$). Thus, there was an asymmetric relationship between changing activity dose and changing weight, depending on whether the men were increasing or decreasing their

running distance; it seems to require a substantially greater change in activity 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) reducing men's running distance significantly increased their waist circumference for all distances under 48 km/wk; 2) the relationship was nonlinear, with detraining from 8 to 0 km/wk producing a nearly fourfold-greater increase in waist circumference per kilometer per week than detraining from 48 to 32 km/wk; 3) increasing running distance also had a nonlinear effect on waist circumference, with significant waist reduction only at longer training distances; and 4) training and detraining affected waist circumferences asymmetrically; the slopes for increasing and decreasing running distance differed significantly between 0 and 8 km/wk ($P = 0.003$), 8 and 16 km/wk ($P = 0.008$), and 16 and 32 km/wk ($P = 0.01$).

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 Δ BMI vs. change in kilometers per week between 0 and 8 km/wk ($P < 0.0001$), 8–16 km/wk ($P = 0.01$), and 48–64 km/wk ($P = 0.04$). There was a significant sex difference in the functional relationship between Δ BMI versus change in kilometers per week for runners who decreased their running distance ($P = 0.0004$), with women having smaller increases in BMI than men between 16 and 32 km/wk ($P = 0.007$).

Our approach specifies that the total expected change in adiposity is the sum of the slopes for each 1-km/wk interval 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/m² for going from 10 to 9 km/wk, 0.034 kg/m² for going from 9 to 8 km/wk, and 0.068 kg/m² for each of the four 1-km/wk decrements between 8 and 4 km/wk, or a total of 0.340 kg/m² ($2 \times 0.034 + 4 \times 0.068$). Figure 3 displays the expected cumulative weight gain of starting at a specific baseline running distance and decreasing weekly running distance to 0 km/wk. The expected weight gain is given by the scale to the left. Thus, a man who was running 32 km/wk at baseline and quit had an average expected weight gain of 1.26 kg/m²; one who ran 16 km/wk and quit had an expected weight gain of 0.81 kg/m²; and one who reduced his distance from 32 to 16 km/wk had an expected weight gain of 0.45 kg/m² (1.26 minus 0.81 kg/m²). Figure 3 also displays the expected cumulative weight loss of a man starting at 0 km/wk at baseline and increasing his running distance. The expected weight loss is given by the scale to the right (note that the scale becomes more negative from the bottom to the top of the scale). Thus, a nonrunner who at follow-up was running 54 km/wk would have an average expected weight loss of 0.51 kg/m²; one who at follow-up was running 41 km/wk would have an average expected weight loss of 0.23 kg/m²; and a runner who ran 41 km/wk at baseline and 51 km/wk at follow-up would have an average expected weight loss of 0.28 kg/m² (0.51 minus 0.23 kg/m²). From bottom to top, the left-hand scale was drawn from smallest to largest weight gain (increasing larger Δ BMI), whereas the right-hand scale was drawn from smallest to largest weight loss (increasing larger $-\Delta$ BMI). This was done so that the graphs would lie on top of each other if the expected weight lost by increasing weekly running distance had the same kilograms per meter squared change as the expected weight gained by decreasing running distance. Otherwise, a runner who quit running and then resumed his initial running level at a later date would be expected theoretically to have a net weight gain represented by the difference between the curves.

Figure 3 shows that 1) decreasing exercise causes significant weight gain at all exercise levels, but the weight gain becomes progressively greater as men and women approach sedentariness, with the greatest gain when going from 8 km (5 miles) 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; and 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 would be expected to produce weight gain that is not lost simply by resuming the same exercise level. The corresponding graphs for changes in waist circumference with increased and decreased running distance (Fig. 4) are consistent with the cumulative effects displayed for BMI in Figure 3.

Discussion

National survey data suggest that slightly more than one fourth of all adults perform some physical activity for ≥ 30 min/d, five or more days per week, or vigorous activity for ≥ 20 min/d, three or more days per week [12]. Our results suggest that sustaining regular activity, without prolonged interruption, may be pivotal to impeding the rise of 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 even erratic week-to-week exercise participation may be less effective in preventing weight gain than one adhered to religiously.

