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Public Health and Moral Panic: Sociological Perspectives on the 'Epidemic of Obesity'

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Introduction

In 2007, the number of people worldwide who were chronically underfed reached 923 million, some 75 million more than in 2003–2005 (FAO, 2008). By contrast, in 1999, over 1 billion adults and approximately 18 million children were overweight or obese (WHO, 2000). Neither rising food prices, nor rising food insecurity among the world's poor – especially landless and female-headed households (FAO, 2008) – appear to be slowing the spread of weight gain and obesity, a trend that has been associated, in particular, with populations undergoing socio-economic transformations associated with urbanization, modernization and globalization (WHO, 2000).

While the term 'epidemic' has been used to describe changes in the prevalence of obesity in developed countries such as the US and UK since at least the early 1990s, the World Health Organization's Consultation on Obesity in 1997 drew attention to the increasingly global nature of weight gain and obesity, observing that:

As standards of living continue to rise, weight gain and obesity are posing a growing threat to health in countries all over the world ... both developed and developing ... and affecting children as well as adults. Indeed, it is now so common that it is replacing the more traditional public health concerns, including undernutrition and infectious disease, as one of the most significant contributors to ill health (WHO, 2000, pp1–2).

Further, the paradoxical coexistence of undernutrition and weight gain should not be viewed exclusively at the global level. Households containing both overweight and underweight individuals (generally obese adults and malnourished children) are common in middle-income countries (Prentice, 2006; see also Doak et al, 2005). At the same time, the diet of obese individuals is often nutritionally inadequate due to the predominance of energy-dense foods that are high in fat and/or sugar, but low in fibre, vitamins and minerals (Markovic and Natoli, 2009).

What does it mean, though, to be overweight or obese? These conditions are generally defined in terms of fat accumulation that is sufficient to increase the risk of psychosocial and/or medical morbidity. There are several ways in which this may be calculated. The body mass index (BMI) has been most widely used in epidemiological research since the 1970s as a convenient, acceptably accurate and low-cost measure of adiposity (or fatness) (Eknoyan, 2008). BMI is calculated as weight (kg)/height (m)². In adults, four BMI categories are used: underweight (<18.5), normal weight (18.5–24.9), overweight (25–29.9) and obese (>30) (WHO, 2006). By contrast, international measures for children and adolescents are age- and gender-specific to allow for the significant variability in age-related growth patterns. In adults, the interpretation of risk associated with BMI may also differ for different populations – especially for Asian and Pacific populations (WHO, 2006). The use of BMI in public health has been challenged, however, on the basis that its generalizability to non-Anglo Saxon populations and its sensitivity as a measure of adiposity are questionable. Although BMI correlates with total body fat, it is poorly correlated with fat distribution – particularly excess visceral abdominal fat, which is strongly associated with many obesity-related conditions such as cardiovascular disease, Type 2 diabetes and colon cancer (Field et al, 2001; Larsson and Wolk, 2007).

Despite these limitations, weight gain and obesity as defined by BMI are associated with increased risk of developing a number of health problems (see Table 9.1) including cardiovascular disease, Type 2 diabetes, osteoarthritis and some cancers. The risk of disease increases with increasing BMI (WHO, 2006). In Australia, the most significant obesity-related conditions (in terms of burden of disease) are cardiovascular disease (including coronary heart disease, stroke, hypertension, heart failure and peripheral vascular disease) and Type 2 diabetes. Cardiovascular disease is the leading cause of death and the second leading cause of disease burden in Australia (AIHW, 2008), with obesity attributed as the primary cause in 21.3 per cent of cases (Diabetes Australia, 2008). Type 2 diabetes is projected to be the leading specific cause of disease burden for males – and the second leading cause for females – by 2023 (AIHW, 2008). Similarly, obesity is recognized as the primary cause of 23.8 per cent of Type 2 diabetes cases (Diabetes Australia, 2008).

