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Obesity, Health at Every Size, and Public Health Policy

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Abstract

Obesity is associated with chronic diseases that may negatively affect individuals’ health and the sustainability of the health care system. Despite increasing emphasis on obesity as a major health care issue, little progress has been made in its treatment or prevention. Individual approaches to obesity treatment, largely composed of weight-loss dieting, have not proven effective. Little direct evidence supports the notion of reforms to the “obesogenic environment.” Both these individualistic and environmental approaches to obesity have important limitations and ethical implications. The low levels of success associated with these approaches may necessitate a new non–weight-centric public health strategy. Evidence is accumulating that a weight-neutral, nutrition- and physical activity–based, Health at Every Size (HAES) approach may be a promising chronic disease-prevention strategy.

Obesity is defined as having a body mass index (BMI; defined as weight in kilograms divided by the square of height in meters) in excess of 30. Obesity is associated with numerous chronic health conditions, including diabetes, hypertension, heart disease, and certain cancers.[1] The directionality of such associations is largely unknown, confounding may be present, and causality has only definitely been assigned to obesity with respect to osteoarthritis and ovarian cancer.[2] Despite these limitations, to counter the health effects of obesity-associated conditions, individuals frequently are encouraged to lose weight to improve individual and population health. However, diet-induced weight loss stimulates somatic and psychological ‘homeostatic pressures’ that induce weight regain.[3] These mechanisms include hormonal alterations, reduced satiety and energy expenditure, and increased hunger.[3,4] These adaptations stimulate weight regain in more than 90% of weight losers.[5,6] In acknowledgment of the limited effectiveness of individual approaches to weight loss, increasing emphasis has been placed on environmental reforms. However, when weight loss is the key motivator of such changes, they are hindered by a limited evidence base and ethical difficulties. These concerns suggest public health would benefit from a shift in focus from weight loss to disease prevention for individuals of all ages and sizes, with a focus on health rather than weight-loss outcomes, and environmental reforms devoted

to enhancing livability, accessibility, and equity. Evidence is accumulating that a weight-neutral, nutrition- and physical activity–based, Health at Every Size (HAES) approach may be a promising chronic-disease prevention, and overall well-being, strategy.

EXISTING APPROACHES

Current public health approaches to obesity largely consist of promoting individual weight loss dieting or alterations to "obesogenic environments."

Weight Loss Dieting

A particularly potent argument against public health's existing anti-obesity tactics is the proven ineffectiveness of weight loss attempts at an individual level. Indeed, as early as 1992, the American National Institutes of Health (NIH) released a consensus statement that dieting is an ineffective method to produce sustained weight loss.[7,8] Mann et al.[9] conducted a comprehensive review of the long-term consequences of weight loss dieting in long-term randomized trials, observational studies without a control group, and prospective studies without randomization. The studies reviewed by Mann et al.[9] were methodologically biased to show long-term weight-loss maintenance. The effectiveness of dieting presented by the studies was likely overestimated attributed to confounding of the effects of diet and exercise, low follow-up rates, self-report of participants' weight by phone or mail, and many participants' use of subsequent diets following the studies in question. Regardless, the trials demonstrated no consistent health benefits, including sustained weight loss. Indeed, one-to-two thirds of dieters regained more weight than initially lost.

