QUEST, 2004, **56**, 12-27

Weight Loss for the Obese: Panacea or Pound-Foolish?

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Despite increasing prevalence of dieting, Americans continue to get fatter. Because traditional weight loss programs have poor long-term efficacy, a non-weight-centered paradigm might be more effective for improving the health of individuals considered overweight or obese. Many obesity-related health conditions can be ameliorated via physical activity and improved nutrition, independent of weight loss. Epidemiological evidence suggests that, compared to reported intentional weight loss, increasing physical activity or fitness, independent of body weight or changes in body weight, is associated with greater reductions in all-cause mortality rates. In view of the potential risks associated with calorie-restrictive dieting and weight fluctuation (yo-yoing), public health may be better served by placing greater emphasis on lifestyle changes and less importance to weight loss *per se*.

Weight loss is quite common in the United States, with an estimated 43.6% of women and 28.8% of men trying to lose weight (Serdula et al., 1999). The prevalence of dieting has increased considerably during the past 4 decades (Bacon et al., 2002). Despite efforts to lose weight, Americans are heavier than ever before (Flegal, Carroll, Ogden, & Johnson, 2002; Mokdad et al., 2001). Although most people lose weight for aesthetic reasons, health concerns are usually high on the list of reasons to lose weight. Indeed, it is for health concerns that overweight individuals are routinely advised by health care professionals to lose weight. Current public health recommendations encourage weight loss for overweight and obese individuals, with energy-restricted diets an integral part of the weight loss prescription (National Heart, Lung, and Blood Institute, 1998).

Although weight loss can be achieved relatively quickly via dieting and exercise, long-term efficacy of weight loss programs is poor (Anderson, Konz, Frederich, & Wood, 2001). Worse yet, long-term prospective studies on men and women indicate that a history of dieting may increase chances for subsequent—and significant—weight gain (Coakley, Rimm, Colditz, Kawachi, & Willett, 1998; Korkeila, Rissanen, Kaprio, Sorensen, & Koskenvuo, 1999). A recent prospective

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study on children ages 9-14 indicated that dieters gained significantly more weight during a 3-year follow-up compared to nondieters. The researchers concluded that "dieting to control weight was not only ineffective, it may actually promote weight gain" (Field et al., 2003, p. 900). Ironically, calorie-restrictive approaches to weight control may contribute to the very thing dieting is intended to cure—obesity! A temporal relationship between the increasing prevalence of obesity and of dieting has been noted (Bacon et al., 2002).

Even if it does not result in obesity, dieting may lead to a life of yo-yoing. Actually, chronic weight fluctuation may be the norm for most Americans (Williamson, 1993). Weight fluctuation has been associated with a number of adverse health outcomes (Blair, Shaten, Brownell, Collins, & Lissner, 1993; Ernsberger & Koletsky, 1993; Lissner et al., 1991). In view of this and the high recidivism rates for weight loss programs, perhaps it is time to rethink the current focus on weight loss for overweight and/or obese individuals.

Is weight loss, particularly via calorie restriction, the best course of action for the increasing numbers of Americans considered overweight or obese? Must overweight or obese individuals lose—or "normalize"—weight to improve their health and longevity prospects? Are the health risks of overweight/obesity as dire as commonly perceived? Might a non-diet, non-weight loss-centered approach to wellness be a viable alternative for millions of individuals stigmatized as "too fat?" This paper addresses these questions.

A Closer Look at the "Obesity Kills" Statistic

At the heart of the medical rationale for weight loss recommendations is the commonly held notion that obesity is a major contributing cause of premature death. When former U.S. Surgeon General C. Everett Koop launched his Shape Up America! campaign in 1994, he asserted that obesity is responsible for the deaths of approximately 300,000 Americans every year. Although no source was cited, it is likely that the statistic was taken from a paper published in the *Journal of the American Medical Association* a year earlier, entitled "Actual causes of death in the United States" (McGinnis & Foege, 1993). Since then, this figure has been cited countless times in scientific and medical journals and also has been mentioned repeatedly in the media (a Lexis database search performed in September 2003 revealed that this statistic has been cited more than 2,000 times since 1998).