With such a substantial burden of disease attributed to weight gain and obesity, it is far from surprising that these conditions are so frequently framed in both the scientific literature and the mass media as an epidemic. Indeed, we would argue that the framing of obesity as an epidemic was so pervasive by

Table 9.1 *Relative risk of health problems associated with obesity*

| Relative risk | Associated with metabolic consequences | Associated with weight |
|----------------------|---|-----------------------------|
| Greatly increased | Type 2 diabetes | Sleep apnoea |
| | Gall bladder disease | Breathlessness |
| | Hypertension | Asthma |
| | Dyslipidaemia (raised blood lipids such as cholesterol) | Social isolation/depression |
| | Insulin resistance | Daytime sleepiness/fatigue |
| | Atherosclerosis | |
| Moderately increased | Coronary heart disease | Osteoarthritis |
| | Stroke | Respiratory disease |
| | Gout/hyperuricaemia | Hernia |
| | | Psychological problems |
| Slightly increased | Cancer (breast, endometrial, colon) | Varicose veins |
| | Reproductive abnormalities | Musculo-skeletal problems |
| | Impaired fertility | Bad back |
| | Polycystic ovaries | Stress incontinence |
| | Skin complications | Oedema/cellulitis |
| | Cataract | |

Source: Australian Government (2009)

the late 1990s that it became the dominant narrative, or discourse, on weight gain and obesity. Researchers and medical professionals used the term with little or no qualification: the 'epidemic of obesity' screamed out of newspaper headlines. Yet, in more recent years, it seems that for every headline alerting readers to the spread and dangers of obesity there is another that questions the veracity of the epidemic narrative. For example, in January 2009, most of Australia's major newspapers carried stories claiming that the obesity epidemic was an illusion and, more specifically, that the childhood obesity epidemic was a myth. In the following month, the very same newspapers claimed, in contrast, that the childhood obesity epidemic was now affecting babies. While the latter claims are no less pervasive, the obesity epidemic narrative is increasingly used as a point of departure from which to debate the accuracy and consequences of claims regarding increasing body weight. The pervasiveness of this narrative, and the contestable knowledge claims associated with it, are worthy of sociological attention.

Our objectives in this chapter are twofold. The first is to review the existing evidence regarding changing patterns of weight gain and obesity with a view to establishing whether or not this is an issue that has claimed the attention of sociologists and other social scientists. The second is to review sociological contributions to the understanding of weight gain and obesity, and to comment on where a sociological research agenda might productively focus. While Australian data will be the focus of our empirical attention, empirical and theoretical contributions will also be drawn from a variety of countries for comparative purposes. This analysis indicates that there is very little about the obesity epidemic that is unique to Australia, or to anywhere else.

Are we witnessing an epidemic? Changing patterns of weight gain and obesity

The 1999–2000 Australian Diabetes, Obesity and Lifestyle Study found that rates of weight gain and obesity among urban Australian adults (calculated according to BMI using measured data) were 39 per cent and 21 per cent, respectively (Cameron et al, 2002). The prevalence of weight gain was considerably higher among men (48 per cent) than among women (30 per cent), while the prevalence of obesity was slightly lower among men (19 per cent) than among women (22 per cent). These rates were comparable with those reported for other developed countries, including the UK (17 per cent for men and 21 per cent for women) and Germany (19 per cent and 21 per cent), but slightly lower than those reported for the US (28 per cent and 33 per cent). Consistent across all of these countries was a steady increase in obesity, calculated according to BMI, with age, with levels peaking in the 55–64 year age group (Cameron et al, 2002). The 1995 data for Australian children (2–18 years) indicate that 15 per cent of boys and 16 per cent of girls were overweight at that time, and a further 5 per cent of both groups were obese (Magarey et al, 2001).