Only recently have the long-term physiological changes following weight loss begun to be illuminated. In part, this gap in knowledge arises from low numbers of weight-loss maintainers.[1] Recent evidence on the physiological changes that follow weight loss in obese animal models and humans suggests organisms' endocrine systems actively promote weight regain.[3] Such changes involve highly integrated alterations including reduced satiety, increased hunger, suppressed energy expenditure, a decrease in nutrient availability, enhanced metabolic flexibility, an increase in energy efficiency and storage in peripheral tissues, a decline in adipose energy depletion signaling from leptin and insulin, altered neural activity, and alterations in hepatic, adipose tissue, and skeletal muscle metabolism.[3] Importantly, similar changes have been identified in humans for a full year after weight loss.[4] One year following weight loss, subjective hunger, ghrelin (hunger stimulating hormone), gastric inhibitory polypeptide, and pancreatic polypeptide were all elevated among overweight and obese individuals who lost weight in an intervention. Decreases in insulin, leptin, polypeptide YY, and cholecystokinin were also observed.[4] These findings align with the reduced rates of nonresting and resting energy expenditure and thermic effect of feeding present in individuals who have sustained a loss of at least 10% of body weight over a year.[10] Such processes may help explain the very high rates of recidivism in weight-loss dieters.[5,6] Given these rates of recidivism, it is concerning that weight regain is largely composed of fat; the weight regained does not replace bone mass or lean mass lost during weight loss.[8,9] Furthermore, weight loss may not be harmless and may increase stress, release of persistent organic pollutants, and risk of osteoporosis.[11]

Those individuals who do sustain substantial weight loss over time generally must maintain high levels of dietary restraint, physical activity, and self-monitoring behaviors.[11] This is evident in reports from members of the National Weight Control Registry (NWCR). The NWCR is composed of individuals who have sustained a 30-pound weight loss for at least a year.[5] The registry has been critiqued for its nonrepresentativeness of the US population, its reliance on self-reported data, and its high rates of loss to follow-up.[5] Ogden et al.[12] conducted a latent class cluster analysis of the NWCR to detect distinctive methods, experiences, and perspectives on weight loss and weight maintenance among successful weight losers. Although a majority of weight losers were satisfied with their weight loss and

reported good health and healthy behaviors, particularly high levels of exercise, other results were more distressing. For example, more than one quarter (26.9%) of participants reported high rates of stress, depression, and dissatisfaction with their weight loss. This cluster struggles with their weight, frequently weight cycle, and compared with other members of NWCR, report poorer health. Interestingly, these individuals are trying to maintain the greatest weight loss. The cluster identified as trying to maintain the second largest weight loss (9.9%), reported low levels of physical activity, frequent skipping of meals, and the poorest levels of health apart from those who had lost more weight. The relatively poor psychological and physical health of the individuals in these 2 clusters, and their struggles to maintain weight healthily, suggest that they may be striving to maintain an overly extreme weight loss.

Current public health approaches assume that intentional weight loss is always positive and that a BMI in the range of 18.5 to 24.9 is necessarily the healthiest range for all individuals. Importantly, despite its widespread adoption, BMI is acknowledged to be a crude measurement of obesity that may not adequately account for regional body fat distribution and subsequent risk.[13] In certain populations, BMI may not adequately measure adiposity or differentiate between populations, particularly with respect to athletes and athletic populations.[14] Perhaps most importantly, BMI is a population-level proxy measure of obesity, and an individual's BMI may say little concerning that individual's health.[15] Additionally, overweight status (BMI = 25–30) has been shown to be associated with decreased mortality risk in US, Canadian, and international samples.[11,16–18] Obesity (BMI > 30) has even demonstrated a protective or neutral effect among some chronic disease or older populations.[17,19,20] Minor (≤ 5%) intentional weight loss may reduce mortality risk in obese individuals with related comorbidities, but weight loss heightens mortality risk among healthy obese individuals.[21] Therefore, in addition to being extraordinarily difficult to maintain, weight loss may not always be health enhancing, particularly for the large proportion of obese persons who may be cardiometabolically healthy,[22] or whose ill health may be attributable to numerous non–weight-related confounding factors.[2]

