

POINT–COUNTERPOINT

The epidemiology of overweight and obesity: public health crisis or moral panic?

Paul Campos,^{1*} Abigail Saguy,² Paul Ernsberger,³ Eric Oliver⁴ and Glenn Gaesser⁵

National and international health organizations have focused increasingly on a perceived obesity epidemic said to pose drastic threats to public health. Indeed, some medical experts have gone so far as to predict that growing body mass will halt and perhaps even reverse the millennia-long trend of rising human life expectancy.¹ In response to such concerns public health agencies across the world have sprung into action, searching for policies or incentives to mitigate the alleged ‘disease’ of obesity.

Yet even as the volume of alarm grows louder, a growing number of researchers, drawn from a broad array of academic disciplines, are calling these claims into question. The authors of this article come from this latter group. In our view the available scientific data neither support alarmist claims about obesity nor justify diverting scarce resources away from far more pressing public health issues. This article evaluates four central claims made by those who are calling for intensifying the war on fat: that obesity is an epidemic; that overweight and obesity are major contributors to mortality; that higher than average adiposity is pathological and a primary direct cause of disease; and that significant long-term weight loss is both medically beneficial and a practical goal. Given the limited scientific evidence for any of these claims, we suggest that the current rhetoric about an obesity-driven health crisis is being driven more by cultural and political factors than by any threat increasing body weight may pose to public health.

Claim #1: ‘Almost all countries (high-income and low-income alike) are experiencing an obesity epidemic...’ WHO, 2003 (p. 61).²

The claim that we are seeing an ‘epidemic’ of overweight and obesity implies an exponential pattern of growth typical of epidemics. The available data do not support this claim. Instead, what we have seen, in the US, is a relatively modest rightward skewing of average weight on the distribution curve, with people of lower weights gaining little or no weight, and the majority of people weighing ~3–5 kg more than they did a generation ago.³ The *average* American’s weight gain can be explained by

10 extra calories a day, or the equivalent of a Big Mac once every 2 months. Exercise equivalents would be a few minutes of walking every day. This is hardly the orgy of fast food binging and inactivity widely thought to be to blame for the supposed fat explosion.

While there has been significant weight gain among the heaviest individuals⁴ the vast majority of people in the ‘overweight’ and ‘obese’ categories are now at weight levels that are only slightly higher than those they or their predecessors were maintaining a generation ago. In other words we are seeing subtle shifts, rather than an alarming epidemic. Biologist Jeffery Friedman offers this analogy: ‘Imagine that the average IQ was 100 and that five percent of the population had an IQ of 140 and were considered to be geniuses. Now let’s say that education improves and the average IQ increases to 107 and 10% of the population has an IQ of >140. You could present the data in two ways. You could say that average IQ is up seven points or you could say that because of improved education the number of geniuses has doubled. The whole obesity debate is equivalent to drawing conclusions about national education programmes by saying that the number of geniuses has doubled.’

In the US, to take a much-cited example, the so-called ‘obesity epidemic’ is almost wholly a product of tens of millions of people with BMIs formerly in the 23–25 range gaining a modest amount of weight and thus now being classified as ‘overweight’, and, similarly, tens of millions of people with BMIs formerly in the high 20s now having BMIs just >30. This movement of population cohorts from just below to just above the formal definitions of overweight and obesity is what public health officials are referring to when they point out that rates of obesity have exploded over the course of the last generation. (Furthermore, there is some evidence that adult and childhood BMI may have ceased to increase, as shown by comparison of NHANES data from 1999 to 2000 and from 2001 to 2002).⁵

In any case the real question is whether these developments represent some sort of genuine health crisis. This is true only if crossing the threshold of BMI 25 or 30 is analogous to contracting a life-threatening disease. But this analogy holds only to the extent that overweight and obesity actually cause increased mortality.