There are at least five aspects of obesity and overweight prevalence data that contribute to the construction of obesity as an epidemic or crisis. First, the data indicate a considerable increase in the prevalence of obesity, which more than doubled in both the US and Australia between 1980 and 2000 (Cameron et al, 2002). Even more dramatically, the prevalence of obesity among Australian boys (7–15 years old) more than tripled between 1985 and 1995, while the prevalence among girls in the same age group increased fourfold (Magarey et al, 2001). Since 2000, the data indicate that the prevalence of obesity in Australia may have stabilized, or at least that the rate of increase has slowed (see ABS, 1997, 2006; Barr et al, 2006; Gill et al, 2009). However, this brings us to the second aspect of obesity prevalence data of relevance here: namely, that irrespective of whether rates are stable or growing, approximately one in five adults in Australia and other developed nations are now classified as obese, and a further one in two as overweight. Crudely put, a lot of people are affected by weight gain and obesity, and are therefore likely to be affected by one or more associated health problems. The third indication of a crisis is that obesity and weight gain affect some groups more than others. In Australia, such groups among the adult population include those who have not completed a tertiary education, who come from a low income household, and/or who live in an area of relative disadvantage (ABS, 2007). Indigenous women, moreover, are 1.4 times more likely to be obese than the general population of Australian women (Phillips, 2008). Residents of outer regional, remote and very remote areas are classified as obese at higher rates (23 per cent) than are residents of inner regional areas (19 per cent) and major cities (17 per cent). A recent study of two rural areas in the Australian states of Victoria and South Australia reported 30 per cent of participants as obese, and 39 per cent as overweight (Janus et al, 2007). This correlation between obesity and various forms of

social disadvantage leads some to argue that obesity needs to be seen as much as a social justice issue as a public health issue (Monaghan, 2005).

A fourth area of concern is the escalation of overweight and obesity among children, along with the recognition that overweight and obese children are more likely than their peers both to experience obesity as adults and to suffer disproportionate rates of chronic disease at younger ages (Speiser et al, 2005; Gill et al, 2009). These issues amplify the moral dimension of obesity narratives and either introduce, or reinforce, issues around parenting, maternal nutrition, advertising, schooling and so on. The fifth and final issue of relevance here is that high rates of weight gain and obesity are not solely a characteristic of the developed North, but also increasingly affect the populations of Asia, Latin America, the Middle East, Africa and the Pacific (Prentice, 2006). The so-called nutrition transition – the replacement of traditional diets, which are high in cereals and vegetables, with energy-dense Western diets – appears to accompany processes of urbanization and industrialization just as surely as do motorized transport, sedentary employment and passive entertainment (Popkin, 2001). Much like a pathogen-induced epidemic, therefore, the spread of historically high rates of weight gain and obesity has a spatial dimension, beginning in the US and thence spreading to Europe, to settler states such as Australia, and on to emerging economies and beyond (Prentice, 2006).

At a time when the proportion of residents of a given area that are now classified as obese has reached somewhere between a third and a half, the sheer visibility of seemingly excessive and potentially dangerous body fat is surely of concern. Yet, there are several aspects of the obesity epidemic narrative that have attracted criticism. One set of criticisms, which are related to the consequences of constructing the issue of changes in bodyweight as an ‘epidemic’, will be dealt with later in this chapter. Here, we are concerned with a second set of criticisms focused on the understanding of overweight and obesity from an epidemiological point of view.

To begin, it is alleged that the rate and level of increase in bodyweight has been overstated. According to Campos et al (2006) what we have seen has not been the exponential growth pattern typical of epidemics, but rather a small ‘skewing’ to the right of the population distribution of BMI – one that amounts to nothing more than an average weight gain among American adults of 3–5 kilograms over the course of a generation. In turn, these authors claim that such a small gain can be explained by the consumption of as little as 10 extra calories by an individual, the equivalent of a few minutes’ less walking every day. While this weight gain has tipped many people over the threshold BMI values that are used to classify them as overweight or obese, the argument goes that this is likely to have few meaningful consequences for health. In particular, Campos et al (2006) claim there is limited evidence that anything other than extreme obesity is associated with increased mortality and that there are more documented risks from being underweight and from what is known as ‘weight cycling’ (or yoyo dieting) than there are from being overweight (see also Monaghan, 2005; Blair and LaMonte, 2006). They also claim that there are only a small number of conditions for which causal relationships

have been established between fat tissue and disease, suggesting that statistical associations between weight gain/obesity and chronic disease may be better explained by treating obesity as a symptom of disease rather than as a risk factor. The poor record of public health interventions in encouraging long-term weight loss is also raised as an issue based on the documented risks of several weight loss methods, including diet drugs, surgery, eating disorders and fad diets, as well as the evidence that improving aerobic activity and fitness improves health independently of effects on bodyweight (see also Lee et al, 1999; Farrell et al, 2002; Monaghan, 2005; Blair and LaMonte, 2006). From this evidence, it is argued that the obesity epidemic narrative distracts attention from the far more important and achievable task of promoting higher levels of physical activity.

