Obesity - A Public Health Crisis

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Introduction

Obesity is a chronic disorder with multiple causes which may affect an individual in isolation, or act collectively at a population level. Virtually all obese people develop symptoms of chronic disease by the age of 40, and the majority will require medical intervention for obesity related disease before they are 60 (Lean 1998). Obesity may well represent the most important threat to the cardiovascular health of technologically advanced countries in the 21st century, and like many risk factors for disease, obesity results from behaviour and shows a social gradient; especially among women. Obesity is more common among lower-income individuals, those with less education and some ethnic/racial minorities (Rhoades, Altman & Cornelius 2004).

The associated contributors are psychological, physiological, neurological, endocrinological, behavioural, environmental, nutritional, genetic, social, and occasionally pathological or drug related. The interplay between these contributors is extremely complex and is further complicated by the inter-individuality of the condition. Professor Arya M Sharma of the University of Alberta summed it up nicely in his editorial in Obesity Reviews July 2009 when he said: “Telling someone with obesity to simply eat less is about as effective as asking someone with depression to simply cheer up”! (Sharma 2009).

The common view is that obesity in the UK (as elsewhere) is driven primarily by the increased consumption of energy-dense nutrient-poor foods, combined with a marked reduction in physical activity across all age ranges. However, whilst actual evidence to confirm the contribution of energy in or energy out as the major driver of obesity remains equivocal, data on dietary intake in the US population show a clear trend toward increased dietary energy intake. Dietary surveys and food disappearance data are consistent in indicating an increase in calorie intake of about 200 kcal/day/person over the past 20 years (Popkin, Nielsen & Siega-Riz 2002).

Over the last few decades, our way of life has changed dramatically. There are many more labour-saving devices; we walk less; eat out more; have regular takeaway and shopping deliveries. Food is ubiquitous and more palatable than ever before. Sophisticated marketing and pricing strategies encourage us to ‘go large’ or to ‘buy one, get one free’. Towns have been planned around the car and large supermarket chains have replaced local fishmongers, butchers and greengrocers. New houses are now designed without a communal dining area; a concept that would have seemed incredulous fifty years ago. The Foresight Report (King 2007) argues that it is the interplay of such factors that creates what has been termed in their report as an ‘obesogenic environment’. 
It is also known that the response to an obesogenic environment differs for each of us, with one eminent commentator stating:

“Somehow the (appetite) forces are orchestrated to sustain a beautiful symphony in normal weight people, and a chaotic dissonance in people with the disease of obesity” (Frank 1993).

The traditional models of weight control using calorie restriction and exercise seem to have limited effectiveness in the long term (Wilding 2007) and it is becoming evident that a combined and cohesive long term strategy involving the ‘individual’ and the ‘environment’ is now needed.

**An Evolutionary Perspective**

Throughout our evolution the omnipresent threat of famine to the human race has resulted in an unimaginable toll of deaths. Famine is brought on by large scale food insufficiency resulting normally from drought or floods leading to widespread failure of plant growth. In the absence of vegetation all animal life will eventually perish as body weight decreases to a point where life is unsustainable and starvation becomes inevitable. The frequency of such climatic disturbances and subsequent regular food shortages, has led to the development of highly evolved human mechanisms for defending against starvation.

These famine resistant mechanisms are complex and primal, which is why there has been so little progress in pharmacological interventions for obesity. If body fat is lost too quickly, this signals famine and primeval safety mechanisms come into play, slowing metabolism, conserving fat stores and reducing lean mass. Voracious hunger ensues; acquiring adequate nourishment completely occupies the mind. However in the developed world with no real threat of famine and an abundant supply of food, weight for most people travels in an upward only direction, the results of which can also be fatal.