Claim #2: ‘Mortality rates increase with increasing degrees of overweight, as measured by BMI.’—WHO, 2003 (p. 61)²

This claim, central to arguments that higher than average body mass amount to a major public health problem, is at best

¹ School of Law, University of Colorado, CO, USA.

² Department of Sociology, University of California, Los Angeles, CA, USA.

³ Department of Nutrition, Case Western Reserve University School of Medicine, OH, USA.

⁴ Department of Political Science, University of Chicago, Chicago, IL, USA.

⁵ Curry School of Education, University of Virginia, VA, USA.

* Corresponding author. E-mail: paul.campos@colorado.edu

weakly supported by the epidemiological literature. Except at true statistical extremes, high body mass is a very weak predictor of mortality, and may even be protective in older populations. In particular, the claim that 'overweight' (BMI 25–29.9) increases mortality risk in any meaningful way is impossible to reconcile with numerous large-scale studies that have found no increase in relative risk among the so-called 'overweight', or have found a lower relative risk for premature mortality among this cohort than among persons of so-called 'normal' or 'ideal' [sic] weight. Among the obese, little or no increase in relative risk for premature mortality is observed until one reaches BMIs in the upper 30s or higher. In other words, the vast majority of people labelled 'overweight' and 'obese' according to current definitions do not in fact face any meaningful increased risk for early death. Indeed the most recent comprehensive analysis of this question within the context of the US population found more premature deaths associated with a BMI of <25 than with a BMI above it. This was largely owing to the finding that lowest death rates fell within the BMI range of 25–29.9—some 86 000 fewer 'excess' deaths than was observed in the referent group, the so-called 'normal weight' BMI range of 18.5–24.9. Additional analyses that controlled for potential confounders such as length of follow-up, weight stability, weight loss caused by illness, or smoking status did not change the results. For this nationally representative cohort of US adults—National Health and Nutrition Examination Surveys I, II, and III—the 'ideal' weight for longevity was 'overweight'.⁶

These most recent findings from the NHANES data should come as no surprise. Data from NHANES I published in 1998 revealed essentially the same thing—a U-shaped relationship between BMI and mortality. Significantly increased mortality was only associated with either extreme of BMI. As noted by the authors, 'the resulting empirical findings from each of the four race/sex groups, which are representative of the US 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'.⁷

These findings from representative US cohorts are consistent with global observations. In a quantitative analysis of a number of previously published studies, involving >600 000 men and women, Troiano *et al.*⁸ observed a similar U-shaped relationship between BMI and mortality, with the lowest mortality rates between BMIs of 23 and 29. Most of the range considered 'overweight' was not associated with higher risk. On the other hand, low BMI was. For example, mortality rates for men with BMIs between 19 and 21 were the same as those for men with BMIs between 29 and 31. Troiano *et al.* emphasized that, 'this quantitative analysis of existing studies revealed increased mortality at moderately low BMI for white men comparable with 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'.⁸

Rarely do the risks of thinness get any media attention. In the recent Flegal study,⁶ for example, underweight (BMI < 18.5) was associated with an estimated 33 746 excess deaths, despite the very small percentage (2.7%) of the subject pool that was in this category. In most of the NHANES cohorts, the relative risks associated with underweight were greater than those associated

with even high levels (BMI > 35) of obesity. Furthermore, when Flegal and her colleagues used a BMI range of 23–24.9 as the referent category, underweight (BMI < 23) was associated with 81 705 excess deaths. In other words, including men and women in the lower end of the 'normal weight' range increased the number of deaths associated with thinness by nearly 48 000. Of course, if the referent BMI group had been in the BMI range of 25–29.9, the excess deaths associated with being underweight or 'normal weight' would have exceeded 100 000, with the majority of these coming from the BMI range currently defined as 'healthy'.

