Commentary: Understanding the epidemiology of overweight and obesity – A real global public health concern

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

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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

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research and related promotion activities is noteworthy. At the same time, however, they ignore some basic pathways linking dietary and physical activity patterns to weight dynamics to health. Furthermore, they selectively examine the literature and, as is easily done, arrive at the conclusion that there is much ado about nothing in the 'obesity epidemic or pandemic'. A different reading of the way to study in a causal manner the role of these factors as they affect health provides us with a very different conclusion.

Rapidly changing diets and reduced physical activity levels have led to a marked increase in the prevalence of diet-related chronic diseases in both developed and developing countries.[2,3] The pathway linking weight status to health is complex. Figure 1 provides a simplified view of the major research relating the key factors in this pathway. How one studies this pathway clearly affects one's conclusions. The manner in which obesity fits into this causal pathway and the methods for studying this are at the core of the discussion. Considerable progress in the scientific study of obesity's determinants and consequences has led to a growing understanding of the responsible causal pathways, risk factors, and mechanisms. In particular, there is now strong evidence relating dietary factors and physical activity levels to the risk of obesity, hypertension, certain cancers, diabetes, stroke, and other coronary heart disease (CHD).

Much of the debate with Campos *et al*. lies in their selective use of research on these pathways and misunderstanding of basic epidemiological principles. Clearly there are a large number of ways that diet directly affects the health outcomes noted in Figure 1 (pathways B1 and B3). The same is true for physical activity (pathways C2 and C3). An important thing to note is that some of these factors work through obesity (pathways B2, C1, D1, D2, and D3), as well as independently affect disease. Diabetes is a critical example. One of the foremost researchers in the diabetes world summarized some of the major relationships in two broad articles on the topic. In each he showed how increases in weight could directly affect diabetes independent of the physical activity effects. He then went on to show independent physical activity pathways.[4,5] Later we review further literature on the obesity-disease topics but these overviews by Zimmet *et al*.[5] provide some sense of the vast literature underlying each relationship and how a selective use of literature could miss the whole picture of the pathway and lead to a distorted conclusion.

There certainly are important issues raised by Campos *et al*.[1]—some of which need careful review and do lead to areas where further research is needed. However, in contrast to their four main claims, our position is clear that increase in obesity and its health consequences are real, and the scientific community needs to provide more responsible solutions to this serious public health concern, instead of nullifying existing scientific evidence based on discriminatory use and fallacious interpretation of literature.

This article proceeds by discussing each of the points raised by Campos *et al*.[1]

Global increases in weight are real!

It is asserted that the weight increases are modest. What is implied is that weight increases are occurring in a small proportion of the population and these changes are small. On the one hand, many of Campos *et al*.'s inferences to 'insignificant'