The thrifty genotype hypothesis (Neel 1999; Chakravarthy & Booth 2004; Prentice 2005) suggests that in any population some people will be genetically more energy efficient, capable of storing further body fat in a given environment than others. Whist this may not make for an ideal hunter gatherer, it would surely be an advantage come the inevitable cyclical famine. It is these ‘thrifty genotypes’ that are now considered genetically susceptible to obesity in our plentiful urban societies.
Additional to the influence of the genotype it is now known that the phenotype can also be influenced by early life exposure to nutritional changes. For instance under-nutrition during the first trimester in the womb will predispose to a thrifty phenotype (termed nutritional thrift) where physiological changes to the embryo will lead to the individual gathering more body fat, having lower levels of lean mass and being more susceptible to metabolic disturbances and ultimately cardiovascular disease in adult life. Undernutrition in mid gestation is associated with obstructive airways disease and microalbuminuria, whilst nutritional insult during late gestation decreased glucose tolerance (Painter, Roseboom & Bleker 2005).

Epigenetics, a fascinating and novel branch of science is casting new light on how behaviour impacts genes, which can then be passed through generations, via what is being termed the ‘memory of the genes’. Genetic expression is altered, but the sequence of DNA remains unchanged. The significance being that behaviours and environmental exposures during life are ‘imprinted’ onto the genes and passed down to the next generation, altering genetic inheritance, which was hitherto considered sacrosanct (Waterland & Jirtle 2004).

Epigenetics has led to the emergence of nutrigenomics (Elliott & Johnson 2007) which suggests that the identification of genetic variations will allow early life nutritional interventions, or even corrective therapies aimed at preventing chronic disease. Both epigenetics and nutrigenomics whilst in their infancy offer profound insights into the recent and dramatic increase in obesity. Each may well offer novel approaches for future treatments. However, for now more needs to be learned about the interplay between behaviour, nutrition and the way our genes express themselves.
Prevalence of Obesity

In recent years Britain has become a nation where overweight is the norm with one of the fastest growing rates of obesity in the developed world. The Foresight Report ‘Tackling Obesities: Future Choices’ states that the prevalence of obesity has more than doubled in the last 25 years in the UK and the total number of people who are overweight or obese now exceeds 24 million. The Foresight document “Future Choices” written in 2007 projects that about 28 per cent of women and 33 per cent of men in the UK will be obese by 2010. On top of this a further 42% of men and 32% of women are already classified as overweight (Sproston & Primatesa 2004). Therefore the best estimates for overweight and obesity combined in the UK currently are Men 75% and women 60%.

Foresight’s extrapolations go on to suggest that we can anticipate some 40% of Britons being obese by 2025 and that this will grow to 60% of adult men, 50% of adult women and about 25% of all children under 16 being obese by 2050.

These projections from Foresight however have been questioned and indeed the validity of the Foresight Report itself has been challenged. In his address to students at University of Chester this year, Dr David Haslam of National Obesity Forum (NOF) offered his opinion that the report was flawed and subsequently unreliable. Further to this Professor Mike Thomas (University of Chester) in his address to students also in September 2009 stated that he did not expect the obesity epidemic to materialise in the UK.

Figure 1. Prevalence of obesity by age and sex in the UK. Source: Health Survey England 2007
Prevalence Worldwide

According to the World Health Organisation currently more than 1 billion adults are overweight with at least 300 million of these obese. In 2000 the number of overfed people on the planet surpassed the numbers of underfed (Gardner & Halweil 2000). Whilst this is progress, the grotesque spectacle of wide scale obesity living cheek by jowl with chronic malnutrition is now a reality in many developing countries.

Current obesity levels range from below 5% in rural China (Li, Fan & Cai 2007), Japan (Yoshiike, Seino, Tajima, Arai, Kawano, Furuhata et al. 2002) and certain African nations (Abubakari, Lauder, Agyemang, Jones, Kirk & Bhopal 2008), to over 75% in urban Samoa (Keighley, McGarvey, Quested, McCuddin, Viali & Maiava 2007). But even in relatively low prevalence countries like China, rates are almost 20% in cities; the greatest threat from obesity to traditional societies is urbanisation.

Figure 2. Trends in adult prevalence of obesity in various countries (Foresight, 2007)

The rising numbers affected by obesity reflects profound changes in societies over recent decades. Economic growth, modernization, urbanization, nutrient modification and globalization of food markets are just some of the forces driving a worldwide energy balance transition underpinning the obesity epidemic. The emergence of obesity in developing countries initially affected the higher socioeconomic strata of the population. But more recent trends show a shift
in prevalence from the higher to the lower socioeconomic level. This change increasingly results in the existence of households with an undernourished child and an overweight adult, a situation called the “dual burden” of disease (Doak, Adair & Bentley 2005; Caballero 2007).