But the greatest problem with the statistical linkages between body mass and mortality is that other confounding factors are not considered, leaving little basis for drawing causal inferences. Most epidemiological studies estimating the relationship between body weight and mortality do not control for fitness, exercise, diet quality, weight cycling, diet drug use, economic status, or family history. Furthermore, in studies that control for some of these factors, the data are usually self-reported and thus of extremely questionable reliability. (See, for example, the five-point exercise scale used in the Nurses' Health Study.) By contrast, when one or more confounders are controlled for in a rigorous fashion, the already weak association between higher body mass and greater mortality tends to be greatly attenuated or disappear altogether. For example, all of the excess mortality associated with obesity in the Framingham study can be accounted for by the impact of weight cycling.⁹ Obese Framingham residents with stable body weights were not at increased risk. The same result has been obtained in NHANES.¹⁰

Fitness is closely intertwined with obesity, and has powerful influences on health and mortality. Data from the Aerobics Center Longitudinal Study show that low cardiovascular fitness accounted for all of the excess all-cause mortality among obese men.¹¹ Similar data by these researchers have been reported for women.¹² In short, it seems probable that body weight, like height or baldness, is for the most part a proxy for many unmeasured variables. From a public health perspective, the most significant aspect of such a conclusion is that most of these unmeasured variables, especially the lifestyle factors, are more readily modifiable than body mass.

Many common weight loss treatments generate particularly problematic confounders. For example, over-the-counter diet pills used by millions, including phenylpropanolamine and herbal ephedra, have been linked to heart attack and strokes and recently banned.^{13–16} The adjusted odds ratio for stroke in women taking phenylpropanolamine for weight loss was 16.6,¹⁶ many times higher than the relative risk for stroke associated with a BMI > 30, which in one typical study was 1.29 (not significant).¹⁷ And the higher a person's BMI, the more likely they are to use these and other hazardous weight loss methods, including surgery. One study found that 22% of weight loss clinic clients surveyed used phenylpropanolamine for weight loss.¹⁸ If only one in 13 obese persons were exposed to over-the-counter diet pills containing phenylpropanolamine, then all of the excess risk of obesity could be accounted for by increased diet pills use. No epidemiological study to date has assessed mortality risks after taking the known hazards of stimulant diet pills into account.

In short, the causal links between high and low body mass and increased mortality remains highly speculative. We actually

know little about why the very thin and the very heavy are more likely to die than those in the 'normal,' 'overweight', and 'Type I obese' (BMI = 30–34.9) categories, and it is likely that there are multiple causal pathways across the weight spectrum. For instance, intervening with dietary supplementation to cause weight gain in underweight elderly persons has been shown to reduce mortality and prolong survival.^{19–21} Meanwhile, equivalent data showing that weight loss in obese persons reduces mortality are lacking.

Claim #3: 'The data linking overweight and obesity to adverse health outcomes are well established and incontrovertible.'²²

When the weakness of the epidemiological link between BMI and health risk is pointed out, it is sometimes asserted that BMI is an inexact measure of adiposity and that high levels of body fat, rather than high body mass *per se*, represent the real health risk. Yet when epidemiological studies have compared BMI with percentage of body fat as a marker for disease risk, BMI is consistently superior to percentage of body fat.^{23–25} This suggests that body build rather than fatness may be the source of risks associated with high BMI. Despite much speculation, very little evidence has been produced regarding the question of exactly how adiposity is supposed to cause disease. With the exception of osteoarthritis, where increased body mass contributes to wear on joints,²⁶ and a few cancers where oestrogen originating in adipose tissue may contribute,²⁷ causal links between body fat and disease remain hypothetical. It is quite possible, and even likely, that higher than average body fat is merely an expression of underlying metabolic processes that themselves may be the sources of the pathologies in question. For example, much evidence suggests that insulin resistance is a product of an underlying metabolic syndrome that also predisposes persons to higher adiposity because compensatory insulin secretion promotes fat storage. Modern molecular genetics confirms the thrifty gene hypothesis that mutations favouring fat storage and survival of famine also confer risk of diabetes.²⁸ Thus, obesity may be an early symptom of diabetes rather than its underlying cause.