How robust is this argument? Certainly, it does not seem to square with the US Department of Agriculture's estimates that the 'average American' consumes almost 25 per cent more energy compared to that consumed 30 years ago (Rigby, 2006). However, focusing on what the average person living in any country does and does not do is potentially misleading. Campos et al's (2006) argument that shifting the BMI distribution curve at a particular population level translates into small average changes in bodyweight among individuals within that population misses the point of studying distributions in the first place. Changes in average weight – or even average BMI – are not particularly useful indicators of public health due to the potential for those individuals and sub-populations whose weight has not changed to pull down the average, and thus to mask weight gains among other sub-populations that are rather higher than average (Kim and Popkin, 2006). What is of interest, then, is how many people fall within problem categories, where those people come from, what has predisposed them to excessive weight gain and so on. It is well established, as discussed above, that some ethnic, socio-economic and other sub-populations experience significantly higher rates of weight gain and obesity than do others. In the US, increases in bodyweight have also been greater among adults already in the overweight and obese categories than among those in the normal weight category, resulting in a large increase in the proportion of people classified as morbidly obese (Kim and Popkin, 2006).

Further to these issues of classification is the misleading suggestion that increasing bodyweight has few negative health consequences, and that the re-classification of BMI with weight gain is purely arbitrary. Kim and Popkin (2006) accept Campos et al's (2006) argument that the relationships between BMI, adiposity (fatness), nutrition, physical activity and chronic health are complex and not always well understood. They also agree that nutrition and physical activity may each impact on chronic disease independently of any interaction with weight gain or obesity (see also Blair and LaMonte, 2006). It does not follow from this, however, that overweight and obesity do not function either as intermediate conditions, or as direct causes of chronic disease. Rigby (2006), for example, reports that only small changes in weight are required to increase risks of chronic disease starting from a normal weight BMI of around 21. He also points out that, in addition to mortality, there are a

number of issues around quality of life and disability that warrant consideration in any assessment of the consequences of weight gain and obesity. Similarly, Hillier et al (2006; see also Blair and LaMonte, 2006) show that metabolic syndrome can be alleviated with modest weight loss. That consistent and long-term weight loss has proven difficult to achieve across so many public health interventions only suggests, according to Lawlor and Chaturvedi (2006), that extra importance should be attributed to prevention and to understanding key points of intervention during the life cycle.

Critics of obesity epidemiology highlight a number of important issues; namely, the complexity and uncertainty surrounding aspects of the relationship between BMI and health, the importance of targeting all risk factors for chronic disease and not simply the most visible, and the danger of assuming that an individual's bodyweight is the primary cause of chronic health conditions. Yet, this does not amount to a compelling case to dismiss weight gain and obesity as public health issues. Uncertainties and knowledge gaps may, in fact, contribute to an underestimation of the burden of disease arising from obesity (Canoy and Buchan, 2007). They may also be expected to contribute to what Dixon and Winter (2007) refer to as an environment of 'competing authorities', which exposes consumers to multiple conflicting messages – an environment that may have its own unmeasured impact on obesity epidemiology.

Causes of overweight and obesity

The prevailing view among health authorities is that weight gain and obesity result from a chronic imbalance between energy intake and energy expenditure (with intake exceeding expenditure) over an extended period of time (WHO, 2000). If we eat too much and/or exercise too little, we will get fat. However, the increasing prevalence of weight gain and obesity is not seen to result solely from overconsumption and inactivity, but from a range of environmental, social and behavioural factors that interact to determine energy intake and expenditure (WHO, 2000). Swinburn et al (2004) summarize those behavioural and environmental factors often put forward to explain weight gain and obesity, along with the strength of evidence currently available for each factor (see Table 9.2). They find 'convincing' evidence that factors such as sedentary lifestyles and a high intake of energy-dense foods increase the risk of weight gain/obesity. Heavy marketing of both energy-dense foods and fast-food outlets, as well as adverse social and economic conditions and the consumption of high-sugar drinks are identified to be 'probable' risk factors. Large portion sizes, frequent eating out, and yoyo dieting (rigid restraint followed by binge eating) are classified as 'possible' risk factors, while insufficient data are deemed available to determine the influence of alcohol on weight gain and obesity.