Our findings support the current consensus that substantial exercise is required to produce weight loss [4,7,14,18]. Figure 2 and Figure 3 show that 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. The longitudinal data presented here are consistent with cross-sectional analyses of these runners at baseline [26,27] showing that differences in running distance are associated with greater differences in adiposity at shorter distances than at longer distance (i.e., convex dose-response relationship).

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 160–170% of basal expenditure [6]. The IOM guidelines correspond to walking 60 min/d, 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 (i.e., 32 km/wk expends about 30 kcal/kg, assuming that 1 km = 1.5 METs from the estimates provided in [1]; walking briskly for 1 hour consumes 4 METs or 28 kcal/kg/wk if performed daily, because 1 MET equals approximately 1 kcal/kg [1]). The threshold for symmetry in men is also generally consistent with the exercise levels recommended by others to achieve long-term weight loss or prevent regaining weight [9,10,18,19] and to prevent significant accumulation of visceral fat [20]. The healthy-weight subjects identified in the IOM report may simply represent those individuals whose physical activity is sufficient to ensure that their usual variations in daily, weekly, or monthly activity produce 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 may be greater than the weight losses from increasing exercise, leading to a net weight gain.

This report has focused exclusively on the consequences of increasing and decreasing activity; elsewhere, we have demonstrated that the maintenance of physical activity at a consistent level attenuates age-related weight gain in proportion to the exercise dose [24]. The tendency to gain weight with age occurs even among fit, vigorously active men and women [3,22], and it is

necessary to increase the exercise performed each year if age-related weight gain is to be prevented altogether [3,22,29]. Another earlier report from this cohort examined weight changes in 5417 runners who quit running altogether and 416 sedentary men and women who began running [28]. Those analyses show that exercise cessation caused weight gain, and starting exercise caused weight loss (actually, less weight gain), but they provide no overall assessment of the amount of weight gain or loss for incremental changes in running distance at high and low mileages.

To our knowledge, this is the first paper to specifically assess the changes in weight between specific baseline and follow-up exercise levels, and to specifically contrast the consequences on body weight of increasing and decreasing exercise. Other large, long-term prospective studies have compared changes in activity with changes in weight [3,5,15,21], but their analyses are unlikely to 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; for instance, a 10-km/wk increase in walking distance includes those who initially walked 5, 10, or 20 km/wk at baseline, who then walked 15, 20, or 30 km/wk at follow-up. Other prospective studies that have related a single measure of physical activity to prior [15,11] or subsequent weight change [5,30] also provide somewhat limited evidence for causality, because they do not involve changes in activity. The prospective studies by Williams and Wood [29] and DiPietro et al. [3] consider 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 analyses are based on well-quantified activity that was sustained over many years. Nevertheless, several limitations of our study must be acknowledged. Body weights and running distances were by self-reports, and, although generally reliable, this could introduce bias into the estimates. Waist circumferences were provided with minimal instruction, and there could be wide variation across individuals in their assessment. Detailed food records were not obtained, and, therefore, we cannot assess the contribution to obesity of changing dietary patterns with changes in exercise. We also did not monitor running levels between baseline and follow-up, which could affect weight. Additional studies are required into the physiological explanation of these findings. In summary, our findings suggest that an effective public health policy for preventing weight gain may need to include a strategy to keep physically active men and women active. They also suggest that it might 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. Our analyses suggest that the weight gain from becoming inactive may not be reversible simply by resuming prior activity. When the priority of regular exercise changes because of obligations to family and work, the temptation to forgo activity must be countered by the knowledge that the benefits gained by being active are not readily reclaimed. The leanness dividend from investing in exercise may be forever lost, or only reclaimed at a considerably greater effort than simply sustaining a minimum level of vigorous exercise equivalent to running 16 km/wk.