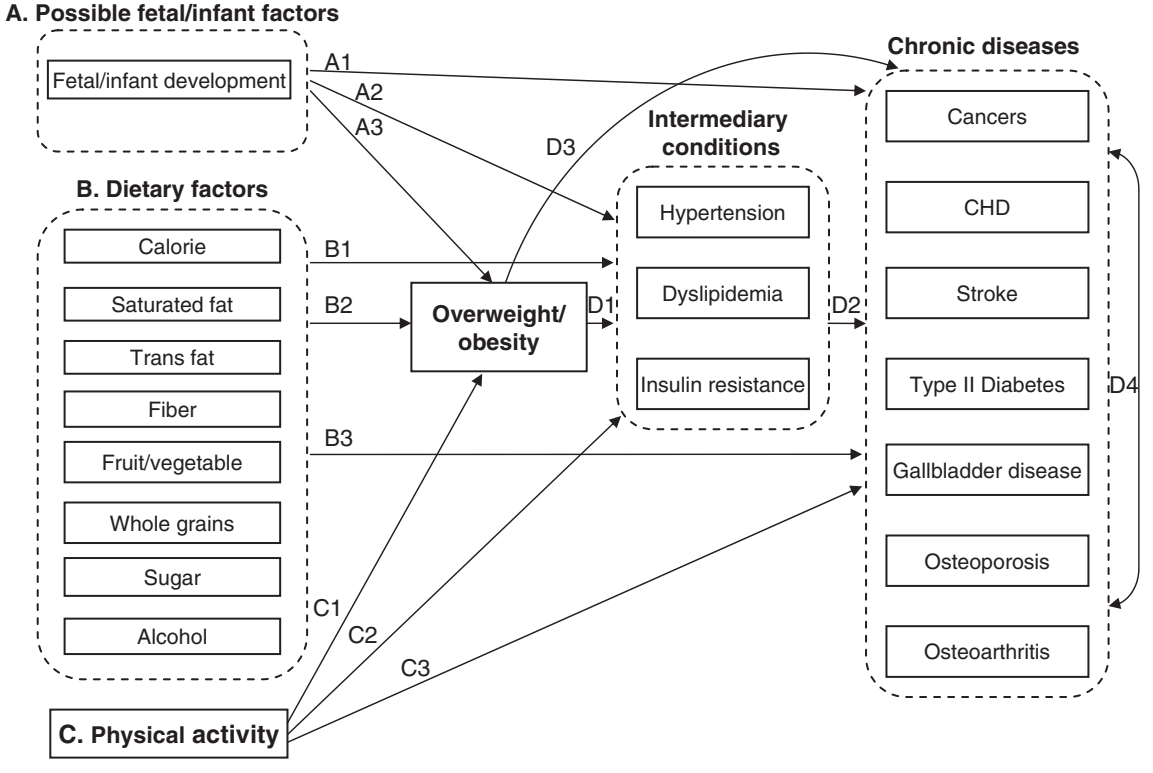

Figure 1 Key pathways for diet, physical activity, and obesity on nutrition-related non-communicable diseases (note direction of effects are not presented)

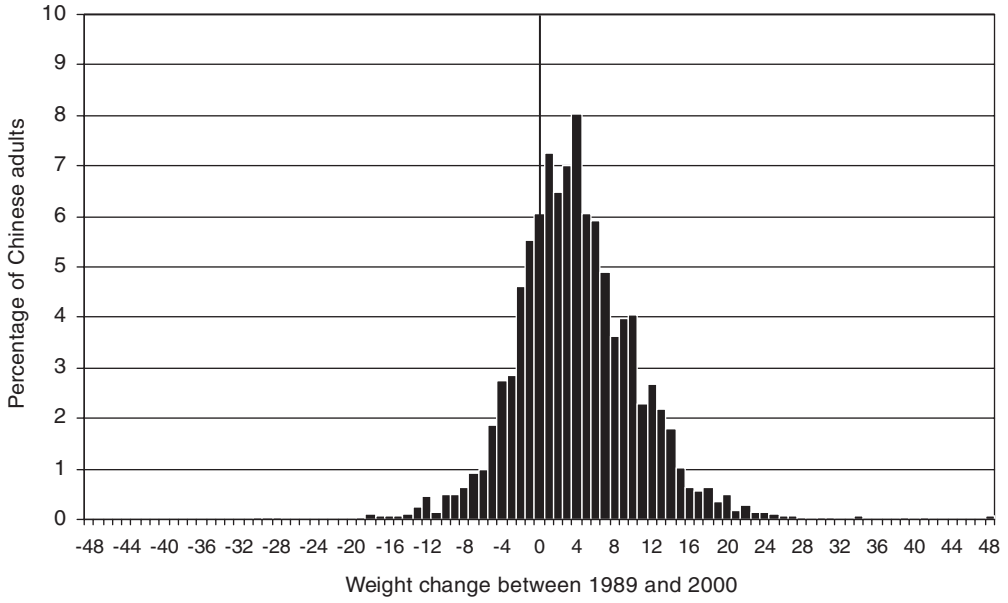

Figure 2 Distribution of weight change between 1989 and 2000 (in kg) in Chinese adults aged 20–45 ($n = 2739$). *Source:* China health and nutrition surveys 1989 and 2000