Aiming for a narrow "healthy" range of body size may induce individuals to engage in disordered eating practices.[23] These behaviors may remain hidden, and these individuals may still be valorized for their "healthiness," particularly by others trying to lose weight. This is evident among members of, and in the espousal of seemingly bulimic practices in, commercial weight-loss groups.[24,25] That is, weight-loss groups' weight-loss strategies may include advice similar to bulimic practices of binge eating and subsequent compensation through dietary restriction or compulsive exercising. Furthermore, a public health approach in which obese individuals are viewed as unhealthy burdens on the health care system and individually responsible for their weight may promote weight stigma.[26] This stigma may produce ill health through direct stress-induced neuroendocrine pathways or adverse coping mechanisms.[26] Weight bias is highly prevalent among health care professionals, and this may pose a barrier to individuals receiving adequate health care.[27] Additionally, weight discrimination may diminish an individual's socioeconomic standing, which will have further deleterious effects on health.[28] In summary, the efficacy and safety of weight-loss dieting is questionable, as are the health consequences of the most common forms of obesity.[16,17] In addition to being ineffective, focusing on individuals as the cause and likeliest cure for obesity promotes weight stigma, which may be particularly health debilitating.[26]

Environmental Antiobesity Reforms

An ecological perspective on obesity causation was first proposed by Egger and Swinburn[29] in 1997 and further developed by Swinburn, et al.[30] in 1999. Their definition of the obesogenic environment included the micro- and macrocomponents of individuals' physical, political, economic, and sociocultural circumstances. More recently, the obesogenic environment is thought to be composed of

factors such as the reduced time-cost of food, the increased availability of high-calorie, nutrient-poor food, and the increased motorization and mechanization of daily life.[31] These factors are thought to have all simultaneously produced an energy imbalance in the daily lives of individuals and consequent population-wide weight gain.[31]

Rectifying the obesogenic environment would require comprehensive government reforms.[31] Existing evaluations of health interventions appear to support the notion that the most effective interventions for chronic disease prevention and health promotion focus on larger-scale interventions that make healthy choices more convenient and affordable, restrict unhealthy products, focus on community development, and support healthy social, economic, and environmental policy.[32] The most cost-effective approaches to obesity, as determined by modeling studies, are those that include a taxation on unhealthy foods, restriction of junk food advertisement to children, and improved nutrition labeling.[33] Targeted family-based programs for obese children and school-based campaigns to increase physical education and reduce sweetened beverage consumption and television viewing are also cost-effective in models.[33] These modeling studies and reviews of previous public health interventions have led proponents of obesogenic environmental reforms to extrapolate that obesity prevalence could be lessened through structural modifications that would facilitate energy expenditure or inhibit caloric intake.

Evidence for the Obesogenic Environment. Appeals to the obesogenic environment may appear to be an effective and nonstigmatizing approach to obesity policy. However, a number of limitations must be considered regarding these strategies. Particularly problematic is the lack of direct empirical evidence that environments necessarily predispose individuals to developing obesity, which calls into question the validity of using obesity as a justification for environmental reforms. A recent review, for example, highlighted that all evidence used to support the obesogenic environment is observational and inconsistent, and the proposed relationship between environment and obesity must be viewed as presumptive.[34] Models are thus based on hypothetical contributors to an overly simplistic energy balance model, rather than empirical evidence.[33] Furthermore, obesity may not even indicate poor health, and reduced obesity prevalence may not improve population health. Kirk et al.[35] assert that a main issue hindering effective obesogenic environment research is nonconsensus over what aspects of the environment, a necessarily complex, dynamic, and multilevel concept, should be implicated under the obesogenic environment rubric. Other issues persist in environmental obesity research. Randomized control trials are difficult to conduct, and impossible for the highest level of upstream environmental determinants given small number of units (e.g., high-end food policies).[36] Thus the highest quality of research originates from natural experiments or quasi-experimental designs.[36] However, the majority of environmental research remains observational.[35,37] Causality is difficult to assess, as multilevel studies can only suggest causality, and cross-sectional studies may not establish causality.[35,36] Difficulties arise in identifying valid, reliable, and consistent dietary, social and material resource, and spatial measures.[37] Accounting for all individual level factors and their potential mediating and confounding effects is particularly challenging.[37] Furthermore, there may be numerous unknown or unmeasured effects or multiple interactions of social and individual factors that may affect interpretation.[36]

Kirk et al.[35] conducted a scoping review on obesogenic environment research. The review determined that most studies focused on the physical microenvironment, and many studies used physical activity, rather than diet or BMI, as outcome measures. Few studies focused on the economic or political (such as household or workplace policies) microenvironment. Also limited were studies conducted on the macro level, including urban development, health and transportation systems, the media, and the food industry. Most importantly, the review found that such studies are methodologically stymied by a

general inability to measure potential environmental effects appropriately and a comprehensive theoretical framework that would help conceptualize the intricate and multifaceted obesogenic environment.