Physical activity and nutrition emerge from this analysis as the least controversial contributors to weight gain and obesity. Despite concerns that Australians are less active than in the past, physical activity patterns have remained relatively constant over the last 10 years. Moreover, approximately half of all adults are considered sufficiently active (AIHW, 2008) as to significantly reduce their risk

Table 9.2 *Evidence table for factors that might promote or protect against weight gain and obesity*

| Evidence* | Decreases risk | No relationship | Increases risk |
|--------------|--|-----------------------------|--|
| Convincing | Regular physical activity High dietary NSP (non-starch polysaccharides/fibre intake) | | Sedentary lifestyles High intake of energy-dense foods# |
| Probable | Home and school environments that support healthy food choices for children Breastfeeding | | Heavy marketing of energy-dense foods and fast-food outlets Adverse social and economic conditions (developed countries, especially for women) High-sugar drinks |
| Possible | Low Glycemic Index foods | Protein content of the diet | Large portion sizes High proportion of food prepared outside the home (western countries) 'Rigid restraint/periodic disinhibition' eating |
| Insufficient | Increased Eating frequency | | Alcohol |

Source: Swinburn et al (2004)

*Strength of evidence: the totality of the evidence was taken into account. The World Cancer Research Fund schema was taken as a starting point and was modified in the following manner: randomised controlled trials were given prominence as the highest ranking study design (RCTs not a major source of cancer evidence); associated evidence was also taken into account in relation to environmental determinants (direct trials were usually not available or possible).

#Energy-dense foods are high in fat and/or sugar; energy-dilute foods are high in non-starch polysaccharides (dietary fibre) and water, such as fruit, legumes, vegetables and whole grain cereals.

of cardiovascular disease, stroke, hypertension, Type 2 diabetes, osteoporosis, obesity, colon cancer, breast cancer, anxiety and depression (CDCP, 1996). There is a significant overlap, therefore, between those groups classified as overweight/obese and those classified as sufficiently active (independent of their BMI) to reduce the risk of chronic disease. Limited data are available on the total energy intake of Australians. However, the 2004–2005 National Health Survey found that the majority of Australians did not meet current nutrition guidelines – with 86 per cent consuming less than five servings of vegetables each day, 46 per cent consuming less than two servings of fruit each day, and with saturated fat accounting for 13 per cent of total energy intake (compared to the recommended level of <10 per cent) (NHMRC, 2003). Consuming more fruit and vegetables, and limiting the intake of high-fat foods reduces the total energy density of diets, thereby moderating weight gain and promoting weight maintenance (Savage et al, 2008). Australian studies have found that men, and people of lower socio-economic status, are more likely to have sub-optimal intakes of fruit and vegetables (AIHW, 2008).

As noted above, the importance of environmental and social factors in the prevalence of weight gain and obesity suggests that the task is not simply one of convincing people to eat a more nutritious diet – albeit, a more positive message

than convincing them to eat less – and to take more exercise. Rather, the task is one of addressing the various ways in which contemporary societies have come to constitute what is referred to as an obesogenic, or obesity promoting, environment. Dixon and Broom (2007) summarize the features of the obesogenic environment in terms of the commodification of food preparation and leisure; time pressures; changing parenting practices such as the increasing treatment of children as ‘consumers’; technology and sedentarization; car reliance; aggressive marketing; and competing/confusing messages about food and health. The interrelationships between these features are reflected in data concerning food consumption outside the home. In Australia, food purchased and consumed away from home now accounts for approximately one-quarter of total energy intake (Magarey et al, 2006). Consumption of foods purchased from fast-food outlets is becoming a regular behaviour for many people, with approximately one-quarter of Australians consuming fast food for dinner at least once per week (Scully et al, 2008) and the average Australian family spending 15 per cent of their food budget on fast food and takeaway foods (DAA, 2008). The consumption of foods prepared outside the home, in general, has a detrimental effect on energy and nutrient intakes (Burns et al, 2007) while the consumption of fast food is positively linked with weight gain and obesity (Rosenheck, 2008). Fast-food consumption is predicted by several factors: age (consumption decreases with increasing age), being a car driver, having children above the age of five years, not owning a home and, importantly, having higher household incomes (Mohr et al, 2007).