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References

1. Ainsworth BE, Haskell WL, Leon AS, et al. Compendium of physical activities: classification of energy costs of human physical activities. *Med Sci Sports Exerc* 1993;25:71–80. [PubMed: 8292105]

2. Booth ML, Bauman A, Owen N, Gore CJ. Physical activity preferences, preferred sources of assistance, and perceived barriers to increased activity among physically inactive Australians. *Prev Med* 1997;26:131–137. [PubMed: 9010908]
3. DiPietro L, Kohl HW 3rd, Barlow CE, Blair SN. Improvements in cardio respiratory fitness attenuate age-related weight gain in healthy men and women: the Aerobics Center Longitudinal Study. *Int J Obes Relat Metab Disord* 1998;22:55–62. [PubMed: 9481600]
4. Erlichman J, Kerbey AL, James WP. Physical activity and its impact on health outcomes. Paper 2: prevention of unhealthy weight gain and obesity by physical activity: an analysis of the evidence. *Obes Rev* 2002;3:273–287. [PubMed: 12458973]
5. Haapanen N, Miilunpalo S, Pasanen M, Oja P, Vuori I. Association between leisure time physical activity and 10-year body mass change among working-aged men and women. *Int J Obes* 1997;21:288–296.
6. Institute of Medicine. Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids (Macronutrients). Washington (DC): The National Academies Press; 2002. p. 936p
7. Jakicic JM, Clark K, Coleman E, et al. American College of Sports Medicine position stand. Appropriate intervention strategies for weight loss and prevention of weight regain for adults. *Med Sci Sports Exerc* 2001;33:2145–2156. [PubMed: 11740312]
8. Jakicic JM, Marcus BH, Gallagher KI, Napolitano M, Lang W. Effect of exercise duration and intensity on weight loss in overweight, sedentary women: a randomized trial. *JAMA* 2003;290:1323–1330. [PubMed: 12966123]
9. Jeffery RW, Wing RR, Sherwood NE, Tate DF. Physical activity and weight loss: does prescribing higher physical activity goals improve outcome? *Am J Clin Nutr* 2003;78:684–689. [PubMed: 14522725]
10. Klem ML, Wing RR, McGuire MT, Seagle HM, Hill JO. A descriptive study of individuals successful at long-term maintenance of substantial weight loss. *Am J Clin Nutr* 1997;66:239–246. [PubMed: 9250100]
11. Littman AJ, Kristal AR, White E. Effects of physical activity intensity, frequency, and activity type on 10-y weight change in middle-aged men and women. *Int J Obes Relat Metab Disord* 2005;29:524–533.
12. Macera A, Jones DA, Yore MM, et al. Prevalence of physical activity, including lifestyle activities among adults - United States, 2000–2001. *MMWR Morb Mortal Wkly Rep* 2003;52:764–769. [PubMed: 12917582]
13. Miller WC, Koceja DM, Hamilton EJ. A meta-analysis of the past 25 years of weight loss research using diet, exercise or diet plus exercise intervention. *Int J Obes Relat Metab Disord* 1997;21:941–947. [PubMed: 9347414]
14. National Institutes of Health. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults. The Evidence Report. *Obes Res* 1998;6(suppl 2):51S–209S. [PubMed: 9813653]
15. Owens JF, Matthews KA, Wing RR, Kuller LH. Can physical activity mitigate the effect of aging in middle-aged women? *Circulation* 1992;85:1265–1270. [PubMed: 1555270]
16. Sallis JF, Hovell MF, Hofstetter CR. Predictors of adoption and maintenance of vigorous physical activity in men and women. *Prev Med* 1992;21:237–251. [PubMed: 1579558]
17. Sallis JF, Hovell MF, Hofstetter CR, et al. A multivariate study of determinants of vigorous exercise in a community sample. *Prev Med* 1989;18:20–34. [PubMed: 2710760]
18. Saris WH, Blair SN, vanBaak MA, et al. How much physical activity is enough to prevent unhealthy weight gain? Outcome of the IASO 1st Stock Conference and consensus statement. *Obes Rev* 2003;4:101–114. [PubMed: 12760445]
19. Schoeller DA, Shay K, Kushner RF. How much physical activity is needed to minimize weight gain in previously obese women? *Am J Clin Nutr* 1997;66:551–556. [PubMed: 9280172]
20. Slentz CA, Aiken LB, Houmard JA, et al. Inactivity, exercise, and visceral fat. STRIDE: a randomized, controlled study of exercise intensity and amount. *J Appl Physiol* 2005;99:1613–1618. [PubMed: 16002776]