weight change over time based on cross-sectional trend analyses at the population level reflect ecological fallacy.[6] For example, their statement about the *average* American's weight gain explained by a few calories a day or a few minutes of walking could be very misleading, because average weight gain at the population level does not necessarily equate with weight gain at the individual level. The actual weight change of a population comprises a wide range of distributions. There may be a portion of the population who lose or do not change weight over time that would pull the mean weight change of the population down, which would make rather significant weight gains in some other groups of the population look trivial; ignoring these dynamics within a population may mask true changes that are happening among individuals. Also, what Campos *et al.*[1] refer to as cessation of increase in weight based on the comparison of 1999–2000 and 2001–02 data is misleading, as it may simply be a reflection of sampling error, especially for population subgroups with relatively small sample size.[7]

In addition, what has been referred by Campos *et al.* as a subtle shift in BMI [body mass index or (weight in kilograms)/(height in meters)^2] observed in the nationally representative data actually portends a remarkable increase in overweight/obesity. As the weight distribution shifts to the right, a greater proportion (the majority of people who belong to the centre of the distribution curve) of people enters into a higher range of BMI.

Data from around the world show radical increases in obesity.[8,9] We suggest looking at available data that can explain some of the real shifts to assess the global epidemic of obesity. We begin with the example of China, utilizing unique longitudinal data on Chinese adults (20–45 years old at baseline in 1989) from nine provinces.[10] Our analysis shows that among adults, the weight change during the 11 year follow-up period (from 1989 to 2000) indeed has quite a wide distribution, and it is the *majority* of the people (73%) who gained weight (Figure 2). The average weight gain among those who gained weight during the follow-up period is 7.0 and 6.3 kg for men and women, respectively, uniformly across all baseline BMI categories. This is in contrast to Campos *et al.*'s first set of claims. Campos *et al.* in fact did not look at such longitudinal data to support their claims of a significant weight gain in only a minor portion of the people and people just crossing the 'border' to become classified as overweight and obese compared with a generation ago.

This China example is important as it provides an example of the type of weight shifts we see in Mexico, many other South American countries, most Middle Eastern and South-east and East Asian countries.[8]

When we shift to higher income countries, there have been different types of shifts in the US and Russia and the few other more developed countries where overweight was very high. In these countries the increases in the past few decades are not as great in the normal weight category compared with the morbidly obesity categories. Among adults, in the US, the most profound is the large increase in the proportion of people with BMIs >35 and 40. Among US adolescents and children, however, the shifts have been across the BMI distribution and thus quite different from what has been observed among adults. Campos ignores these patterns.[7,11,12]

Health consequences of obesity are real and serious!

The recent two articles by CDC scholars—first Mokdad *et al.*[13,14] cited death rates from obesity of 400 000 and then Flegal *et al.*[15] showed 112 000 deaths—really created their own drama that most in the scholarly field dismissed. The death estimates from the first article were only partially adjusted for confounding factors. They also did not account for variation by age in the relation of body weight to mortality. The Flegal *et al.*[15] study with its attribution of 112 000 deaths to obesity, while disputed for some questionable assumptions that might increase considerably

the number of deaths, is well done and believable. What is important is that these are a lot of deaths but death probably does not describe the major health problems of obesity, which are morbidity, disability, hospitalization, and earlier entry to nursing homes. The quality of life is seriously compromised in obese individuals, but these health outcomes have only recently been measured and documented in depth. We discuss these issues later.