Biological theories posit that humans have an evolutionary preponderance towards weight gain (manifested in a preference for energy-dense foods, weak satiety and strong hunger traits), which makes them susceptible to obesogenic environments (Canoy and Buchan, 2007). We do not wish to debate this here. However, it is important not to substitute a simple behaviourist explanation for weight gain and obesity (for example, the lack of self-control) with an equally simple biological one. Dixon and Broom (2007) advance a social-ecological approach to the understanding of weight gain and obesity, one based on the acknowledgment that cumulative exposures to obesogenic environments promote changes in individual dietary and physical activity behaviours, with ensuing impacts on BMI and health. At the same time, such a model also recognizes that behavioural and biological processes can only be understood in the context of political, social and economic processes. Reduced physical activity, for example, can be at least partly explained by the fact that non-motorized transport and leisure are systematically discouraged by urban layouts, transport systems, retail and other geographies that make walking and cycling inconvenient, if not dangerous (Dodson et al, 2006). Limited access to practical and safe alternatives to car use disproportionately affects people living in outer suburbs characterized by low socio-economic status (Bostock, 2001). Conversely, these same communities are exposed to a higher-than-average concentration of fast-food outlets (Reidpath et al, 2002).

Sociological critiques: Public health crisis or moral panic?

The majority of sociological writing on weight gain and obesity falls into two camps. The first accepts the dominant epidemiological construction of weight gain and obesity as public health crises and seeks to contribute to the understanding of these crises through exploration of the environmental, social, political and economic dimensions of the obesogenic environment identified by Dixon and Broom (2007). The second camp, by contrast, challenges the framing of weight gain and obesity as a crisis or epidemic and instead reconceptualizes these conditions in terms of moral panic, as propagated by groups with an interest in the obesity epidemic narrative. In sociology, a moral panic is understood to be an exaggerated – and often irrational – outpouring of concern over perceived threats to social order. A moral panic most commonly develops during periods of rapid social and economic change. It is frequently directed at stigmatized minority groups and provides ideological support for attempts at social control. Use of this term is, therefore, deliberately provocative and emotive.

Reconceptualizing weight gain and obesity as a moral panic rests on two principal lines of argument. The first is that increases in the prevalence of weight gain and obesity do not fit traditional criteria for classification as an epidemic. They are not diseases that can be contracted or transmitted, and they are not growing at exponential rates. Within epidemiology, however, epidemics are not defined as the transmission of particularly virulent diseases but as the incidence or prevalence of illness or other health-related events outside the ‘normal’ range of expectations (Flegal, 2006). From this perspective, the classification of obesity as an epidemic is entirely appropriate (Flegal, 2006). A slightly more sophisticated take on this argument is offered by Boero (2007), who shifts the focus from adherence to technical definitions to the consequences of applying terminology to the particular issue of weight gain and obesity as it is constructed within public discourse. Boero (2007) points out that the rapid and seemingly indiscriminate spread of pathogenic epidemics like cholera and influenza played a major role in the rapid spread of fear and calls to vigilance in relation to these conditions. She argues, based on an analysis of US media reporting on obesity, that casting non-pathogenic phenomena in the same language helps to propagate fear, to privilege medical discourses and expertise, to open previously private domains of consumption and parenting to surveillance and intervention, and to legitimate the stigmatization of obese individuals. Further, these processes are gendered and racialized with women – particularly women from ethnic minorities – and their mothering practices singled out most frequently as targets of blame and reform (Boero, 2007). The sense of urgency engendered by the construction of obesity as an epidemic, for Boero (2007), feeds moral panic as well as the individualization and medicalization of what would better be understood as a social problem.

