Contrasting Perspectives

The Health Risks of Obesity Have Been Exaggerated

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Despite intense focus on obesity as a public health crisis, weight control efforts have been largely ineffective (1–4). A single-minded focus on weight loss is misguided; not only are success rates for long-term weight loss maintenance disappointing, a weight-centered paradigm is based on the flawed premise that obesity is the primary cause of many major weight-related health conditions.

The health risks attributed to obesity are exaggerated. Our rationale is based on the following: 1) a moderate-to-high level of cardiorespiratory fitness (CRF) attenuates, or eliminates, mortality risk associated with high body mass index (BMI); 2) a metabolically healthy obese (MHO) phenotype diminishes risk associated with high BMI; 3) removal of body fat does not improve cardiometabolic health; and 4) data on intentional weight loss and mortality do not support the conventional wisdom that high BMI itself is the primary cause of obesity-related health conditions.

We are not arguing that obesity is entirely benign, nor do we contend that we should be complacent about obesity or ignore it. Instead, we emphasize that treatment, if warranted, be non–weight loss-centered, with a focus on improving healthy behaviors.

FITNESS IS MORE IMPORTANT THAN FATNESS

Countless studies on the relationship between BMI and health outcomes, including mortality, have been published, with divergent results indicating no clear optimal BMI associated with lowest mortality risk (5). Additional studies are unlikely to resolve these disparate findings because almost all existing studies fail to acknowledge physical traits and behaviors that may covary with BMI and which greatly influence mortality risk. A perfect example is the 2017 report from the Global Burden of Disease (GBD) study that concluded that high BMI accounts for four million deaths annually (6). Despite published evidence that CRF is a more powerful predictor of mortality risk than traditional risk factors, including obesity (7–9), the GBD study failed to even acknowledge the influence of CRF on the BMI-mortality relationship.

The importance of CRF compared with obesity was demonstrated in a meta-analysis of 10 prospective studies that included measures of both CRF and BMI (10). In seven of the studies, CRF was split into “fit” and “unfit” categories based on quintiles (fit, 2nd to 5th quintiles), and in three of the studies CRF was split on the basis of MET scores or V̇O2max. Compared with fit normal-weight individuals, all-cause mortality hazard ratios for fit-overweight (1.13) and fit-obese (1.21) were not statistically significant, and, more importantly, were substantially lower than those for unfit-overweight (2.14) and unfit-obese (2.46) individuals (10). The fact that the hazard ratio for unfit-normal-weight individuals (2.42) was much greater than those associated with fit-overweight or fit-obese (i.e., 2.42 vs 1.13 or 1.21), demonstrates that CRF is much more important than fitness with regard to all-cause mortality risk. It is important to note that in these studies the definition of “fit” was rather modest, typically the top 80% of the CRF distribution. Such a level of CRF can be achieved by adherence to current public health recommendations for physical activity (PA) (11).

HEALTHY OBESITY

Obesity cannot be considered a single phenotype, as cardiometabolic risk varies widely across the BMI spectrum. There has been considerable debate over the concept of MHO (1,3,9,12,13). Metabolically healthy obese is typically defined as having no more than one component of the metabolic syndrome, or the absence of the metabolic syndrome (which permits up to
two components of the metabolic syndrome) (1,3). These are not good definitions because they include individuals who are not truly cardiometabolically healthy.

No studies have defined MHO as meeting zero criteria of the metabolic syndrome, and doing so may improve risk assessment (3). Another major weakness of the MHO definition is the failure to include CRF or objective measures of PA (9). Both CRF and PA contribute to the lower risk associated with the MHO phenotype (3,9,13).

Aside from the problems inherent in defining the MHO phenotype, risk associated with obesity as compared with risk associated with having cardiometabolic abnormalities that are used to define MHO, is illustrated by the meta-analysis of Kramer et al. (12). Compared with metabolically healthy normal weight, no higher risk of all-cause mortality or any cardiovascular event was observed among metabolically healthy overweight individuals. Among obese individuals, a modestly higher risk (1.24) was observed for MHO. By contrast, compared with metabolically healthy normal weight, relative risks associated with having an unhealthy cardiometabolic risk profile among normal weight (3.14), overweight (2.70), and obese (2.65) were substantially higher. This demonstrates that cardiovascular disease (CVD) risk factors are far more important to health status than is BMI.