Campos is correct to note that the relationships between levels of obesity and overweight are very complex and the final answer on these relationships is not very clear.[16] Definition of optimal BMI may need to be continuously challenged based on comprehensive scientific evidence. At the same time, they are wrong in stating the studies of obesity and mortality should control for dietary and activity patterns—the very factors that cause obesity. Just as you should not control for diabetes (a mediator in this case, which is critical to the causal chain, and will attenuate the association of the exposure with the outcome if treated as a confounder) in trying to understand how obesity affects mortality, one must not control for the underlying determinants of energy imbalance and obesity. Also, Campos *et al.*'s comment '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' shows poor understanding of epidemiological methods, as *controlling* multiple factors in analyses of observational studies does *not* convert non-experimental studies to experimental studies, from which only can we draw causal inferences.[17]

Systematic reviews of the literature show clear links between obesity and adverse health and economic outcomes!

Literature supporting increased morbidity in obese individuals is quite overwhelming. Epidemiological studies have shown the links between obesity and adverse health outcomes, and physiological mechanisms are provided to support these relationships. Overweight persons face not only the full array of health problems noted in Figure 1 but they also retire earlier, go at younger ages into nursing homes, have higher absenteeism rates, and are more likely to be disabled. Below we briefly summarize some of the major obesity–morbidity relationships—direct effect of obesity on a chronic health condition and further links between the health condition to other health issues, if present.

Hypertension: Increased blood pressure, as body weight increases, has been observed in both normotensive and hypertensive individuals. The pathogenesis of obesity-related hypertension is supported by the physiological mechanisms that leptin, free fatty acids, and insulin—whose levels are increased in obesity—may act individually and synergistically to stimulate sympathetic activity and vasoconstriction. In addition, obesity-induced insulin resistance and endothelial dysfunction may act as amplifiers of the vasoconstrictor response. Increased renal tubular re-absorption of sodium may also occur, caused by an increased renal sympathetic nerve activity, direct effect of insulin, hyperactivity of rennin-angiotensin system, and possibly by an alteration of intrarenal physical forces.[18] Both obesity and hypertension predispose to cardiovascular morbidity and mortality.[19] In turn, a history of hypertension is shown to increase the risk of type 2 diabetes independently of other known risk factors, including obesity.[20] Elevated blood pressure is also a powerful risk factor for CHD and a significant predictor of mortality from stroke—for men and women.[21]

Dyslipidaemia [imbalance in total, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, and triglycerides]: Obesity has been associated with increased levels of triglycerides and decreased HDL-cholesterol, both of which are independent risk factors for cardiovascular disease.[22] Elevated levels of total cholesterol, LDL-cholesterol, and triglycerides are major risk factors for CHD in both men and women,[23] whereas HDL levels are correlated inversely with the risk of CHD.[24]

Insulin resistance (glucose intolerance, impaired glucose tolerance, hyperinsulinaemia): Insulin resistance has been strongly associated with overweight and obesity in many epidemiological studies.[25] Hyperinsulinaemia, in turn, is believed to increase the risk of colon cancer by directly promoting colon carcinogenesis and stimulating insulin-like growth factor-I receptors.[17,26,27] A similar mechanism is provided for the case of endometrial cancer.[28]

Cancers:

(i) *Colon cancer:* Higher BMI is associated with increased risk for colon cancer, with the association stronger for larger adenomas and for men. These may suggest an effect of factors related to adiposity on the promotion of cancer and a possible counteracting effect on these factors by oestrogens.[29]

(ii) *Breast cancer:* A contrasting pattern has been shown by menopausal status. Higher body weight, especially higher adult weight, is associated with increased breast cancer risk among post-menopausal women.[29]

(iii) *Oesophagus cancer:* An increased incidence of gastric reflux in persons with high BMI has been proposed as the underlying cause of more than a 2-fold increase in the risk of oesophageal cancer.[29]

(iv) *Endometrium cancer:* Convincing evidence from epidemiological studies shows a linear increase in the risk of endometrial cancer with increasing adult obesity.[25,29] A hormonal mechanism is provided to support the association. Obese women have higher levels of serum oestrone and estradial, and decreased levels of sex hormone-binding globulin, both increasing the amount of bioavailable oestrogen. Elevated oestrogen levels stimulate endometrial epithelial cells, which is conducive to the development of cancer.[30]