The second line of argument concerning the reconceptualization of weight gain and obesity as a moral panic draws on the challenges to obesity epidemiology posed by Campos et al (2006) and others. If weight gain and

obesity are not the objective public health threats that the obesity epidemic narrative would have us believe, then the obvious questions for sociologists focus on who is propagating this narrative, what do they stand to gain from it and why are we so vulnerable to it? These issues are addressed by Monaghan, for example, by asking:

if, after controlling for smoking and other variables, physically fit people have similar mortality risk independent of body composition ... why should clinicians tell a physically active person with a relatively high body fat per centage that this is unacceptable? Is it because body fat has become a highly visible, often enduring, deeply personalized corporeal marker for inferior social status in a way that smoking and hypertension are not? (Monaghan, 2005, p310).

Elaborating on relevant dimensions of social status, Monaghan also contends that:

the highly publicised war against fat is about moral judgements and panic (manufactured fear and loathing). It is about social inequality (class, gender, generational and racial bias), political expediency and organisational and economic interests. For many everyday people, including men and boys (but more often women), it is also about striving to be considered good or just plain acceptable in a body-oriented culture ... it is about occupational identity and relationships... All of this is independent of (potential) health problems commonly attributed to adiposity rather than highly consequential socio-economic factors (Monaghan, 2005, p309).

Large food companies, medical researchers, public health agencies, politicians and the media are all easy targets of a moral panic critique which takes the rejection of obesity epidemiology as its starting assumption. The economic and political interests of these groups – combined with ideological commitments and negative attitudes to minorities – are claimed to legitimate the demonization of obesity despite the alleged lack of scientific evidence (Monaghan, 2005; Campos et al, 2006). This explanation is neat. It has even inspired a significant political movement for obesity acceptance (Sobal, 1995). But it is far from convincing. In fact, the conclusion that proponents of the view that weight gain and obesity should be considered public health issues are universally driven by self-interest, ideological blindness and/or social prejudice beggars belief (Kim and Popkin, 2006).

Drawing on the same critique of obesity epidemiology, Guthman and DuPuis (2006) attempt to develop a more historically informed understanding of obesity as moral panic by theorizing the obese body as a site in which the material and discursive contradictions of contemporary capitalism, and neoliberal attempts to regulate it, are played out. One of the more pervasive

strategies of neoliberal governance, they note, is the devolution to individuals and communities of responsibility to solve the social and environmental problems generated by global capitalism (that is, by the individualization of social problems observed by Boero, 2007). In the case of obesity, however, citizens are not simply left to their own devices to deal with their weight while the food industry continues to promote and sell energy-dense and nutrient-poor foods. Personal responsibility, Guthman and DuPuis (2006) go on to argue, is construed simultaneously as the capacity to consume and as the potentially conflicting capacity to impose self-discipline. Overeating is both encouraged and vilified. This is probably true. However, even if capitalism and neoliberal attempts to govern it create contradictions and problems, to what and for whom are obese bodies and/or obesity epidemic narratives a solution, or even a partial solution? And how effective a solution could they be if the epidemic nature of overweight and obesity is not accepted in the first place? Guthman and DuPuis' critique is far more sophisticated than analyses that simply impute interests to anyone and everyone implicated in obesity and public health. Nonetheless, their critique generates two contradictions of its own: first, a contradiction between treating neoliberalism as a political rationality disconnected from the needs and goals of identifiable agents and institutions, and yet materialized in the practices and bodies of 'consumers'; and second, a contradiction between that same embodiment of obesity through overeating and a rejection of weight gain and obesity epidemiology.

We would suggest that all this still begs a question: what happens to conceptualizations of the obesity-epidemic-narrative-as-moral-panic if obesity epidemiology is not rejected? We would suggest that much of the underlying critique remains intact and is, in fact, strengthened. Stripped of the emotive language of moral panic, this critique has much to contribute in terms of understanding how several of the analyses presented – the construction of obesity as an epidemic within public discourses, the stigmatization of obese individuals and sub-populations, competing claims about the causes, consequences and potential solutions to increasing bodyweight, and so on – contribute to the competing/confusing messages about food and health that Dixon and Broom (2007) identify as components of the obesogenic environment. This point brings us full circle to those authors who accept that weight gain and obesity are serious public health issues, and suggest that the peculiar contribution of the sociological imagination to the resolution of these issues lies in understanding the obesogenic environment and how sub-populations and individuals interact with it (Dixon and Broom, 2007).