The major problem with this "obesity kills" statistic is that it is virtually impossible to establish a definitive *causal* link between obesity and mortality. For example, the authors of the 1993 article in *JAMA* did not even list obesity as a potential contributing cause, but rather "diet/activity patterns." Although poor diet and a sedentary lifestyle can lead to obesity, the studies used to generate this statistic included persons in all body mass index (BMI) strata, not just the obese. To equate a physical trait with behaviors is unjustified, as the authors of the article emphasized in a subsequent letter to the *New England Journal of Medicine*: "The figure you cite applies broadly to the combined effects of various 'dietary factors and activity patterns that are too sedentary,' not to the narrower effect of obesity alone. Indeed, given the contribution of multiple diet-related factors to problems such as high blood pressure, heart disease, and cancer, we noted explicitly the

difficulty of sorting out the independent contribution of any one factor" (McGinnis & Foege, 1999, p. 1157).

That clarification notwithstanding, another paper, published in 1999, now appears to be the one most frequently cited to support the assertion that obesity is the second leading cause of preventable death in America. Based on their analysis of data from six epidemiological studies, the authors concluded that "obesity is a major cause of mortality in the United States," accounting for approximately 300,000 deaths each year (Allison, Fontaine, Manson, Stevens, & VanItallie, 1999).

To estimate the annual number of deaths attributable to obesity, Allison et al. first calculated mortality hazard ratios for all BMI groupings in the six studies selected (which included the Framingham Heart Study, Nurses' Health Study, Tecumseh Community Health Study, Alameda Community Health Study, American Cancer Society Prevention Study I, and the National Health and Nutrition Examination Survey I Epidemiologic Follow-up Study). In general, hazard ratios increased as BMI increased beyond about 25 to 27; for example, men and women with BMIs between 30 and 35 had a roughly 50 percent higher mortality rate compared to men and women with BMIs between 23 and 25. Hazard ratios were converted to actual numbers of deaths by extrapolation from each of the six studies to the entire U.S. population. Depending upon the study, annual death toll estimates attributable to obesity ranged from 236,111 to 383,410, with an overall average of approximately 300,000. Hence the authors' conclusion that obesity is a "major cause of mortality in the United States."

The major limitation in their analysis is best revealed by the authors' own admission: "Our calculations assume that all (controlling for age, sex, and smoking) excess mortality in obese people is due to their adiposity (Allison et al., 1999, p. 1536)." The authors made no attempt to determine whether other factors, such as physical inactivity, low fitness levels, poor diet, risky weight loss practices, weight fluctuation, use of weight loss drugs, less than adequate access to health care, etc. could have explained some or all of the excess mortality in large people.

Overweight and obese individuals are more likely to be sedentary and have lower aerobic fitness levels than non-overweight persons. Both sedentary lifestyle and low aerobic fitness (which can be altered independently of weight loss) increase risk for premature death (Hahn, Heath, & Chang, 1998; Lee, Blair, & Jackson, 1999). It is entirely possible that much of the health risk associated with obesity is really due to a lack of exercise and associated low fitness levels. In fact, data from the Aerobics Center Longitudinal Study (ACLS) show that low aerobic fitness levels accounted for 100 percent of the excess mortality among obese men (Lee et al., 1999). Similar results have been shown for women in the ACLS (Farrell, Braun, Barlow, Cheng, & Blair, 2002). Although a recent study on men and women in the Lipid Research Clinics Study indicated that fitness did not entirely reverse the increased risk associated with obesity, fitness did attenuate the risk (Stevens, Cai, Evenson, & Thomas, 2002). Additionally, data from the Behavioral Risk Factor Surveillance System show that lack of physical activity is more important than excess body weight as a predictor of cardiovascular mortality (Hahn et al., 1998).

