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Addressing the panic about 'obesity': Policy to protect health

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Abstract

Body size has come to be regarded as a major problem facing many countries in the 21st century. Whereas variations in height, hair colour and other bodily differences are simply accepted as normal human diversity; variations in body size are considered differently. Large body size is now firmly identified and labeled as overweight or obese and we are in the midst of what some authors have described as a moral panic about obesity (Gard and Wright 2005). The panic focuses on the seeming escalation of overweight and obesity and has especially brought the issue of lifestyle under ever increasing scrutiny. In this paper we first examine the taken for granted assumptions which underpin obesity discourse including the physiological aspects and consider the nature and impact of the moral panic about obesity on individuals living in larger bodies. We address the potential or emergent challenges to accepted wisdom about obesity and then consider how health policy in any country, given the current state of obesity science, can support the greatest number of people to enjoy better health.

Introduction

The scenarios for moral panics change but in the history of such panics, areas of commonality can be seen and are certainly characteristic of panic about obesity (Rosenberg 1992). In many moral panic scenarios there is, at some level, a real problem. Experts have fundamental disagreements about the nature, cause, consequences and appropriate responses to the problem and these disagreements are researched and argued in academic fora. At the same time the media "feasts" on the topic; on one level as a voice for the debate but also as a mechanism for creating a feedback loop in which widely perpetrated myths become accepted wisdom and the public (and many health professionals) are unlikely to attempt to access objective information. In most western nations the media frenzy about body size has reached historic levels with television especially, featuring more and more 'reality' programs which purport to address the crisis of obesity and lifestyle.

Over the last thirty or so years health has changed from being a passive to an active status. Achieving and maintaining good health has evolved from being a matter of good luck to being the product of good management. Rather than seeing an individual as the victim of an unhealthy environment it is now more likely that we will consider that individual's failure to take adequate precautions in terms of a wide range of possible behaviours including smoking, eating,

sleeping, and being sedentary. Hansen and Easthope (2007, 11) note that avoiding disease through personal effort has become the predominant discourse when speaking and writing about health. Interest in personal responsibility has been further fueled by the changing demographic in which there are now increased numbers of older people with greater propensity for costly degenerative diseases.

For many years the greatest myth perpetuated in some academic and all populist fora has been that body size, unlike any other human variation, is a matter of choice. Underpinning the notion that body size can be chosen is the seemingly self evident statement that maintaining a "normal" weight is a simple matter of balancing energy ingested with energy expended. Once this is accepted the scene is set for a scenario of blame and condemnation of larger individuals who are then presumed to eat excessively and to be inactive. Because a number of chronic diseases are considered to be closely associated with large body size, it has become possible to blame large people, not only for their personal failings, but also for the considerable challenges currently confronting health systems. In addition such a mind-set has, until comparatively recently, limited or constrained the nature of questions posed in the field of obesity research and the lack of primacy given to particular research evidence.

For many years a steady stream of research evidence has contradicted our taken for granted assumptions. There is extensive research to show that body size and fat distribution is genetically determined, that reduction dieting is not only futile but also makes a significant contribution to increasing body weight and to distorted attitudes towards food and body image. There is also research evidence that the relationship between body size and health is poorly understood, frequently portrayed inaccurately and not yet fully understood (Cogan and Ernsberger 1999; Ernsberger and Koletsky 1999; Campos et al. 2006; Troiano et al. 1996; Gard and Wright 2005). No RCTs or other source of reliable evidence exist which conclusively prove the relationship between energy in and energy out (Grundy et al. 1999; Gard and Wright 2005)

In summary, neither the cause nor cure for obesity has ever been agreed. The connection between large body size and poor health is not at all clear-cut and has not been adequately separated from the characteristics of an individual's lifestyle or socio-economic status in research

scenarios. Yet such evidential challenges have failed to dent the predominant world-view that fat people have only themselves to blame thus creating a cultural milieu in which living as a fat person is a painful experience.

The experience of being overweight

Public discourse consistently affirms the notion that fat people are undisciplined eaters, inactive and as a consequence creating a significant drain on public health services. Such beliefs support social marginalization of fat people, underpin unhelpful clinical encounters and preclude more thoughtful examination of the issue. In addition the focus on personal responsibility for fatness reduces the focus on the recognized contribution of poverty and ethnicity to access to good health and good health care (Marmot 2005, 2006).