Reviews of existent research suggest only limited support for the obesogenic environment. A systematic review conducted by Giskes et al.[38] on environmental effects on fat and energy intake established there was limited evidence that environment influenced fat or energy intake and that any such evidence was produced from observational, cross-sectional studies. A more recent review on food-related environmental factors also was unable to confirm the impact of the obesogenic environment.[36] However, some reviews have found support for an association between the built environment and BMI or obesity.[30] Greater support is available for a relationship between physical activity accessibility and BMI or obesity than for food environments and BMI or obesity.[30] Evidence suggests walkability, density, accessibility of recreational spaces, and attractiveness increase physical activity and may lower risk of elevated BMI.[37] Isolated longitudinal studies have found some support for environmental factors and their effect on BMI and obesity.[37] Accessibility to healthy food increase is associated with dietary quality, but less evidence exists to support a link to BMI or obesity.[37] This may be an artifact of less research focusing on diet, because of dietary measures-related difficulties.[35]

Recent independent studies also failed to confirm a basic relationship between environment and obesity. McPhail et al.[39] found that Canadian adolescents' socioeconomic class or access to fast food did not affect their likelihood to consume fast food. Kim and Leigh[40] established that middle-class US familes were more likely to eat at fast-food restaurants than were lower or higher income individuals, whereas higher income individuals were more likely to eat at full-service restaurants. An and Sturm[41] found that California children and adolescents' proximity to fast-food outlets and convenience stores, and distance from larger grocery stores, is not associated with healthy food consumption or BMI. Lee[42] determined that elementary-school children residing in poorer and minority neighborhoods in the United States had greater access to both fast-food outlets and grocery stores. Food access was also not found to independently predict weight gain in children over time. This access also did not account for socioeconomic and minority differences over time in weight gain, which disadvantaged poorer and minority students. Interestingly, higher income and White majority neighborhoods also have a greater share of fast-food outlets, of all food establishments, compared with minority or poorer neighborhoods.[42] Junk food in schools was not a determinant in the development of obesity by the eighth grade in the United States.[43] A recent study by the US Department of Agriculture asserted that assessments of the affordability of a healthy diet are largely dependent on the metric used to measure food prices. For example, if food is measured by portion, it appears less expensive than if food is measured by weight or calorie.[44]

Ethical Considerations of Environmental Reforms. Ethical implications of broad-based antiobesogenic environmental reforms are also essential to consider. As limited interventions have been implemented, this involves considering their potential implications on target audiences based on proposed projects and deployed rhetoric. Broad alterations to the environment may increase stigma on obese individuals who do not lose weight, despite these environmental modifications.[45] Should individuals not lose weight despite environmental reforms, this might be viewed as proof that their obesity is the result of an unwillingness to practice healthy choices.[46] Such stigma may contribute to poorer health outcomes.[26] Obesogenic environment approaches may contribute to a homogenizing view of lower-income lifestyles and the perception of lower-income individuals as passive and lacking in agency.[47] Furthermore, this view may serve as a mask for gender-, race-, or socioeconomic-based discrimination and essentialist arguments, and the moralization of a health issue.[47–53] The obesogenic environment rationale has also been criticized as being overly simplistic and dismissive of the role of neoliberalism, its systematic overproduction and overconsumption, and consequent societal anxieties and values.[48,52] Importantly, broad-based reforms may become increasingly invasive and truly limit the freedom of

choice of individuals.[46] A compelling argument can be made for the type of environmental restructuring proposed by obesogenic environment arguments, without relying on mobilizing fear of fat to support their institution.[46] By focusing on other measures of health and well-being, or sustainability, livability, and accessibility, valuable and legitimately effective reforms could be undertaken. In summary, limited evidence suggests that environment affects obesity prevalence in a direct, simplistic manner, and reforms based on restructuring particular environments to remedy obesity rates have important ethical implications. Furthermore, as the empirical evidence suggests, none of these actions may necessarily result in thinner populations.