Low fitness and sedentary lifestyle, however, are not the only factors that might explain the higher mortality rates of obese persons. The prevalence of dieting, particularly yo-yo dieting, is much greater among obese people, especially women. Consequently, obese people are far more likely to experience extreme

weight fluctuations during their lifetime. Weight cycling may elevate blood pressures (Guagnano, Pace-Palitti, Carrabs, Merlitti, & Sensi 1999; Guagnano, et al. 2000), reduce HDL-Cholesterol levels (Olson, et al., 2000), deplete body reserves of omega-3 fatty acids (Tang, Nishimura, & Phinney, 1993), and increase risk for gall bladder disease (Syngal et al., 1999), kidney cancer (Lindblad, Wolk, Bergstrom, Persson, & Adami, 1994), and breast cancer (Djuric et al., 2001; Uhley et al., 1994). Weight cycling also has been reported to increase risk of premature death, primarily from cardiovascular disease (Blair et al., 1993; Dyer, Stamler, & Greenland, 2000; Lissner et al., 1991). In fact, a 1991 report from the Framingham Heart Study (which is one of the studies used by Allison et al.) revealed that virtually all of the "excess" cardiovascular disease mortality in obese men and women could be explained by lifetime weight fluctuation (Lissner et al., 1991).

Obese women and men are also far more likely to have tried extreme and calorically unbalanced diets, such as starvation-type fasting and liquid protein diets. Also, obese women and men are considerably more likely to take weight-loss drugs, the hazards of which have been well chronicled (Berg, 1999; Connolly, et al., 1997; Haller & Benowitz, 2000; Kernan et al., 2000).

No published report on the alleged impact of obesity on mortality has satisfactorily eliminated the possibility that the higher death rates observed in women and men with higher-than-average BMIs are instead attributable to one or more of the many possible contributing factors, such as those mentioned above, that are more prevalent in this population.

Health Risks of Overweight and Obesity Exaggerated

It is generally true that persons with high BMI, or with higher than average amounts of body fat, tend to have more health problems than their thinner counterparts. This association between weight and poorer health is not necessarily causal nor universal. Not all overweight and obese persons have weight-related health problems. "Metabolically normal" obese persons have been well described in the literature (Sims, 2001). Consequently, their health does not necessarily improve with weight loss (Reaven, 2003). Furthermore, as will be addressed in a later section, most weight-related health problems can be ameliorated independently of weight loss. This suggests that obesity may be inappropriately blamed for a host of health problems that are really a result of an imprudent lifestyle (that fosters weight gain and health problems at the same time).

The presumed link between body fat (or BMI) and poor health is not nearly as straightforward as it may seem. Notwithstanding the well-cited and highly publicized studies associating overweight and obesity with increased mortality, most epidemiological studies reveal that aside from the extremes, BMI is not that strong a predictor of death rates (see Gaesser, 1999, for review). Even in studies that show higher risk at high BMI, the threshold at which risk increases significantly is not distinct and may be well above the BMI cut point (> 25) considered overweight.

For example, Troiano and colleagues analyzed data from a number of previously published studies, comprising a total of 356,747 men and 248,501 women (Troiano, Frongillo Jr., Sobal, & Levitsky, 1996). Among nonsmoking men stud-

ied for up to 30 years, the relationship between BMI and mortality was U-shaped, with the lowest mortality rates observed between BMIs of 23 and 29. Thus most of the BMI range that is considered overweight (i.e., BMI 25 to 29.9) was not associated with higher risk. Low BMI, on the other hand, was. For example, mortality rates for men with BMIs between 19 and 21 (i.e., within the "normal" range) were the same as for men with BMIs between 29 and 31 (i.e., extreme overweight or moderately obese). The researchers remarked, "This quantitative analysis of existing studies revealed increased mortality at moderately low BMI for White men comparable to that observed at extreme overweight, which does not appear to be due to smoking or existing disease. Attention to the health risks of underweight is needed, and body weight recommendations for optimum longevity need to be considered in light of these risks" (p. 63).

As for nonsmoking women, the researchers found no distinct minimum mortality point. The BMI range corresponding to the lowest death rates was quite large, stretching from about 18 all the way to 32. Thus a 5' 4" woman could weigh anywhere from 105 pounds to 185 pounds and have the same risk of premature death. Only beyond a BMI of 32 did the mortality rate begin to increase.