21. Taylor CB, Jatulis DE, Winkleby MA, Rockhill BJ, Kraemer HC. Effects of life-style on body mass index change. *Epidemiology* 1994;5:599–603. [PubMed: 7841241]
22. Williams PT. Evidence for the incompatibility of age-neutral overweight and age-neutral physical activity standards from runners. *Am J Clin Nutr* 1997;65:1391–1396. [PubMed: 9129467]
23. Williams PT. Vigorous exercise and the population distribution of bodyweight. *Int J Obes* 2004;28:120–128.
24. Williams PT. Maintaining vigorous activity attenuates 7-year weight gain in 8,340 runners. *Med Sci Sports Exerc* 2007;39:801–809. [PubMed: 17468577]
25. Williams PT, Hoffman K, La I. Weight-related increases in hypertension, hypercholesterolemia, and diabetes risk in normal weight male and female runners. *Arterioscler Thromb Vasc Biol* 2007;27:1811–1819. [PubMed: 17510467]
26. Williams PT, Pate RR. Cross-sectional relationships of exercise and age to adiposity in 60,617 male runners. *Med Sci Sports Exerc* 2005;37:1329–1337. [PubMed: 16118580]
27. Williams PT, Satariano WA. Relationships of age and weekly running distance to BMI and circumferences in 41,582 physically active women. *Obes Res* 2005;13:1370–1380. [PubMed: 16129719]
28. Williams PT, Thompson PD. Dose dependent effects of training and detraining on weight in 6,406 runners during 7.4years. *Obes Res* 2006;14:1975–1984.
29. Williams PT, Wood PD. The effects of changing exercise levels on weight and age-related weight gain. *Int J Obes Relat Metab Disord* 2006;30:543–551.
30. Williamson DF, Madans J, Anda RF, Kleinman JC, Kahn HS, Byers T. Recreational physical activity and ten-year weight change in a US national cohort. *Int J Obes* 1993;17:279–286.

FIGURE 1.

The additive contributions of the changes in weight for six predetermined intervals of running distance to an individual's total weight change. The variables 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 , $j=1,2, \dots,6$. Specifically, if an individual ran 14 km/wk at baseline and 36 km/wk at follow-up, his or her Δ BMI is represented by 2/8 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 4/16 of the predicted Δ BMI in going from 32 to 48 km/wk.

FIGURE 2.

Effect of a 1-km/wk change in running distance on Δ BMI and change in waist circumference (i.e., slopes of change in kilograms per meter squared per change in kilometers per week) in 21,912 men and 7923 women. Slopes that are significantly different from zero are designated by the following symbols: * $P < 0.05$; † $P < 0.01$; ‡ $P < 0.001$; § $P < 0.0001$. Brackets denote $\pm$ SE. Training and detraining produced significantly different effects on BMI between 0 and 8 km/wk ($P < 0.0001$), 8–16 km/wk (men: $P = 0.009$; women: $P = 0.01$), 16–32 km/wk (men: $P = 0.003$), and 48–64 km/wk (men: $P = 0.002$; women: $P = 0.04$). Training and detraining also produced significantly different effects on change in waist circumference between 0 and

8 km/wk (men: $P = 0.003$; women: $P = 0.05$), 8–16 km/wk (men: $P = 0.008$), and 16–32 km/wk (men: $P = 0.01$).

Figure 3.

Cumulative effects of increasing and decreasing running distance on ΔBMI in 21,912 men and 7,923 women. The right vertical axis is the estimated weight loss of starting at the sedentary state at baseline and increasing running (i.e., training) and therefore progresses from weight gain at the bottom of the scale (positive) to a weight loss (negative) at the top of the scale. The left vertical axis is the estimated weight gain of starting in the active state at baseline and becoming sedentary by follow-up (detraining) and therefore progresses from weight loss at the bottom of the scale (negative) to a weight loss (positive) at the top of the scale. The axes were drawn this way so that the difference between the curves is the estimated weight gain for an extended hiatus in exercise.

FIGURE 4.

Cumulative effects of increasing and decreasing running distance on changes in waist circumference. The right vertical axis is the estimated waist reduction of starting at the sedentary state at baseline and increasing running (i.e., training); it progresses from waist gain at the bottom of the scale (positive) to a waist reduction (negative) at the top of the scale. The left vertical axis is the estimated waist gain of starting in the active state at baseline and becoming sedentary by follow-up (detraining); it progresses from waist reduction at the bottom of the scale (negative) to a waist gain (positive) at the top of the scale. The axes were drawn this way so that the difference between the curves is the estimated gain in waist circumference for an extended hiatus in exercise.