Health professionals generally fail to utilize evidence when attempting to address issues of overweight and obesity. It has been argued in the literature that "moral concerns shape debates over scientific evidence because fatness has been considered both evidence of medical pathology and moral turpitude since the turn of the 20th century" (Stearns, 1997 cited in Saguy & Riley, 2005, p. 871). This has allowed a strange combination of judgment, blame and righteous indignation as a basis for clinical encounters, which are entirely counterproductive to any useful outcome. In such a context any clinical encounter about body size is bound to be fraught.

Obesity may or may not be a health risk depending on the individual. Because we have framed fatness as a preventable health risk, regardless of the accuracy or otherwise of that belief, this has allowed or indeed supported less tolerance and a range of behaviours in health professionals, which would not be seen in other clinical encounters (Brownell, Puhl and Schwartz 2005; Schwartz et al. 2003). People who are overweight or obese may be treated with less clinical vigilance and less respect based on assumptions about their lack of care for themselves. In addition there are reports that they may be less likely to seek help or preventative screening based on fear of judgment and exposure (Paskiewicz et al. 2002).

Previous research (Carryer 2001) revealed the extent to which large woman internalized the social repugnance towards their own large bodies. Many overweight people, especially women,

spend their lives engaged in a literal battle against their non-compliant bodies. Failing to win this battle in an environment that tells them it is simply a matter of disciplined adherence to a healthy lifestyle creates a sense of failure and shame. Not only do large people feel culpable for their size they are confronted daily by their presumed increased risk for a number of life threatening diseases which only compounds their distress.

For reasons not yet perfectly understood, people of lower socio economic status and indigenous populations have higher levels of obesity. Social ostracism, self-loathing and stress are not conducive to health. Poverty and minority ethnicity also make known contributions to poor health status thus ensuring that those with the least likely access to health and health care are doubly likely to experience the stigmatization of obesity.

The notion that eating habits and exercise levels may not be the only determinants of body size has not been seriously considered until very recently when pockets of scientific endeavour in different parts of the world are beginning to report challenges to predominant views. Such findings are beginning to make more sense of genetic variation along with metabolic and even bacterial precursors to differential utilization of ingested calories.

The biological aspects of obesity and energy balance

Over 50 years ago Kennedy (1953) formulated the 'set point' hypothesis which was that for any individual there was a preset level of fat storage in the body regulated by a 'lipostat'. According to this 'set point' hypothesis a change or disturbance to an individual's energy balance that caused a change in the total amount of fat stored would result in a compensatory change in the food intake and energy expenditure efficiency to try to maintain the original or 'preset' level of fat storage. An implication of this hypothesis was that the body would defend its fat stores as a physiological response to attempts at weight reduction for example by calorie restriction. Though initially this hypothesis was received with a certain amount of skepticism, a large body of basic physiological research over recent years alongside the development of new experimental techniques has validated the concept that there are physiological mechanism that regulate energy homeostasis including energy stores (Penicaud et al. 2000; Beck 2000; Woods and Seeley 2000; Speakman 2004; Levin 2005, 2006; Blundell 2006; Ferguson 2006; Richard and Boisvert 2006;

Schwartz 2006). An important outcome to this increasing body of core physiological research is that the hypothesis has been modified to incorporate the concept that like a thermostat this "set point" or "lipostat" resets in response to environmental and physiological demands, particularly during early development (Prentice 2005; Gluckman et al. 2007; Taylor and Poston 2007). This hypothesis not only provides a physiological rationale for the fact that for many individuals long term weight loss is difficult to achieve but is also logical in a physiological context. All living creatures depend on energy for basic cellular functions and to meet the needs for variable increased demands such as growth, reproduction and different levels of physical activity. Hence the importance of ensuring adequate energy stores for survival. This can be seen in a variety of creatures, such as migratory birds, hibernating animals and especially warm blooded mammals where there is the extra energy demand to maintain body temperature, as well as meeting the needs for normal body function, reproduction and activity.

The human being is no exception. This is apparent in the rapid increase in fat deposition that occurs particularly in three critical periods during the human life span- prior to and during pregnancy and in the baby, prior to birth and during the first 6 months of life and prior to weaning (Gesta, Yu-Hua Tseng and Kahn 2007). It is also evident in how fluctuations in appetite follow the human growth curve for example with the increased appetite to meet the energy demands for growth during adolescence. In view of this it is not surprising that complex, sophisticated methods exist that regulate energy balance in order to ensure adequate energy stores and that these can respond to changing physiological demands and the environment. The existence of these mechanisms is also supported by the remarkable constancy of human weight, often over many years (and without calorie counting!) and the resistance to weight change that so often frustrates the attempts for those trying to lose weight.