**REMOVING BODY FAT DOES NOT IMPROVE CARDIOMETABOLIC HEALTH**

Two meta-analyses demonstrated that surgical removal of body fat did not improve CVD risk profile (14,15). In one (14), which included 357 patients from 15 studies, liposcopy of 2.06 to 16.3 L had no effect on any marker of CVD risk. In the other (15), which included a systematic review (11 studies) and meta-analysis (4 studies), abdominal liposuction was not associated with any improvement in triglycerides, high-density lipoprotein cholesterol, fasting glucose or blood pressure. Data on insulin were mixed, with some studies showing an improvement in fasting insulin and insulin sensitivity in the systematic review, but no effect of liposcopy on insulin sensitivity was observed in the meta-analysis. Even in obese women with type 2 diabetes, liposuction of 10.5 kg of abdominal fat had no effect on insulin sensitivity or blood markers of inflammation and other markers of CVD risk (16).

The amount of fat removed in these studies is comparable to, or greater than, the amount of weight loss typically associated with lifestyle interventions that improve markers of cardiometabolic health. This calls into question whether body fat per se is the underlying cause of cardiometabolic abnormalities. The fact that CVD risk markers can be significantly improved by changes in diet and exercise in the absence of fat loss also undermines the argument that body fat is the primary cause of the adverse cardiometabolic risk profile associated with high BMI (17).

**LOSE WEIGHT, LIVE LONGER? NOT ALWAYS**

If obesity is the primary cause of higher mortality associated with high BMI, weight loss should consistently be associated with reduced mortality risk. Several meta-analyses on the association between intentional weight loss and mortality risk have been performed, with mixed results. In a meta-analysis of lifestyle randomized control trials (RCT), among obese individuals, with follow-up 1.5 to 12.6 yr, intentional weight loss averaging 5.5 kg was associated with a 15% lower all-cause mortality risk (18). A more recent meta-analysis of weight-loss RCT reported that intentional weight loss of approximately 2.5 to 3.4 kg after 1 to 3+ yr was associated with an 18% lower all-cause mortality risk, but was not associated with reduced mortality from CVD or cancer, or in developing cancer or new cardiovascular events (19). Another meta-analysis showed that intentional weight loss was associated with a 16% lower mortality risk among unhealthy obese adults, but was not associated with lower risk for obese individuals considered healthy (20). In fact, among healthy individuals, including overweight, intentional weight loss was associated with a 9%–11% higher mortality risk. Among obese patients with type 2 diabetes, who would be expected to be the optimum beneficiaries of reduced body weight, intentional weight loss has not been shown to reduce mortality risk (21,22).

Even in acknowledging the reports showing a benefit of weight loss, the reduced mortality risk is approximately 15% to 18%. For context, improving CRF has been reported to be associated with a 35% to 59% reduction in all-cause mortality (23,24). Weight loss did not contribute to the lower mortality associated with increased CRF, and weight gain over time was not associated with increased mortality risk after adjusting for change in CRF (24).

**CONCLUSIONS**

Our reasons for asserting that the health risks of obesity have been exaggerated are not just academic, but rather have important implications for public health messaging. A monolithic view of obesity as the primary cause of morbidity and mortality associated with high BMI results in a weight-loss-centered focus as the cure (2). Not only has this proven ineffective (25), but continued focus on weight loss as a primary outcome of obesity treatment may have adverse health consequences for the people who attempt weight loss the most. Weight cycling and use of unhealthy weight loss practices are all more prevalent among those with high BMI, and have been associated with adverse health outcomes (1,5,17,25).

Cardiorespiratory fitness is an important vital sign and a stronger predictor of mortality than is obesity (7–9). Yet CRF is not mentioned in weight management guidelines (2). Removal of body fat, in the absence of changes in diet and exercise, does not improve cardiometabolic health. By contrast, substantial health benefits and reductions in risk of chronic diseases can be achieved via lifestyle changes with little, if any, weight loss (4,8,17). We urge health care professionals to promote the benefits of a healthier lifestyle independent of weight loss and inspire patients to focus on physical activity to increase fitness, rather than try to reach a specific weight loss target.
We agree with some points made by Jakicic et al. (26), most important of which is that all individuals should be encouraged to engage in lifestyle behaviors that include sufficient physical activity and a healthy diet. We are pleased to see that the authors acknowledge that such behaviors “may have effects on health independent of body weight status” and may reduce health risks “even in the absence of weight loss.” However, we disagree with their proposition that weight loss should be the primary goal for individuals with obesity, as this view is predicated on the tenuous assumption that higher body weight (or body fat) is the primary cause of obesity-related morbidity and mortality. Disentangling the hypothesized cause-effect relationship between body fat and associated health problems is straightforward. For one, as highlighted in our initial perspective, surgical removal of body fat without lifestyle change does not improve cardiometabolic risk profile. Moreover, most “weight-related” cardiometabolic risk markers can be ameliorated independent of weight loss (17).