A report on 13,242 Blacks and Whites participating in the NHANES I Epidemiologic Follow-up Study revealed a U-shaped relationship between BMI and mortality (Durazo-Arvizu, McGee, Cooper, Liao, & Luke, 1998). The higher risk associated with thinness and obesity was noted for all groups and was not influenced by smoking status. Among Black men and women, lowest mortality rates occurred at a BMI of about 27 (i.e., "overweight"). Although minimum mortality in Whites occurred at a BMI between 24 and 25, the researchers emphasized that in all groups studied, there was a very broad BMI range of about 9 BMI units corresponding to only slightly elevated risk (20 percent or less). This translates to BMIs between approximately 22 and 31 for Blacks and 20 and 29 for Whites, and includes more than two-thirds of the U.S. population. The authors of the study concluded, "the resulting empirical findings from each of four race/sex groups, which are representative of the U.S. population, demonstrate a wide range of BMIs consistent with minimum mortality and do not suggest that the optimal BMI is at the lower end of the distribution for any subgroup" (p. 739).

Other studies show a similar characteristic "J"- or "U"-shaped relationship, with higher mortality at the extremes of BMI (Ernsberger & Koletsky, 1999). The higher risk for thin individuals has frequently been attributed to smoking. However, higher risk for thin individuals has been reported in a number of studies that have examined the BMI-mortality relationship separately for non- or never-smoking persons (see Gaesser, 1999, for review).

Furthermore, some of the more high-profile studies implicating excess weight as a contributing cause to higher mortality rates have overstated their findings, as the results from the American Cancer Society Cancer Prevention Study II illustrate (Calle, Rodriguez, Walker-Thurmond, & Thun, 2003). After a 16-year follow-up of more than 900,000 men and women, the authors concluded, "increased body weight was associated with increased death rates for all cancers combined and for cancers at multiple specific sites" (p. 1625). They also suggested that "more than 90,000 deaths per year from cancer might be avoided if everyone in the adult population could maintain a body-mass index under 25.0 throughout life" (p. 1634). The data from this study, however, are not wholly consistent with either of these assertions.

For example, the combined 16-year mortality rate from cancer for men and women with BMIs 18.5 – 24.9 was 908/100,000; for men and women with BMIs 25.0 – 29.9 the cancer mortality rate was slightly lower, 886/100,000. Increased body weight—at least within the overweight range—was *not* associated with increase death rates for all cancers combined. Only in the BMI ranges > 30 did cancer mortality increase. When the analysis was restricted to men and women who had never smoked, the cancer mortality risk associated with overweight was higher, but the higher risk was rather small by epidemiological standards (1.11 – 1.14) and might easily be explained by potential confounding factors not measured by the authors. Furthermore, mortality from lung cancer (by far the most common site) was generally highest in the BMI range of 18.5 – 24.9.

The authors obtained no information on changes in body weight during the 16-year follow-up. In this regard two points are worth making. First, because most Americans gain weight during adult years, it is very likely that many of the study participants who began the study with a BMI < 25 (i.e., the "low risk" range according to the authors of the study) were in the "overweight" range 16 years later. Thus to conclude that 90,000 deaths from cancer might be prevented if everyone could maintain a BMI < 25 throughout life is unwarranted. Additionally, it must be mentioned that in the earlier American Cancer Society Cancer Prevention Study I, intentional weight loss generally was not associated with lower mortality rates (Williamson et al., 1995; Williamson et al., 1999). This was certainly true for overweight men and women without reported health problems and for men with reported health conditions. In the one subgroup for which reported intentional weight loss was associated with reduced cancer mortality during a 12-year follow-up (women with reported health conditions), there was no dose-response relationship. A loss of 1-19 pounds was associated with the same reduction in mortality rate as was a loss of 20 or more pounds. Interestingly, unintentional weight *gain* was not associated with increased cancer mortality. Thus across-the-board recommendations for all overweight adults to lose weight in order to reduce risk of dying from cancer are not evidence-based.

Lose Weight – Live Longer?

In fact, there is not much in the way of evidence to support the commonly held notion that overweight and obese persons would live longer if they lost weight. Because weight loss is frequently associated with improvements in health status, it may seem logical to assume that weight loss would reduce death rates for overweight and obese persons. However, there is very little evidence to support this assumption. In fact, most epidemiological studies examining the relationship between weight change and subsequent mortality rates suggest that weight loss is associated with increased death rates compared to individuals who maintain fairly stable weights (Andres, Muller, & Sorkin, 1993).