The claim that adiposity is itself pathological is also belied by the results of interventions that remove body fat from their subjects. For instance, a recent study involved removing an average of 10 kg of body fat (by liposuction) from 15 female subjects. The study found no improvements in any health markers over the next 10–12 weeks, during which time the women were contacted weekly by researchers, to reinforce the importance of not changing their diet or physical activity.²⁹ This contrasts with the significant improvements in health associated with much smaller amounts of fat loss produced by programmes designed to decrease weight through lifestyle modification. Such contrasting results suggest strongly that the diet and exercise modifications undertaken by the subjects in these programmes, rather than any subsequent loss of body fat, are the causes of the observed health improvements.

Indeed, disentangling the presumed cause-effect link between body fat and 'weight-related' health problems is fairly straightforward. Exercise and nutrition can effectively reduce blood pressure—an effect that is independent of changes in body weight.^{30,31} In the DASH trial the reductions in blood pressure

among participants with hypertension were comparable with those achieved with pharmacotherapy. Blood lipids can also be improved with changes in exercise and diet, largely independent of changes in body weight or body fat.^{32,33}

Improvements in insulin sensitivity and blood lipids as a result of aerobic exercise training have been documented even in persons who actually *gained* body fat during the intervention.^{32,34} This outcome is entirely inconsistent with prevailing beliefs about body fat and health. It is also important to note that these are not new findings. Despite having been available to the scientific community for 35 years, these 'non-conforming' findings remain largely ignored.

So too have the data showing that some body fat depots, particularly subcutaneous fat on the hips and thighs, may actually provide significant health benefits.^{35–37} Thigh and hip fat in particular have been reported to be associated with lower plasma triglycerides and higher HDL-cholesterol levels.^{35,37} The researchers in one report noted that, 'the total amount of fat in legs and hips was negatively correlated with risk of cardiovascular disease'.³⁶ In the Nurses' Health Study, women who were overweight to extremely obese (BMIs between 25.2 and 48.8) with large hips and small waists had a coronary heart disease risk that was only one-half that of women of about average, or slightly less than average, weight (BMIs between 22.2 and 25.2) who had small hips relative to their waists.³⁷ In other words, the build or shape of the body seems to matter more than its fatness.

That some body fat depots are actually protective may explain the published documentation of 'metabolically normal' obese persons, i.e. 'fat' men and women with entirely normal metabolic profiles.³⁸ Such individuals are most likely at their own natural healthy weight, and do not need 'treatment'.³⁹ This illustrates a more general point: discussions about obesity and overweight as a health risk tend to treat weight as a health behaviour, akin to smoking.^{40,41} Thus overweight and obesity are commonly referred to as 'preventable causes of illness'. Yet the relationship between behaviour and weight is complex, and intertwined with immutable factors such as genetics, and body build and shape. The average individual's control over his or her weight is limited at best. This brings us to Claim #4.

Claim #4: Significant long-term weight loss is a practical goal, and will improve health.

At present, this claim is almost completely unsupported by the epidemiological literature. It is a remarkable fact that the central premise of the current war on fat—that turning obese and overweight people into so-called 'normal weight' individuals will improve their health—remains an untested hypothesis. One main reason the hypothesis remains untested is because there is no method available to produce the result that would have to be produced—significant long-term weight loss, in statistically significant cohorts—in order to test the claim. It is particularly striking that studies that have found health benefits associated with various levels of weight loss generally record no dose response: in other words, people who lose a small amount of weight, or even gain weight, get as much health benefit from the intervention as those who lose larger amounts.

Data from the National Health Interview Survey (follow-up from 1989 through 1997) illustrate the point. Among overweight

and obese men and women, with and without type 2 diabetes, those who reported trying to lose weight (but without success) experienced a reduction in mortality rate that was the same as, or greater than, those who reported that they were successful at weight loss. In other words weight loss itself did not appear to be beneficial.⁴² Indeed, in this same study, weight loss was associated with a mortality hazard ratio of 3.36 and weight cycling with a hazard ratio of 1.83.¹⁰ By contrast, obese people with stable body weight had no increase in mortality.