That genetic factors play an important part human in obesity comes from earlier twin and population based studies. These observations have been strengthened over recent years with advent of research into the human genome. A significant number of large scale genetic epidemiology studies have tended to confirm the role of genetic factors not just in animal models (Levin 2005) but also in human obesity (Loos and Bouchard 2003; Clement 2005; Rankinen et al. 2005; Saunders et al. 2007). An estimated 30-80% of weight variation may be determined by

genetic factors and an increasing number of genetic linkages to obesity are being identified. The concept that genes that predispose to fat storage may have provided survival advantage in times of food scarcity was the basis of the 'thrifty genotype' hypothesis (Neel 1962) put forward to account for the observed differences in susceptibility to obesity between different ethnic groups such as the Pima Indians (Loos and Bouchard 2003) and in New Zealand those of Polynesian descent (Goulding et al. 2007). This core concept has current relevance but needs to be modified to accommodate the polygenic nature of obesity and that it arises through the joint interactions of multiple genetic and environmental factors (Loos and Bouchard, 2003). Cultural and socio-economic factors, health literacy and life style choices are important factors that interact with this increased susceptibility and modify final outcome.

Alongside the genetic studies there has also been an increasing understanding of the physiological mechanisms at the molecular level that influence energy intake such as appetite, satiety and food preferences that may more obviously link with obesity but also energy storage, metabolism and expenditure and how these mechanisms may be influenced by genes (Woods 2000; Berthoud 2006; Schwartz 2006; Levin 2006; Blundell 2006). Obesity results from chronic disruption of these mechanisms, or when they are overwhelmed by the demands of our current obesogenic environment and life styles, as exemplified by rising statistics for obesity world-wide. The initial breakthrough in our understanding of the physiological mechanisms that regulate energy homeostasis in the body came in the 1970s with the classic experiments of Coleman (1973) on genetically obese mice. Coleman showed that the food intake and body weight of genetically obese mice (*ob/ob*) decreased when their circulation was linked to lean control animals. He inferred from this that there was a blood-bourn signaling molecule which reduced food intake and body weight and that this was lacking in these genetically obese mice. This missing factor was provided via their linked circulations by the lean mice, resulting in weight loss in the obese mice. Similar experiments with another type of genetically obese mice (*db/db*) demonstrated that these mice could produce this circulating adiposity regulator but that this was ineffective because of the absence of the required receptors, resulting in obesity. With the development of the new gene technologies leptin was identified as this adiposity regulator (Zhang 1994) and has been the subject much research since, particularly in animal models. This confirmed leptin's role as a key controller of body fat stores, operating as a negative feedback

signal released by adipose cells that acts on specific receptors in the hypothalamus in the brain to reduce energy intake and increase energy output (Woods 2000). As well leptin was shown to interact with other body systems such as sexual maturation and the stress response (Harris 2000). The identification of cases of human obesity linked with a genetic deficiency of leptin (Montague 1997) or its receptor (Clement 1998) confirmed that similar energy homeostasis mechanisms also operate in humans. However these are rare as causes for human obesity, and initial hopes that leptin could be administered to treat obesity in a similar manner to the use of insulin in the treatment for type 1 diabetes mellitus, were not fulfilled. Paradoxically human obesity is commonly linked not with low levels but with elevated levels of leptin in blood and an apparent resistance to leptin's action (Munzberg et al. 2005; Enriori et al. 2006).

Active ongoing research over recent years is demonstrating that energy homeostasis is a much more complex, distributed, integrated circuitry involving the hypothalamus as well as other parts of the brain, the brain stem, peripheral tissues particularly adipose tissue and the digestive system that interact with a large number of different interacting signaling molecules including metabolites (Berthoud 2006; Schwartz 2006; Levin 2006; Blundell 2006; Moran 2006). Until very recent times the reality for most creatures, including humans, has been to cope with intermittent periods of food shortages rather than an environment of over abundance. Therefore this complexity and the existence of a number of 'back-up" systems that ensure adequate energy stores is not surprising. It follows that evolutionary environmental pressures may have acted to select those whose energy homeostasis mechanisms favored fat storage. In the past this has helped ensure survival but has become disadvantageous in our current obesogenic environment.

The tendency to store (or not store) fat is determined by enzyme catalyzed fluxes through different metabolic pathways involved in fat deposition and breakdown. At a molecular level fluxes through these different metabolic pathways involve neuroendocrine regulation involving the brain, particularly the hypothalamus, the autonomic nervous system, various hormones and other signaling molecules such as 'transcription factors' and the action and amounts of key enzymes and other functional proteins (Palou et al. 2000).