Jakicic et al. cite several studies that show an association between high BMI and either mortality or incidence of several chronic diseases. None of these publications included measures of cardiorespiratory fitness (CRF). It is well established that CRF greatly attenuates, and often eliminates, associations between BMI and mortality, and that risk associated with low CRF is much greater than that associated with obesity (8). Consequently, by not including CRF in risk assessment, associations between BMI and chronic disease risk must be viewed as spurious. To illustrate, a 2008 systematic review and meta-analysis reported that increased BMI was associated with increased risk of cancer (27). But in this meta-analysis, “fitness” is never mentioned. By contrast, when CRF is included in the statistical analysis, cancer mortality risk is greatly attenuated across all BMI strata in both men (28) and women (29). Furthermore, maintaining or increasing CRF has been shown to reduce risk of cancer mortality, whereas changes in BMI or body fat were not associated with cancer mortality risk (30). Decreasing CRF, on the other hand, was associated with increased risk of cancer mortality regardless of changes in BMI (30).

Jakicic et al. cite the Look AHEAD trial to illustrate the beneficial effects of weight loss on improving glycemic control, as evidenced by an approximately 0.64% reduction in glycated hemoglobin (HbA1c) in patients with type 2 diabetes (T2D) who achieved a mean weight loss of 8.6% after 1 yr of lifestyle intervention. But exercise training alone can reduce HbA1c by a similar, or greater, amount with minimal weight loss. A meta-analysis of 27 exercise training studies in patients with T2D indicated that exercise training reduced HbA1c by approximately 0.8% (31). It is not likely that weight loss was the primary cause of the improved glycemic control because the reduction in HbA1c after aerobic training alone (−0.7%) was essentially the same as that for combined aerobic and resistance training (−0.8%) despite the fact the weight loss for the combined training (−5.1%) was more than three times greater than for aerobic training alone (−1.5%). In fact, exercise training has been shown to improve glucose tolerance even in individuals who actually gained body fat after exercise training (32).

Additionally, results from the Diabetes Prevention Program (DPP) are presented as evidence of the beneficial effect of weight loss to reduce risk of T2D. But reductions in T2D incidence similar to that documented in the DPP can be achieved by lifestyle changes in the absence of weight loss, as demonstrated by the PREDIMED trial (33). There are many other examples of improvements in glycemic control (and other cardiometabolic risk markers) with exercise and diet in the absence of weight loss (17). Consequently, assumptions regarding weight loss as the key component of lifestyle-induced improvements in health outcomes must be questioned.

An equally, if not more, plausible explanation is that changes in physical activity and/or diet are the primary causes of the improved health outcomes, and are largely independent of reductions in total body fat, but could be linked to decreases in ectopic fat (34). For example, exercise is effective for reducing hepatic fat (35), which may be the major cause of metabolic complications of obesity (36). Because hepatic fat comprises only approximately 1% of total body fat, this may help explain why exercise produces so many health benefits despite little, if any, loss of total body fat. In this regard it is important to emphasize that exercise affects virtually every cell in the body (37), and there is no reason to believe that the “polypill” effect of exercise (38) is restricted to nonobese individuals or is dependent on weight loss.

**CONCLUDING STATEMENT**

There is no question that obesity prevalence has increased. However, it is important to note that this has occurred during the same time period in which the prevalence of weight loss attempts also increased dramatically (25). Thus, weight loss maintenance has largely been a failure. A continued focus on weight loss as the primary endpoint of obesity treatment is not only futile, but, due to the higher prevalence of weight cycling and unhealthy weight loss practices among individuals with high BMI, potentially hazardous to health (5,25).

We contend that the health risks of obesity have been exaggerated, largely due to a failure to include measures of CRF in data analysis (as well as behaviors that covary with BMI and also impact health risk, such as physical activity, diet, weight cycling, and unhealthy weight loss practices), and also due to spurious conclusions based the presumed contribution of weight loss to improved health outcomes. Pragmatically, it may be better to focus on behaviors rather than specific weight loss targets. Results of the DPP are instructive: approximately 50% more participants were successful at achieving the physical activity goal (≥150 min·wk<sup>−1</sup>) than were able to achieve the weight loss goal (≥7% of body weight) (39). Considering the powerful and weight-loss-independent effects of exercise and diet (17,37,38), these behaviors should be the primary goal for obesity management, and not just used as means to an end (weight loss).
REFERENCES