On the whole, body weight seems like a poor target for public health remediation, particularly in the absence of any safe or effective tools for weight loss. Furthermore, many of the tools that are currently employed towards that end (diet drugs, weight loss surgery, eating disordered behaviour, fad diets, and the chronic weight cycling they induce) have serious side effects, up to and including death. Thus public health interventions designed to lessen rates of obesity and overweight are striving to achieve a presently unachievable goal of unknown medical efficacy. In contrast, as noted above, many studies have found striking health benefits associated with lifestyle changes that produce little or no long-term weight loss. Furthermore, dozens of double blind randomized controlled clinical trials have shown that obese patients are protected from death and heart disease by lipid lowering and antihypertensive medications, without losing any weight whatsoever. One class of drugs, the thiazolidinediones, improves multiple risk factors in obese diabetics while causing significant increases in body fat.⁴³ Under such circumstances, for public health agencies to focus on trying to make people thinner, at the potential expense of initiatives that will improve lifestyle but are unlikely to produce significant long term weight loss, seems grossly inefficient.

Social and political contributors to the obesity panic

Despite the lack of scientific data supporting the central claims of the war on fat, overweight has been a growing object of governmental and popular concern. In recent years, claims that obesity is a serious public health problem on both a national and international level have become epidemic. For instance, between 1980 and 2004, media attention to obesity increased exponentially, from 62 articles published in the Lexis-Nexis US News Sources with 'obesity' in the headings, lead paragraphs, or key terms in 1980, to over 6500 in 2004.⁴⁴ If such heightened concern does not reflect scientific reality, what is driving it?

Part of the answer may lie with overlapping (and often conflicting) set of economic interests among various public health constituencies. For example, many of the leading obesity researchers who have created the official standards for what constitutes 'overweight' and 'obese' have also received sizable funding from the pharmaceutical and weight-loss industries. These obesity researchers also manage weight loss clinics and have an economic interest in defining unhealthy weight as broadly as possible, by overstating the hazards of obesity, and thus providing justifications for regulatory approvals, as well as for government and insurance industry subsidization of their products. In particular, organizations like the International Obesity Task Force (which has authored many of the WHO reports on obesity) and the American Obesity Association (which

has actively campaigned to have obesity officially designated as a 'disease') have been largely funded by pharmaceutical and weight-loss companies. Notably, although expert panels on obesity are largely devoted to evaluating epidemiological evidence and claims, qualified epidemiologists are almost never included as members. In addition, government health agencies, like the Centers for Disease Control and Prevention in the United States, have promoted the urgency of the 'obesity epidemic' while lobbying for greater programme funding and policy setting authority.

Targeting obesity has support across the political spectrum. In the US, discussions of the supposed obesity epidemic usually take place within the context of a larger discussion, which assumes that the increasing weight of the population is a sign of increasing moral laxity and that overweight and obesity are playing a significant role in driving up health care costs. This linkage is attractive for those who are ideologically committed to a focus on 'individual responsibility', rather than on structural factors that continue to drive health care costs ever upward, and leave one out of every seven Americans without health insurance of any kind.⁴⁵ Anxieties about increasing weight resonate with those on the left of the political spectrum as well, who tend to interpret the 'obesity epidemic' as both a by-product and a symbol of rampant consumer overconsumption and greedy corporations.⁴⁶