This is clearly evident for example from the increased fat storage and hormonally determined difference in fat depots that occurs in women compared to men. Increased (or decreased) flow through a metabolic pathway may occur directly and generally rapidly by activation or inactivation of key enzymes via different physiological triggers such as hormones. Over a longer time frame it may also occur by increased or decreased production of specific enzymes or other functionally important proteins as a result of the presence of specific genes that code for them and their expression (Thompson et al. 2007). This depends on the genetic endowment, but also the various transcription factors that control their 'turning on' and 'turning off'- the area of epigenetics- a very active area of current research. That gene expression is selective and controlled is evident by the fact that though all our cells contain the same genes encoded in their DNA, inherited from our parents, the gene coding for hemoglobin will only be expressed in cells destined to become red blood cells as part of cell differentiation and specialization but in other cells in the body this gene is 'silenced'. It is only over the last decade that there is increasing evidence of the importance of similar permanent changes in gene expression occurring early in development as a result of intra-uterine exposures which may have significant impacts on various aspects of metabolism including energy metabolism and storage and the significance of epigenetics, early development and the developmental origins of chronic disease including obesity and its associated health risks of type 2 diabetes and cardiovascular disease (Waterland and Michels 2007).

The importance of environmental factors during the early stage of development came from the groundbreaking epidemiological studies by Barker and Osmond (1986) which showed that low birth weight in full term infants was linked with an increased risk for obesity and cardiovascular disease in adult life. This was followed up by much subsequent research in animal models and human populations which showed that a compromised intra-uterine and perinatal environment was linked with an increased risk for obesity as well as type 2 diabetes mellitus, cardiovascular disease in adult life (Gluckman and Hanson 2004; McMillen and Robinson 2005; Cottrell 2007). These observations provided a basis for the 'thrifty phenotype' hypothesis and the developmental origins of adults disease concept, where the developing fetus makes long term permanent adaptations in response to early environmental factors such as in utero nutrition which, in the long term, may be disadvantageous and involve epigenetic changes which can be carried through

to the next generation (Gluckman and Hanson 2004; Prentice, Rayco-Solon and Moore 2005). Permanent long term changes in gene expression have also been shown to occur in babies who are exposed to high levels of glucose in utero as a result of gestational diabetes in their mother. Here also there is an increased risk for the later development of obesity as a result of a 'resetting' of the homeostasis mechanism to maintain a higher level of body fat (Vickers et al. 2000; McMillen, Adam and Muhlhausler 2005).

Excluding places in the world where there is a gross shortage of food, whatever community, country or time period we choose, we find a mix of obese and lean people even though they may share the same obesogenic environment. Though psycho-social, economic and environmental factors play important roles and these have been well documented, the different strands of basic physiological research outlined above help to provide insight that obesity is a complex interaction of different factors including genetics and early development as well as the environment. Importantly, the acknowledgement that some are more at risk than others because of a chronic disruption or resetting of the mechanisms that regulate energy balance, a particular susceptible time being during early development. It is imperative that these factors are acknowledged when addressing the issue of obesity and its related health risks.

Also relevant are the commonly criteria used to classify some-one as obese and their limitations. The body mass index (BMI, weight in kg/height in m^2) was based on U.S. life insurance data in the 1930s which found that mortality was lowest for individuals with BMIs between 20 and 25 and increased dramatically when the BMI was over 30 or less than 20. It remains the main criteria used in the research literature relating to obesity today. It is important to emphasize that health risks relate to fat mass and that BMI does not distinguish between fat and muscle mass. This is an important consideration since BMI is generally used to make predictions about health risks such as for cardiovascular disease and type 2 diabetes mellitus. Re-analysis of two large epidemiological studies where body fat was also determined and not just BMI found that mortality *increased* by 30% for every standard deviation of weight loss but *decreased* by 15% for every standard deviation of *fat loss* (Allison et al. 1999). As well as total amount of fat it is the distribution in the body that is important. It is visceral obesity 'the apple shape' pattern of fat distribution which is closely linked with insulin resistance, type 2 diabetes, hypertension, hyperlipidemia and cardiovascular disease that carries the greater mortality risk (Shaw, Hall and

Williams 2005). The metabolic syndrome, syndrome X or insulin resistance syndrome are terms used by some for this cluster of clinical parameters which has been shown to be an interaction between genetic, developmental and environmental factors (McMillen and Robinson 2005; Cottrell 2007). These metabolically linked health risks are low with subcutaneous fat accumulation around hips, buttocks and breast- a 'pear shaped' pattern of distribution typically seen in pre-menopausal women. Therefore waist/hip ratio needs to be considered and not just BMI when assessing health risks (Clement 2005; Levin 2005; Gesta 2007).