CARDIOMETABOLIC HEALTH AND OBESITY

Obesity is associated with a number of conditions including diabetes, hypertension, heart disease, and certain cancers[1]; however, causality has yet to be determined in most instances.[2] Furthermore, it is essential not to overlook potential ill health in normal-weight individuals. Critically assessing the studies that shaped biomedical views of the risks conferred by obesity is also imperative for better understanding the relationship between obesity and health. In this respect, longitudinal studies utilizing disease outcomes such as cardiovascular disease (CVD), diabetes, or stroke may be especially important to consider. For example, Pardo Silva et al.[54] reported, based on data from the highly esteemed Framingham Heart Study, that obesity before middle-age in both men and women was associated with numerous negative health outcomes. These included lower life expectancy, myocardial infarction, stroke, an increase in the number of years living with said diseases, and fewer number of years lived free of CVD. Critically, this study did not control for cardiorespiratory fitness (CRF), physical activity, diet, social support, income, occupation, weight history, or weight loss drug use. The study's only controls related to smoking status, number of cigarettes smoked per day, and the exclusion of individuals with diabetes, hypertension, and hypercholesterolemia at start of follow-up.[54]

To better understand underlying cardiometabolic risk in US adults of various weight classes, Wildman et al.[22] examined data from the National Health and Nutrition Examination Survey (NHANES) from 1999 to 2004. They determined the prevalence of overweight and obese adult individuals who are metabolically fit (characterized by 0 or 1 of the following abnormalities: low high density lipoprotein cholesterol level or elevated blood pressure, C-reactive protein, homeostasis model assessment of insulin resistance value, levels of triglycerides, and fasting plasma glucose) and normal-weight adult individuals presenting with cardiometabolic risk clustering. The authors found that more than one half of the overweight participants (51.3%) and approximately one third of the obese participants (31.7%) were metabolically well. By contrast, nearly one quarter of normal-weight individuals (23.5%) had cardiometabolic risk clustering. A predictor of metabolic health was physical activity.[22]

Similarly, data from the 2009–2010 NHANES suggest that rates of high cholesterol are low and declining as of 1999 in the United States.[55] Additionally, in the American Bogalusa Heart Study, the children participants experienced an increase in obesity during the years 1974 to 1993; however, their rates of hypertension decreased.[56] This indicates that despite fears of rising disease risk resulting from increased obesity levels, these risks are less prevalent than assumed. This may be the result of better diets, more physical activity, and medication use.

May et al. also present a complex view of cardiometabolic health in US adolescents from the NHANES from 1999 to 2008.[57] Obesity stabilized during this time at approximately 20%. Prevalence of prehypertension or hypertension and borderline-high or high low-density lipoprotein cholesterol also did not change over this time period.[57] Prevalence of prediabetes or diabetes, however, increased substantially by 14% during these years. Given the stabilization in obesity over this time, this again suggests a severance from the seemingly unassailable link between increasing obesity and elevation in diabetes risk. Importantly, although 61% of obese adolescents and 49% of overweight adolescents had at least 1 CVD risk factor, 37% of adolescents of normal weight also had at least 1 CVD risk factor.[57]

Independent Effects of Fitness and Diet on Health

Increasing evidence suggests fitness and diet may affect health independent of weight status, and that obesity and fitness are nonmutually exclusive. Physical inactivity has a biologically plausible, temporally consistent, and dose-dependent relation to risk factors, chronic morbidity, and mortality.[58,59] Low CRF has an even stronger effect on these outcomes than does physical inactivity.[58] In fact, fitness may be as relevant a predictor of mortality as diabetes mellitus and other CVD risk factors, and it may be a stronger predictor than BMI, obesity, or abdominal obesity.[59]