The exponential increase in mass media attention to obesity in the US and abroad seems to have many of the elements of what social scientists call a 'moral panic'. Moral panics are typical during times of rapid social change and involve an exaggeration or fabrication of risks, the use of disaster analogies, and the projection of societal anxieties onto a stigmatized group.^{47,48} Despite the very weak evidence that obesity represents a health crisis, scientific studies and news articles alike continue to treat the population's weight gain as an impending disaster. A content analysis of 221 press articles discussing scientific studies of obesity found that over half employed alarming metaphors such as 'time bomb'.⁴¹ This same study also found that >60% of the news blamed obesity on individual choices, while only ~30% discussed any structural factors that might influence weight gain. Articles that reported on blacks or Latinos were over eight times more likely than articles that did not blame obesity on bad food choices, and over 13 times more likely to blame it on sedentary lifestyles, while articles reporting on the poor were four times as likely as other articles to blame obesity on sedentary lifestyles. Such findings lend support to the theory that talk of an 'obesity epidemic' is serving to reinforce moral boundaries against minorities and the poor.

Public opinion studies also show that negative attitudes towards the obese are highly correlated with negative attitudes towards minorities and the poor, such as the belief that all these groups are lazy and lack self-control and will power. This suggests that anxieties about racial integration and immigration may be an underlying cause of some of the concern over obesity.⁴⁹⁻⁵¹ Consider the apocalyptic conclusion of a cover story in a prominent American magazine:

What do the fat, darker, exploited poor, with their unbridled primal appetites, have to offer us but a chance for we diet-and-shape-conscious folks to live vicariously? Call it boundary envy. Or rather, boundary-free envy ... Meanwhile in

the City of Fat Angels, we lounge through a slow-motion epidemic. Mami buys another apple fritter. Papi slams his second sugar and cream. Another young Carl supersizes and double supersizes, the supersizes again. Waistlines surge. Any minute now, the belt will run out of holes.⁵²

Previous work indicates that moral panics often displace broader anxieties about changing gender roles.^{49,53} While this hypothesis deserves further research, a recent advertisement that ran in a major American newspaper suggests that this may be at play in the obesity panic. This advertisement blames '30 years of feminist careerism' for an epidemic of childhood obesity and diabetes: 'With most mothers working, too few adults and children eat balanced, nutritious, portion-controlled home-cooked meals. Within a generation 50% of Americans will become diabetic, creating a medical and financial nightmare likely to crush our healthcare system.'⁵⁴ Deeply flawed epidemiological arguments about the dangers of obesity lend credibility to such scientifically baseless claims.

Yet despite all of the moral connotations ascribed to weight gain, we have little idea exactly why people weigh somewhat more now than they did a generation ago. Not surprisingly, some works suggest that increasing caloric intake and decreasing activity levels, in some combination, are sufficient explanations for this trend.⁵⁵ However, other works suggest that some portion of the population's weight gain can be attributed to smoking cessation,⁵⁶ which runs counter to the assumption that the country's weight gain is evidence of both moral laxity and a harbinger of declining overall health.

So what if the so-called 'obesity epidemic' is largely an illusion? What if higher than average weight turns out to have neither much medical nor moral significance? The answer to these questions, all of which we believe are strongly suggested by the epidemiological literature, go far beyond the issues of body mass and health. The current scientific evidence should prompt health professionals and policy makers to consider whether it makes sense to treat body weight as a barometer of public health. It should also make us pause to consider how propagating the idea of an 'obesity epidemic' furthers the political and economic interests of certain groups, while doing immense damage to those whom it blames and stigmatizes.

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Commentary: Understanding the epidemiology of overweight and obesity—a real global public health concern

Soowon Kim¹ and Barry M Popkin^{2*}

¹ Department of Family and Community Medicine, Center on Social Disparities in Health, University of California, San Francisco, CA, USA.

² Department of Nutrition, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA.

* Corresponding author. Carolina Population Center, University of North Carolina, 123 W. Franklin Street, Chapel Hill, NC 27516, USA. E-mail: popkin@unc.edu

Introduction

Campos and his collaborators raise some useful and important questions about the way to understand the impact of overweight and obesity on health.¹ Especially, bringing attention to some of the complexities in overweight/obesity and health relationships and covert financial interests involved in obesity