In the NHANES I Epidemiologic Follow-up Study, Pamuk et al. (1993) reported that among overweight men and overweight and obese women, weight loss of more than 5% of maximum lifetime weight was associated with higher death rates compared to relatively weight-stable study participants (Pamuk, Williamson, Serdula, Madans, & Byers, 1993). The higher death rates associated with weight loss were observed in never smokers and after excluding from data analysis participants who died within the first 5-8 years of follow-up.

Weight loss was also associated with higher death rates in the Multiple Risk Factor Intervention Trial (MRFIT), the Framingham Study, and the Harvard Alumni Study (Blair et al., 1993; Higgins, D'Agostino, Kannel, & Cobb, 1993; Lee & Paffenbarger, 1992). Among Framingham men, weight loss was associated with significantly higher 20-year mortality from all causes, cardiovascular disease, and coronary heart disease. Weight gain, by contrast, was not associated with higher mortality, even though blood pressure increases during the 20-year follow-up were greatest in the men who gained weight (Higgins et al., 1993). The observation that moderate weight gain was not associated with increased mortality is not an unusual finding (Andres et al., 1993).

The higher cardiovascular disease mortality rates associated with weight loss among men in the MRFIT study is surprising since the men enrolled in this study were at high risk for vascular disease. Yet in all statistical comparisons involving weight loss (32 total in this study), weight loss was associated with higher mortality risk during the follow-up (range of relative risk = 1.04 – 3.42). It might be expected that moderately overweight men at high risk for vascular disease would benefit from weight loss. However, among men with baseline BMI between 26.08 and 28.82, those who lost > 5% of body weight during the trial had a cardiovascular disease mortality rate that was 1.95 times higher than the relatively weight stable referent group.

Studies of Intentional Weight Loss

Of course, one major limitation of most studies of weight loss and mortality is the failure to distinguish between intentional and unintentional weight loss. However, even in epidemiological studies that have addressed the issue of intentionality, results for the most part do not support the hypothesis that losing weight will reduce mortality rates in overweight and obese persons.

In the American Cancer Society's Cancer Prevention Study I on men and women with BMIs > 27, the association between self-reported intentional weight loss and subsequent mortality rates (12-year follow-up) depended on health status (Williamson et al., 1995; Williamson et al., 1999). Among men and women with no preexisting illness, intentional weight loss was not associated with reduced mortality rates. In some instances, intentional weight loss was actually associated with statistically significantly higher mortality rates (compared to equally overweight men and women who reported no weight change). Among women and men with one or more obesity-related health conditions, intentional weight loss was associated with a reduced all-cause mortality rate (for women) and mortality from diabetes (for men). The results on men are consistent with a previous report in which intentional weight loss was associated with reduced mortality rates among persons with glucose intolerance (Eriksson & Lindgarde, 1998). However, in the American Cancer Society studies, there was no dose-response relationship: A reported intentional weight loss of 1-19 pounds was associated with the same reduction in death rate as a reported intentional weight loss of 20 or more pounds. This suggests that specific weight loss goals for overweight women and men with obesity-related health conditions may be unnecessary.

In the Iowa Women's Health Study, the association between weight loss and all-cause and cardiovascular disease mortality was evaluated among 25,764 women

ages 55-69, categorized by weight status, health status, and amount and intentionality of weight loss (French, Folsom, Jeffrey, & Williamson, 1999). Among overweight women (BMI > 27.3), in no instance was weight loss—whether intentional or unintentional—associated with significantly lower mortality rates. In fact, intentional weight loss was more often than not associated with slightly (but not statistically significantly) higher death rates compared to women reporting stable weights. It is also worth noting that the all-cause mortality rate of overweight women was not higher, but actually slightly (~5-10%) lower than that of non-overweight women. From the perspective of avoiding premature death, these findings suggest that overweight women might be better off staying that way.