Obese individuals who engage in moderate intensity physical activity for 150 minutes per week have half the death rates and lower rates of CVD than their unfit, normal-weight counterparts.[58] Furthermore, physically active overweight or obese individuals may have greater cardiovascular fitness than inactive individuals, regardless of weight status.[60] Both CRF and obesity were found to affect self-rated health (SRH) among adolescent Portuguese girls. However, the association between obesity and SRH was eliminated in multivariate statistics, suggesting that CRF may mediate this relationship.[61] In California, children involved in a school prevention program, fitness improved, although obesity rates did not.[62] This again suggests that obesity and fitness are not mutually exclusive. Furthermore, if potentially beneficial programs are only assessed based on weight loss outcomes, they may be substantially undervalued.

In US adults of various weight classes, healthy lifestyle habits (e.g., moderate drinking, not smoking, regular exercise, and fruit-and-vegetable consumption) significantly decreased the risk of mortality for all individuals, irrespective of initial BMI. Obese individuals benefitted the most from the adoption of healthy lifestyle habits.[63] In fact, obese individuals who adopted all 4 healthy lifestyle habits, had the lowest risk of mortality compared with every other weight strata and lifestyle combination.[63] Over 6 years, US adults who experienced changes in fatness and fitness displayed alterations in their risk for incidence of metabolic syndrome, hypertension, and hypercholesterolemia.[64] This reduction in risk persisted even when the changes in fatness were controlled for changes in fitness and when changes in fitness were controlled for changes in fatness. However, these adjustments for changes in fitness or fatness did attenuate the reductions seen in health risk. In Katzmarzyk and Lear's[65] systematic review on the effect of physical activity on chronic disease risk factors in obese individuals, only modest benefits for obese individuals with respect to chronic disease risk factors were evident. However, even in intervention groups without a dietary component, significant improvements were produced in some studies in blood pressure, insulin, glucose, triglycerides, C-reactive protein, and cholesterol measures.

All these studies exemplify what are referred to as obesity paradoxes. These are studies that provide findings that seemingly contradict obesity epidemiology orthodoxy.[20] The first recognized obesity paradox is that obese individuals with CVD are more likely to survive than their normal weight counterparts. Other obesity paradoxes identified by McAuley and Blair[20] include that being overweight (BMI = 25.0–29.9) may be protective in relation to mortality compared with being of normal weight; that a large component of overweight and obese populations are metabolically healthy; and that being physically fit, even when obese, may eliminate the association between obesity and increased mortality.

Evidence relating to diet and obesity may eventually be synthesized into an additional obesity paradox. Diet may have an effect on health and mortality, independent of weight measures. Healthy diet was related to a significantly reduced risk of all-cause or CVD mortality among US, German, and British samples.[66–68] Joseph et al.[69] hypothesized that physical activity may also improve eating behavior. Physical activity may induce neurocognitive developments that inhibit hedonic urges for overconsumption. These developments may also improve capacity for goal-oriented behaviors,

inhibitory control, and executive functioning.[69] Studies independently linking diet to health may also help explain why lower fruit and vegetable consumption is not always an indicator of obesity.[70] Obese individuals may still consume a diet high in nutritious quality, without experiencing weight loss.

Adipose Tissue Effects on Health

The mentioned studies indicate that overweight and obese individuals may be physically fit, consume a nutritious diet, and experience high cardiometabolic health. This supports the hypothesis that 2 separate mechanisms are at work in obesity-related conditions. A specific pathway involves excess adipose tissue, particularly visceral adipose tissue, affecting health. A second pathway influences health through lifestyle factors, such as physical activity and diet, independent of fat and size. Of consequence in this regard is recent research into adipose tissue and its functioning as an endocrine organ to affect both health and behavior.