(v) *Kidney cancer:* Studies conducted across nations consistently show a more than 2-fold increase in renal-cell cancer risk among obese (both men and women), compared with those of normal weight.[29] Increased levels of endogenous oestrogens in women may affect renal cell proliferation and growth by receptors present in renal cells or through paracrine growth factors.[31]

Type 2 diabetes: It is well established that excess body fat leads to increasing insulin resistance, and insulin resistance predisposes to diabetes.[22] Since obesity is characterized by a reduced number of insulin receptors and insulin resistance, the combination of epidemiological and metabolic data leaves little doubt that obesity is causally related to type 2 diabetes.[32]

In turn, remarkable similarity of risk factors for type 2 diabetes and colon cancer, coupled with a general hypothesis that hyperinsulinaemia increases the risk of colon cancer, has led to a

theory that type 2 diabetes itself, is a risk factor for colon cancer; this association has been observed in epidemiological studies.[33] Diabetes is also a risk factor for CHD and CHD accounts for much of the serious morbidity and a high proportion of the premature deaths in type 2 diabetes.[34] Diabetic men and women have a 2- to 3-fold and 3- to 7-fold increase in risk of CHD, respectively, compared with their non-diabetic counterparts.[35,36]

Coronary heart disease (CHD): Obesity has been shown to be an independent risk factor for CHD in both men and women.[37,38] In addition, as described above, obesity increases the risk of hypertension, dyslipidaemia, and diabetes mellitus—all of which are risk factors for CHD.

Stroke: BMI is a strong risk factor for total and ischaemic stroke. The pathway from obesity to stroke is thought of as being developed through hypertension, diabetes, and elevated cholesterol.[39]

Gallbladder disease: Epidemiological studies have reported an association between gall bladder disease, overweight, and obesity.[22] The pathogenesis of gall bladder disease involves numerous mechanisms that are present in obese individuals, such as excess hepatic secretion of cholesterol and subsequent supersaturation of bile, increased gall bladder volume, and blunted gall bladder contractility.[40]

Osteoarthritis: Being overweight increases the amount of force across a weight-bearing joint.[41] In addition, adipose tissue may produce atypical hormone or growth factor concentrations that affect cartilage or underlying bone, predisposing them to osteoarthritis development.[42]

Further obesity affects Years of Disability Free Life and productivity, among others. Using the Original and Offspring Framingham Heart Study, Peeters *et al.*[43] have again used the *Years of Life* concept to estimate the life expectancy for individuals, free of disability and classified by weight status. Disability was defined as limited mobility (i.e. limited walking on a level surface and walking up/down stairs) and limited activities of daily living (ADL) (i.e. limited dressing, grooming/bathing, feeding/eating, getting in and out of chairs). Among non-smokers, obese men had 5.70 (95% confidence interval (CI) 4.11–7.35) and obese women had 5.02 (95% CI 3.36–6.61) fewer years free of ADL limitations than normal weight counterparts. Regarding any mobility or ADL limitation, obese men had 6.02 (95% CI 4.35–7.61) and obese women had 5.53 (95% CI 3.76–7.34) fewer years free of limitation than normal weight counterparts. These researchers concluded, however, owing to the higher mortality in the obese and overweight groups, there was no significant difference in the years lived with disability (mobility or ADL limitation) between those overweight or obese and those with normal weight at baseline.

Minimal research has been conducted regarding the effect of obesity on the loss of productivity.[44–50] The research has examined loss of productivity in terms of disability or early retirement and days absent from work. The majority of this work was conducted in Scandinavian countries: Finland, Denmark, and Sweden.[44–48] Most of these studies (six of seven) have found obesity to be associated with disability or early retirement. One study examined sick leave histories and found an increased BMI to be associated with long-term sick leave.[46] Another study conducted in the US found obesity to be associated with the combined outcome of limitations in the kind or amount of paid work, kind or amount of housework, and kind or amount of any activity.[49] Finally, another study found obesity to be associated with days spent ill in bed; however, this association may vary depending on age.[50]

Scholars have studied the economic burden of obesity both in private and public sectors and found the costs to be considerable.[51–54] The major public payers—Medicare and Medicaid—finance about half of these costs,[53] so the costs of obesity are not borne just by the obese individuals and their families, but pose a significant externality on economic systems.