A recent report from the National Health Interview Survey provides even more puzzling information on the association between weight change and mortality rates (Gregg, Gerzoff, Thompson, & Williamson, 2003). A total of 6,391 overweight men and women (BMI > 25), ages 35 or older, were followed for 9 years. Compared to men and women who reported that they were not trying to lose weight during the year before the follow-up started, participants who reported they had intentionally lost weight during the year prior to initiating the follow-up (mean weight loss = 7.1 kg) had a ~10-30% lower mortality rate (depending on statistical model used) during the 9-year follow-up. Interestingly, those who reported that they had tried to lose weight, but were unsuccessful, had a ~10-20% lower mortality rate. In other words, just *trying* to lose weight appeared to be about as beneficial as actually losing weight. Even more puzzling was the finding that a small group (3.7% of the sample) who reported not trying to lose weight, and who actually had *gained* weight (mean gain = 4.3 kg) during the year before the follow-up began, had a mortality rate that was ~25-38% lower than overweight participants not trying to lose weight!

Similar to the results from the American Cancer Society studies, there was no dose-response relationship between reported intentional weight loss and reduced mortality rates. In fact, among those reporting intentional weight loss, a modest weight loss appeared to be the best. Those who reported losing 1-9 kg had a 30% lower mortality rate (95% confidence interval 0.53 – 0.92); those who reported losing > 9 kg had a nonsignificant 10% lower mortality rate (95% CI 0.64 – 1.25). Given that just trying to lose weight, but without success, was also associated with a lower mortality rate, these data suggest that emphasis on weight loss may be unnecessary. Also, since other epidemiological findings fail to support the common perception that losing weight will lengthen the life of overweight persons, categorical recommendations for all overweight persons to lose weight, regardless of current health status, may not be justified.

Reducing Death Rates

In contrast to the equivocal findings from observational studies on intentional weight loss, studies on the association between changes in either fitness or physical activity level, independent of changes in BMI, are far more consistent (Blair et al., 1995; Erikssen et al., 1998; Gregg, et al., 2003; Paffenbarger et al., 1993; Wannamethee, Shaper, & Walker, 1998). In the Aerobics Center Longitudinal Study, for example, during a follow-up period averaging 5.1 years, men who improved their cardiorespiratory fitness levels had a 44% lower mortality rate

compared to unfit men who remained unfit (Blair et al., 1995). Similar results have been reported for men who increased their physical activity level. For example, in the Harvard Alumni study, taking up moderately vigorous sports activity was associated with a 23% lower all-cause mortality rate, an association that was independent of changes in BMI (Paffenbarger et al., 1993).

A recent report on women participating in the Study of Osteoporotic Fractures demonstrated that compared to women who remained sedentary, those who increased physical activity between the baseline and follow-up (mean of 5.7 years) had a 48% lower all-cause mortality rate during an additional follow-up period lasting up to 6.7 years (Gregg et al., 2003). A significantly lower cardiovascular disease mortality rate (relative risk = 0.64) and cancer mortality rate (relative risk = 0.49) were also noted. The lower mortality rates associated with increased physical activity were independent of BMI.

Weight-Related Health Problems Resolved Independently of Weight Loss

Even though weight loss is usually associated with health improvements, sustained weight loss is rare, and thus health improvements only transient. There is a substantial body of literature indicating that most weight-related health problems, such as high blood pressure, dyslipidemias, insulin resistance, and glucose intolerance, can be improved independently of weight loss. In fact, improvements in lipoproteins and insulin action have been reported with exercise training even when body fat is *increased* (Lamarche et al., 1992).

Results from the Dietary Approaches to Stop Hypertension (DASH) clinical trial proved that blood pressures can be effectively lowered by simple changes in diet, without losing weight (Appel et al., 1997). Among 133 men and women with high blood pressure, just eating more fruits and vegetables and consuming low-fat dairy foods with reduced saturated fat was sufficient to reduce systolic and diastolic blood pressures within two weeks after changing their diets. The reductions in blood pressures were comparable to those observed with initiation of pharmacotherapy.

Exercise also can reduce blood pressure in the absence of weight loss. In a review of 44 randomized controlled trials, Fagard (l999) reported that reductions in blood pressure with exercise training were observed in both normotensive and hypertensive individuals and were almost entirely unrelated to changes in BMI (r = 0.09 between changes in BMI and changes in blood pressure).