Although we will not offer a detailed review of the political economy literature in this chapter, we would suggest that research on the political economy of obesogenic environments is particularly advanced (even where this research has been undertaken without an explicit focus on obesity) and has a major contribution to make to population health and the identification of effective points of intervention. Fresh fruit and vegetables, for example, are what is known in the industry as a 'loss leader' – a product that retailers sell at minimal mark-up in order to encourage consumers into their stores.

Further, retailers are increasingly promoting themselves with signifiers of freshness, health and quality (Burch and Lawrence, 2005; and see Chapter 12 of this volume). However, processed foods are far more profitable than fresh foods. Winson (2004) consequently shows how major food retailers in Canada have increased the total shelf space, the number of sales locations and the promotional effort they devote to highly processed fatty, salty and/or sugary foods. Even fresh food sections are increasingly filled with a variety of pre-prepared food (from ready meals to pre-cut salad and vegetable mixes) that increase the value-added to retailers and reduce the affordability of fresh foods to consumers (Burch and Lawrence, 2005). Although retailers may not be able to control exactly what we buy – nor prevent us from purchasing from a rival retailer – the influence they exert through store layouts, allocation of shelf space and use of promotional materials, signage and so on, only needs to shift consumer decision-making at the margins to make a significant difference to health outcomes at a population level. As such, there is a very strong case for holding retailers accountable for their own claims to corporate responsibility. Similar arguments can be developed in relation to the planning of the built environment.

Although it is widely acknowledged that environmental changes more conducive to physical activity and healthy eating patterns are available to address weight gain and obesity at a population level (Hinde and Dixon, 2005), research into the ways individuals, families and other small groups interact with obesogenic environments is arguably less developed (although for a major contribution see Dixon and Broom, 2007). Such research, we would argue, is critical for understanding the non-genetic factors behind the vulnerability some people experience in relation to obesogenic environments, and the likely effectiveness of environmental interventions to address it. Small changes at a population level are extremely important. But so, too, are big changes at a local level, where multiple factors may combine to generate unexpected, unintended and undesirable outcomes. Bostock (2001), for example, has found that the reliance on walking as a mode of transport among single mothers without car access has compounded the social exclusion experienced by these women while also restricting their access to health services and food stores – thus obviating the potential health benefits of regular walking. Clearly, single mothers are particularly vulnerable to aspects of the built environment that render walking with small children dangerous and/or unpleasant. However, although renewing the built environment of lower-income neighbourhoods is clearly important in addressing the contradictory impacts of walking on single mothers, so too is the availability of transport options (such as public transport) that enable a wider range of mobility (Bostock, 2001).

Conclusion

Accepting the material reality of obesity and its consequences – even if it is accepted that our knowledge of weight gain and obesity is socially constructed and incomplete – raises the stakes. Stigmatization and discrimination are not

straightforward tools of social control enabled by 'obesity talk'. They are both causes and consequences of rising bodyweights – components of a negative feedback cycle with potentially deadly consequences for those caught in it. Understanding and breaking the cycle of discrimination and obesity does not require that sociologists defer to the expertise of epidemiologists and population health specialists. Rather, it requires multiple disciplinary perspectives and genuine debate within and between those perspectives.

The importance of what Dixon and Broom (2007) refer to as a social ecology of weight gain and obesity is also widely recognized by population health specialists. Among other recommendations, Swinburn et al (2004) call for more research into: the processes through which low socio-economic status promotes overweight and obesity; the effectiveness of environmental modifications or interventions; the impact of labelling on consumer choice, food formulation and dietary patterns; and the development of indicators suitable for monitoring environmental influences on obesity and weight gain. These recommendations should not define a sociological research agenda, although they do suggest useful points of engagement for sociology with other disciplines and with public health agencies. The peculiar contribution of sociology, we have argued, lies in challenging the individualization of weight gain and obesity as social problems by unpacking the interests and processes involved in producing and reproducing obesogenic environments; by exploring how individuals interpret and experience potentially obesogenic environments; and, following Guthman and DuPuis (2006), by analysing how the body is constituted as a site of social regulation. In a political environment that favours a consumer model of citizenship, together with market-based solutions to the majority of social problems, difficult questions must be raised regarding opportunities for meaningful environmental intervention of the sort favoured by population health specialists.

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