We could go on to review other ways obesity affects an individual—not only physically but also psychologically—and society but stop here. The key issue is to point out that these effects are fairly consistent and profound. To state otherwise ignores a rather substantial literature to pick out a few self-selected references.

Weight loss brings health benefits!

Contrary to Campos *et al.*'s last claim, we find ample evidence that weight loss is beneficial in a number of health parameters and disease outcomes. This evidence, with numerous epidemiological findings, supports a causal link between weight and health. Weight loss, even a modest one, is shown to reduce or remove weight-related disorders in obese individuals[55] such as type 2 diabetes,[56] hyperlipidaemia,[57] and hypertension.[58] There is a direct link between what causes weight loss (i.e. improvement in diet and increased physical activity) and health benefits, but literature also shows an independent effect of weight loss on health[56] and provides physiological mechanisms that support the effect of weight loss *per se*.[55] Campos *et al.*[1] again neglect to see the whole pathways.

Risks related to more aggressive methods of weight loss are real. This certainly brings an emphasis on primary prevention of overweight and obesity. Unless we stop the shift in weight now, public health challenges related to obesity that we are already facing now will grow even greater.

Financial interests and the obesity epidemic

The fact that many countries in all regions of the world have stated that obesity and its linkage with diabetes, heart disease, and stroke is utilizing excessive medical care facilities and represents a major health threat to its population points to the universality of the problem. Furthermore, it is clear that the news media, itself alarmed by the vast increase in overweight and obesity globally and the health consequences of this, has increased exponentially articles on the topic. This is only natural.

Campos *et al.*[1] are correct that there are many with direct financial interests involved in research and promotion activities related to the obesity epidemic. On the one hand, there are organizations funded by the food industry pitted against all discussion of issues such as portion sizing, 'unhealthy or bad foods', excessive added sugar, excessive use of hydrogenated and saturated fats in the processed food industry, among others. On the other hand the drug industry has played a major role in funding several major international groups that attempt to promote programmes and knowledge related to obesity prevention and treatment. Similarly politics, particularly from

the food industry, have affected all dietary guidance that emanates from the US Department of Agriculture. The US does not have free-standing independent organizations unfettered by the need to obtain funds that offer such guidance. There are few major organizations in any country except for groups such as the Center for Science in the Public Interest and a few others in higher income countries who could be said to be unfettered by funding from the food industry or the drug industry.

As with many scholars in the food, nutrition, and obesity research world, Popkin has taken gifts and contracts from the food industry to publish a range of studies in support of and against the various issues of concern to him academically. The authors received no funding for this specific article, but Popkin certainly has taken funding from many sources, which might have affected his judgement. The authors' integrity and that of our colleagues from the public health, economics, and medical professions can only be judged by what we write and state and how open we are on such funding and how active we are in trying to counteract such biases. The source of funding might have something to do with blaming the victim vs blaming the environment. With the other 97% of all research funding coming from NIH, Popkin is much more able to blame the food and activity environment for many of the underlying causes of poor dietary and activity patterns. At the same time others who are equally independent do not feel this way so to state that source of funding is the cause of who blames the victim and who does not is a bit naïve. The food industry has been most likely to blame physical activity and the victim of poor eating decisions and call for freedom of choice. Clearly this comes from self-interest but Campos *et al*. did not talk about such self-interests.

We do know that physical activity patterns and dietary patterns have changed. We cannot fully ascribe how much of the increased energy intake comes from portion sizing, energy density, added sweeteners in the diet, and so on. All of these factors appear important. Nor can we ascribe how much of the reduced energy expenditure comes from reductions in activity at occupational activity, housework, transportation, and leisure.