Overall, different public health interventions have been in the form of simple dietary guidelines and 'anti-obesity' messages but frustratingly, with limited success (Parsons et al. 2005; Penny 2005). The vital challenge therefore is to explore some of the reasons for this limited success and develop more effective strategies for dealing with the increasing obesity problem by putting it in the wider context of the health promotion. After cardiovascular disease and type 2 DM, cancer is the next major cause of morbidity and mortality in many societies. An obesogenic diet is also likely to be low in vegetables, fruit and whole-grains - well established protective factors as regards cancer risks (The American Institute for Cancer Research 1999; Johnson 2007). It is important to recognize that obesity is a complex multi-faceted problem and that there is a clear distinction between health and BMI.

A healthy policy response

Governments in Western countries especially are concerned to address the epidemic of obesity at the highest level of policy formation. At the end of the first decade of the 21st century, the increasing burden of chronic disease is demanding significant attention throughout the world (Lopex et al. 2007; Stanhope and Lancaster 2008). Because of the global issue of increasing chronic illness and contracting health workforces (NZIER 2004) there is pressure to gain control of increasing levels of diabetes, cancer and other long-term conditions. Because obesity is seen as causal rather than another consequence of altered dietary composition, the focus of policy formation draws on a public and medical discourse framed as combating obesity.

Wray and Deery (2008, 240) note the significant physiological and psychological harm engendered by current media preoccupation about body weight. The media is an imperfect

apparatus for the deployment of evidence and in the current climate of hysteria about obesity much misinformation is fed to the public on a daily basis. Wray and Deery (2008) note that it is difficult for anyone to resist and question the selective versions of medical science which prevail, but the end result is a climate in which any real progress on improving population health is unlikely. Health professionals are not immune from misinformation and the stigmatization created supports the potential for mistreatment and inappropriate health care.

The problem is also bigger than the direct consequences for larger bodied individuals. Our current preoccupation with obesity obscures other important aspects of ensuring that people lead healthy and fulfilled lives. It is very difficult to sustain a creative and constructive response to all people's health needs in the midst of the anxiety about epidemic fatness. Campos (2004) has made the observation that the only way to win the war against obesity is to stop fighting the battle. He argues that the current preoccupation with individual blame and responsibility risks focusing on the unproven behaviour of individuals in a manner which offers no constructive outcome for those individuals. More importantly such an approach also occurs at the expense of developing the potential for community embedded processes which increase health for all through a broader focus on the benefits of exercise and good nutrition and a sense of personal well-being and acceptance for all people without attention to individual body size.

Given the strong evidence for the benefits of exercise (Blair, Cheng and Holder 2001; Blair and Church 2004) and for the benefits of good nutrition regardless of body size it seems possible that countries may make the greatest health gains in a broader focus aimed at optimizing health for all rather than focusing on largely futile weight loss agendas. Preoccupation with people who are overweight or obese allows those who are sedentary and have poor eating habits to remain less aware of their own increased health risks.

There are other important determinants of health including the quality of the diet such as the intake of vegetable, fruit and wholegrain products, type of fat and physical activity, smoking, stress and mental health. There is a need for an approach that affirms and empowers those who through a combination of their intrinsic biology and circumstances are classified as obese. Positive goals are needed and such goals need to be realistically attainable as part of effective,

appropriate and culturally sensitive strategies for the promotion of health literacy in conjunction with addressing the social, economic and physical factors. Healthy Eating Healthy Action (New Zealand Ministry of Health 2004) involving and accessible to all sectors of the community particularly those who are most vulnerable is an alternative option than that typified by media programs such as 'the biggest loser' and alienating and stigmatizing those who do not fall into the 20-25 BMI category.

If the response to increasing body size is to be health promoting in its fullest sense, countries could consider developing health policy which accepts biological diversity and that there is no neat template for body size normality. Constructive health policy would address the degree to which all people regardless of social class status, poverty or ethnicity, have good access to healthful nutrition and safe and pleasurable exercise and can live their lives without stigmatization and the fear of impending illness and death as engendered by current obesity discourse.

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