Adipose tissue develops in numerous parts of the body, mainly between muscle and skin, although it is also present surrounding internal organs.[71] These different depots have dissimilar metabolic profiles. Adipose tissue functions in providing mechanical support, insulation, and as a storage site for excess fuel.[71] Such fuel is stored as triglycerides and released as fatty acids in response to hormonal and sympathetic signaling. Additionally, adipose tissue releases its own endocrine signals, such as leptin. These signals include adipokines and other cell types that help regulate food intake and energy expenditure.[71] These cells have effects on numerous body systems and functions including energy homeostasis, blood pressure, the immune system, and the complex, highly regulated, process of adipose tissue expansion. Leakage of nutrients into other organs (e.g., liver, muscle) during periods of nutritional overflow, or excessive or altered adipokines, may result in abnormal somatic functioning.[71] The dysregulated activities of adipokine-related processes form the physiological underpinnings of numerous obesity-related morbidities including type II diabetes mellitus, hyperlipidemia, hypertension, and coronary heart disease.[71]

Thus, obese individuals, particularly abdominally obese individuals, may suffer health risks associated with the endocrine actions of adipose tissue. However, independent pathways related to healthy dietary consumption and CRF may provide obese individuals protection from adverse health outcomes. This may aid in explaining obesity paradoxes; some obese individuals may not suffer from increased mortality risk and may be metabolically healthy,[20] whereas some thinner individuals may be metabolically compromised despite their “healthy” BMI.

HEALTH-AT-EVERY-SIZE AS A NEW PARADIGM

The public appears to be increasingly supportive for less weight-centric approaches to public health.[72,73] HAES advocates healthy diets and health-sustaining physical activity, but is weight neutral. Its proponents do not view weight as an adequate indicator of health or weight loss as an appropriate end goal. HAES promotes consuming healthful foods, honoring internal cues of hunger and satiety, engaging in enjoyable physical activity, and advocating against fat stigma and in support of social justice, rectifying health disparities, and for ethical treatment of people of all sizes.[11,74]

The dietary component of HAES is based on an intuitive eating model.[11,74] Intuitive eating operates on the assumption that is it external cues, such as being coaxed to eat to excess in familial or social settings or engaging in weight-loss dieting, that leads to chronic eating problems and contributes to diet-related ill health. Intuitive eating gains support from early studies conducted on toddlers by Clara Davis, which were subsequently replicated in more recent studies.[75,76] These studies examined the diets of small children who were allowed to eat from an assortment of nutritious foods. No exhortations were given to eat more or less in general or of any particular food. Researchers found that young children, although exhibiting substantial variety in consumption at meals and occasional food jags, ate

relatively the same amount of calories and macronutrients over a 24-hour period over 6 days. The children always had sufficient energy for activity. Therefore, children will self-compensate and can meet their own nutritional needs in the absence of external cues.[75,76] In practice, intuitive eating essentially advocates eating what one wants when hungry and stopping when one is full.[76] As early as 1991, this approach was proposed as a method for preventing obesity. This suggests that intuitive eating has long been viewed as a method of achieving a healthful body size, even among those who problematize obesity.

Critics of HAES fear size acceptance may lead to excessive consumption and weight gain.[11] However, in the 6 completed random control trials of HAES interventions found no negative effects, including weight gain, and there was maintenance or improvement in behavioral, psychological, clinical, and physiological outcomes.[11,77] Intriguingly, when an HAES intervention among premenopausal women employed a control group undergoing a weight-loss intervention that incorporated a social support component, no significant differences in eating behaviors between the groups was evident over time. However, decreases in hunger and overall energy intake in some HAES participants were evident.[78,79]