Discussion

To state that the obesity increase is an illusion and that increased weight and adiposity does not contribute to poor health and functioning is really to misuse the vast literature that shows the large increases in weight and BMI dynamics globally[3,8,9,59] and also the important effects of these changes on global disease profiles.[60]

Campos *et al*. neglect that by falsely invalidating existing evidence of increasing obesity and its health impact, they further harm the most vulnerable subgroups in the populations; it is often the poor, people with lower socioeconomic status, and some socially disadvantaged racial/ethnic groups that disproportionately suffer from the obesity epidemic,[7,61] both adults and children alike.[62] When the alarming increases in weight nationally and globally are ignored, these already marginalized and stigmatized subgroups, socially and health-wise, will suffer even greater from obesity-related morbidity and mortality. We feel that treating a real health problem as non-existing is equally irresponsible as blaming the victims only.

The reality is that obesity is generated by lifestyle behaviours that are largely affected by the environment in which people live.[63] Unless our environments are not altered to promote healthy living, the increasing weight will continue to grow, and so will obesity-related morbidity and mortality. Providing 'freedom' to choose unhealthy lifestyles that will lead to obesity and related health problems is not what a healthy society should do. We encourage the scientific community to join in the efforts to create more responsible solutions for the obesity epidemic at the societal level, rather than denying the problem or being swayed by parties whose number one priority is not necessarily improving public health.

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Commentary: There *is* a public health crisis—its not fat on the body but fat in the mind and the fat of profits

Susie Orbach

Campos *et al.*[1] persuasively rebut the claims of what Alicia Mundy[2] calls 'Obesity Inc'. These are those industries and the supporting media structures, which would have us believe that Obesity, with a capital O, is at epidemic proportions and is swamping our health budgets; that obesity and overweight are indicators for hastened mortality; that higher than average adiposity is a direct cause of nefarious, dangerous diseases; and that long-term weight loss is beneficial and achievable.

The war on obesity is clearly misguided on scientific and statistical grounds. So the question is, why are the ideas associated with it, so compelling? Why do we believe that obesity and overweight are bad for us? How have these two words, fat and overweight, taken on the weight of moral disapproval? How has fat and overweight become demonized? What are the more hidden and less recognized consequences of this belief system, including the psychological injury to millions—some of whom are fat or overweight and some of whom are 'normal' weight and decidedly thin, but they believe, however, that they are too fat? And, briefly, what can be done to re-orientate this so-called public health crisis into an area in which it might do some good rather than increase harm?

How has fat become demonized?

Campos *et al*. pinpoint some of the players who stand to profit from the belief that there is an obesity crisis. These are the pharmaceutical companies searching for their next big drug, the diet companies, and all those industries that foist their wares on a public primed to believe that but for constant vigilance they would slide into obesity. These players and their handmaidens in the media have created the kind of blanket of insecurity that has come to infect our relationship to our body, to our eating, and to our appetites.

Often unrecognized are those industries—the fashion, cosmetic, and media industries—whose dreams and whose products have constructed particularly narrow notions of the kinds of bodies we should live in and from. Purposefully or unwittingly these style industries demonize fat and 'overweight' (a category we should question in and of itself, over what?). They promote thin as the only body to have and this has a devastating impact on the lives of girls and women, leading them to interrupt their appetites in order to achieve bodies that conform to the bodies projected and promoted by those industries.

The export of body hatred

The style industries love to represent themselves as simply a bit of fun that people can take or leave. This is disingenuous. The money these industries spend on positioning their wares as essential routes to achieve desirability and glamour demonstrates their wish to captivate ever-bigger audiences for their 'must have' products. We now know unequivocally that the result of their promotion of thin is having serious psychological and physical impacts on girls and women. Consider for the moment one of the hidden aspects of global culture: the export of body

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