Nutrition and exercise interventions can also improve blood lipid profiles independently of changes in body weight. Ehnholm et al. (1982) placed 54 middle-aged men and women on a low-fat (~24% of total calories) diet for six weeks. Total cholesterol dropped from 263 mg/dl to 201 mg/dl in the men, and from 239 mg/dl to 188 mg/dl in the women. Body weight did decrease modestly, by about 2 pounds. The subjects were then switched back to their usual diet (~39% of total calories from fat) for six weeks. Total cholesterol levels returned to their original levels despite no change in body weight. This suggests that the fat content of the diet, not weight change, was responsible for the changes in cholesterol levels.

More recently, Kraus et al. (2002) reported that six months of exercise training was sufficient to improve lipoprotein profile with minimal weight change in

111 overweight men and women with mild-to-moderate dyslipidemia. Combined exercise and nutrition programs have provided even more compelling results. After three weeks of consuming a low-fat, high-complex starch, high-fiber diet, in combination with daily moderate-to-vigorous aerobic exercise, average cholesterol level dropped from ~234 mg/dl to ~180 mg/dl; low-density lipoprotein cholesterol (LDL-C) decreased from ~151 mg/dl to ~116 mg/dl; and triglycerides were reduced from 200 mg/dl to 135 mg/dl (Barnard, 1991). Correlations between changes in lipids and changes in body weight ranged between 0.07 and 0.17, suggesting that very little of the decrease in cholesterol or triglycerides could be explained by decreases in body weight.

A recent randomized controlled study compared the effects of a special nutrition intervention (diet high in plant sterols, soy protein, viscous fibers, and almonds) vs. statin therapy (lovastatin) combined with a diet low in saturated fat, based on milled whole-wheat cereals and low-fat dairy products, on serum lipids and C-reactive protein in overweight, hyperlipidemic men and women (Jenkins et al., 2003). Control subjects received the same diet as those assigned to the lovastatin group. After one month, significant (~25-30%) and equal reductions were observed in both lovastatin and special intervention groups for total cholesterol, LDL-C, LDL-C/HDL-C, apolipoprotein B, and C-reactive protein. These changes occurred despite no reduction in body weight.

Just as risk factors for heart disease can be affected by changes in lifestyle independent of changes in body weight, the actual disease itself can be influenced by lifestyle modification, without changes in body weight. The results of the Cholesterol Lowering Atherosclerosis Study illustrate (Blankenhorn, Johnson, Mack, El Zein, & Vailas, 1990). Eighty-two moderately overweight middle-aged men with heart disease were placed in a two-year intervention program designed to reduce consumption of dietary fat. Men who reduced their fat intake to 27.5% of total calories showed no new fatty deposits in their coronary vessels (as determined by examination of coronary angiograms taken before and after the two-year study). By comparison, men who failed to make significant changes in fat intake showed some evidence of new lesions in their coronary vessels. Neither group lost any weight during the two-year study.

Improved insulin action can also be demonstrated with changes in diet and exercise, independent of changes in body weight (Barnard, Jung, & Inkeles, 1994; Cononie, Goldberg, Rogus, & Hagberg, 1994; Hickey et al., 1999). Barnard et al. (1994) showed that with intensive exercise and nutrition therapy, 39-71% of persons with type 2 diabetes could discontinue medication within three weeks. Most of the obese subjects were still obese at the end of the 3-week period, but without the need for either insulin or oral hypoglycemic agents. Cononie et al. (1994) and Hickey et al. (1999) showed that daily exercise can improve insulin action in men and women after just one week, with no changes in body weight.

An Alternative, Non-Weight-Centered Paradigm

This is not to say that we should be complacent about obesity. A continued focus on weight loss, however, seems counterproductive and may compromise the health of those who continually battle their weight. Long-term efficacy of calorie restriction weight-loss programs is poor (Anderson, Konz, Frederich, & Wood,

2001), and dieting may actually increase risk for overweight/obesity (Coakley et al., 1998; Field et al., 2003; Korkeila et al., 1999). Thus it might be prudent to adopt a new approach to health and fitness, one that places less emphasis on body weight (or body fat) and more emphasis on behaviors such as physical activity and healthier eating.