Also encouraging is that even with weight regain, maintained behavioral practices, such as those produced in HAES interventions, have sustained health benefits.[80] Among Israeli adult participants in a long-term weight-loss intervention, partial weight regain occurred over time. This partial weight regain stimulated deterioration in a number of biomarkers (insulin, leptin, triglycerides, monocyte chemoattractant protein 1, chemerin, and retinol-binding protein 4). However, sustained dietary alterations produced benefits in other biomarkers (fetuin-A, hsCRP, adiponectin, HDL-C, progranulin, and vaspin levels), in spite of partial weight regain.[78] Thus, it may be that as excess adipose tissue exerts independent effects on cardiometabolic health, so too do sustained dietary behaviors. This finding is very important given the high rate of weight regain among weight loss losers.[5,6] Adopting a HAES approach appears to produce longer-lasting behavioral changes than do weight-loss interventions.[11,77] These maintained behavioral changes may induce healthier somatic functioning, regardless of weight status. By not using weight changes as a marker for health, individuals may be less discouraged by weight stabilization or gain. They may be more likely to continue in behaviors that have an independent benefit on health, rather than potentially growing discouraged and abandoning healthy behavioral efforts.

SYNTHESIS OF BEST APPROACHES

In summary, an exclusive focus on either individualistic or obesogenic accounts of obesity may be flawed in producing effective health promotion policy. Individualistic weight-loss focuses are largely ineffective and may be stigmatizing. Furthermore, weight loss may not be necessary for health improvement in obese persons and may be harmful. It is undeniable that a person's sociocultural, economic, and physical circumstances affect their well-being, but the current obesogenic environment rhetoric presents too simplistic a conceptualization of such a complex and dynamic process. As a result, it tends to oversimplify important socioeconomic, cultural, and ethnic considerations of well-being, and homogenizes diverse life ways. At worst, it may mask other forms of discrimination in its focus on "target populations."[81]

The most effective method of addressing chronic disease may be to focus on health rather than weight. By encouraging physical activity and healthy nutrition among all individuals, everyone, regardless of weight status, may benefit with respect to health and well-being. Individuals' mental and physical health may benefit from environments more suited to active transportation and increased access to produce, whether significant aggregate reductions in obesity are also produced. Furthermore, weight stigma, which may produce harmful weight and appearance preoccupation in all individuals, may be ameliorated by focusing on creating salubrious environments and encouraging healthy behaviors for all people. This is likely more effective than focusing exclusively on obese individuals and on a

potentially unattainable goal, such as weight loss.[46] Obesity rates are declining.[82] Therefore, preventing and treating chronic disease, rather than promoting weight loss, may be a greater public health priority, regardless of weight-loss proponents' views on the benefits of weight loss for population health.

Initiating such changes within the current public health environment may be difficult to implement. In a policy analysis of British Columbia's antiobesity campaigns, high levels of support were found for adopting weight-neutral public health language.[83] Adopting antiweight bias training in medical school was deemed feasible. Also supported was the implementation of obesity research guidelines that ensures research includes measures of socioeconomic status, diet, physical activity, and weight cycling to better delineate their independent effects on health, and reports potential adverse outcomes of lifestyle changes or weight loss. Unfortunately, less support was found for government-funded HAES studies, hindering the development of a more substantial HAES evidence base. Proceeding with initial small changes in terms of language and education may facilitate future willingness to fund such research, however. For example, well-evaluated HAES teacher-resources that encourage healthy behaviors, without triggering body-image issues or eating disorders, have been produced and may be used in place in more weight-centric school health programs.[84]

An analogous approach to obesogenic environment proponents' focus on broader structural issues is Health in Every Respect (HIER).[85] HIER expands the sphere of HAES to include a more holistic perspective on health that incorporates socioeconomic and psychosocial dimensions. It investigates more intricate health-effecting pathways, rather than overly relying on nutrition and physical activity, and offers a critique of evidence-based medicine. It defies the healthism and individualism that may permeate existent public health approaches. HIER advocates political action that more directly implicates the socioeconomic factors that are far more relevant to health than lifestyle factors.[85] This approach may extend the individual benefits of a HAES-approach to more environmental reforms, and underscores the commitment of HAES to social justice and ethical practice.[11,74,85] Most importantly, this approach aligns with compelling international evidence that the most effective approach to improving population health is to redress socioeconomic disparities.[86] Previous weight-centric approaches to public health have largely been ineffective.[9] However, evidence suggests that weight-neutral physical activity and nutrition-based approaches may be a promising new direction for encouraging lasting wellness in all individuals.

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