Bacon et al. (2002) published results of a randomized clinical trial that compared a non-diet, "wellness-centered" intervention to a traditional, "weight loss-centered" intervention in obese women. Seventy-eight nonsmoking, obese women with a history of dieting were randomized to one of two groups. The diet group was encouraged to moderately restrict consumption of fat and total calories and used traditional cognitive-behavioral methods to alter their eating habits. Those in the non-diet group focused on enhancing body- and self-acceptance, improving quality of diet, and were "supported in leading as full a life as possible, regardless of their body weight or whether they succeed at weight control" (p. 586). All participants were encouraged to increase physical activity. Subjects in both groups attended 24 weekly supervised sessions, followed by a voluntary 6-month aftercare program, which included optional monthly group support sessions.

As expected, only the diet group lost weight: 4.6 kg at 24 weeks, 5.9 kg total weight loss at 52 weeks. However, the attrition rate was much higher: 41% (16 of 39) vs. 8% (3 of 39). The lower attrition rate in the non-diet group was perhaps due to having a more favorable experience: significantly more (93% vs. 51%) participants in the non-diet group indicated that they "agreed" or "strongly agreed" with the statement, "the program has made me feel better about myself." Conversely, significantly fewer (7% vs. 35%) non-diet group members "agreed" or "strongly agreed" with the statement, "I feel like I have failed (or am failing) in the program."

From the perspective of "metabolic" fitness, both groups improved similarly. At 52 weeks, total cholesterol (mg/dl) decreased from 201 to 168 in the diet group, and from 201 to 169 in the non-diet group; LDL-cholesterol (mg/dl) decreased from 119 to 107 in the diet group and from 120 to 111 in the non-diet group; triglycerides (mg/dl) decreased from 172 to 125 in the diet group and from 180 to 137 in the non-diet group. Both groups experienced similar reductions in systolic blood pressure (mmHg): 126.9 to 118.7 in the diet group, and 125.3 to 120.8 in the non-diet group.

Evidence from two large-scale diabetes prevention trials suggests that it may be easier for individuals to adopt physical activity recommendations as compared to adhering to weight loss guidelines (Diabetes Prevention Program Research Group, 2002; Tuomilehto et al., 2001). In each of these trials, participants were up to twice as likely to achieve the physical activity goal as compared to weight loss goals established by the investigators. It is worth noting that the weight loss goals in these studies were not excessive, 5-7% of initial body weight. The physical activity goal in the U.S. study was 150 minutes of moderate-intensity activity per week (Diabetes Prevention Program Research Group, 2002); in the Finnish study the activity goal was 210 minutes of moderate-intensity activity per week, or about 30 minutes per day (Tuomilehto et al., 2001). Thus these studies suggest that it may be easier to get people to "move more" than to "eat less." Epidemiological evidence provides additional justification for such a strategy: compared to reported intentional weight loss, increases in physical activity and/or cardiorespiratory fitness

have been consistently associated with greater reductions in all-cause mortality rates (Blair et al., 1995; Erikssen et al., 1998; French et al., 1999; Gregg et al., 2003; Paffenbarger et al., 1993; Wannamethee et al., 1998; Williamson et al., 1995; Williamson et al., 1999).

A downside of a weight-centered approach to treating health problems linked to poor diet and a sedentary lifestyle is that exercise and healthy eating might be viewed merely as means to an end (weight loss), rather as having intrinsic value of their own. If individuals quit an exercise program out of failure to reach a particular weight loss (or reduced body fat) goal, then all the benefits of the exercise are lost as well. "Yo-yo fitness" might be as common as yo-yo dieting.

In America today, millions of men and women and boys and girls are stigmatized as "too fat," and the stigmatization of the obese has increased during the past 40 years (Latner & Stunkard, 2003). This obviously does not help to improve the health and well-being of the increasing number of individuals considered overweight or obese. If we acknowledge that healthy bodies can come in many shapes and sizes, and begin to treat lifestyle factors, such as physical activity and diet, as more than just means to an end (weight loss), then the pubic health message can be simplified. Many of the health problems associated with obesity are essentially the same as those linked to poor diet and sedentary lifestyle. A non-weight-centered paradigm allows for a more compassionate and open-minded view of body weight and may offer a viable alternative to traditional interventions plagued with poor long-term efficacy. Unlike restrictive dieting, there is little, if any, downside to physical activity and improved quality of diet. Those stigmatized as "too fat" need to be reassured that the roads to good health are wide enough for everyone.

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