**Childhood obesity**

Childhood obesity is already at epidemic levels in some inner city areas in the UK. According to Camden PCT, based on a child population of 14,300, it is estimated that 58% are overweight and obese. The 2008 NCMP data analysis found that even the very young were affected with 17 primary schools showing 40% of children above the 91st centile and 5 schools with >50% at year 6. (Camden Primary Care Trust 2009).

The problem is global and increasingly extends into the developing world; for example, in Thailand, the prevalence of obesity in 5-12 year old children rose from 12.2% to 15.6% in just two years (Aekplakorn & Mo-suwan 2009). Even more alarming was the rise in obesity in one group of New Zealand adolescents which showed an increase from 19.4% to 30.7% in just eight years (1997 – 2005) representing a year on year annual increase of 7% (Utter, Scragg, Denny & Schaal 2009).

Much evidence is now accumulating to suggest that lifetime weight trajectories are set at the very early stages of life (Buchan, Heller, Clayton, Bundred & Cole 2005; Greenhalgh 2005; Langley-Evans, Bellinger & McMullen 2005; Moore & Davies 2005). It is becoming more widely acknowledged that adult strategies alone to combat population obesity will ultimately prove ineffective.
Health consequences of obesity

Obesity is a chronic condition and a serious medical problem nationwide. It is estimated that in the UK alone 9,000 premature deaths each year are as a direct result of levels of obesity. This equates to 6% of all deaths, compared to 10% for smoking (National Audit Office 2001). Since 2001 smoking rates have fallen whilst obesity rates have grown significantly. Years lost to obesity averages 9 compared to 10 years for smoking (Doll, Oeto, Boreham & Sutherland 2004). Obese people are also more likely to become or remain disabled (Walter, Sunst, Mackenbach, Hofman & Tiemeier 2009).

Obesity is associated with increased mortality rates in people of all ages, and mortality varies more or less in proportion to the level of obesity (Lew & Garfinkel 1979; Solomon & Manson 1997). The risk for someone with a BMI of 30 is about 50% higher than a healthy BMI (BMI 18.5 to 25), and for a BMI of 35 the risk is more than doubled (Manson, Bassuk, Hu, Stampfer, Colditz & Willett 2007). Whilst this relationship has historically been viewed as strongest until the age of about 50, the effect of being overweight on mortality persists into the ninth decade of life (WHO, 1998). There is also a link between duration of overweight and mortality risk - those who have been overweight for the longest are at highest risk. In-fact those who were obese as children, but who are now normal weight are at an increased risk of morbidity and mortality (Telama, Yang, Viikari, Valimaki, Wanne & Raitakari 2005).

Excess adipose tissue associated with overweight has major detrimental implications on the body. It increases the work of the heart and leads to anatomical changes within it. It also alters pulmonary, endocrine and immunological functions with subsequent adverse effects on health. Such alterations result in serious health complications including: cardiovascular disease; non-insulin-dependent diabetes mellitus; obstructive pulmonary disease; hypertension and joint disease (Must, Spadano, Coakley, Field, Colditz & Dietz 1999; Walter et al. 2009) Cancer is also now strongly linked to obesity and related lifestyle choices (Barnard 2004).
Obesity and Cancer
Not only are obese people more susceptible to cancer, but their prognosis is significantly worse once diagnosed. Men that are obese are 33% more likely to die from cancer, and obese women have a 50% higher chance of dying from breast cancer. Additional to obesity, cancer is now being strongly linked to nutrition and physical activity status (Bray 2004; Barnard 2004; Wiseman 2008). The cancers most strongly associated with obesity are liver, colon and pancreatic cancer in men and breast, uterine, endometrial, kidney and cervical cancer in women (Calle, Rodriguez, Walker-Thurmond & Thun 2003).

Cardiovascular Disorders Associated With Obesity
Cardiovascular disease (CVD) is the main cause of death in the UK accounting for just under 233,000 deaths a year; four out of ten of all deaths (British Heart Foundation 2003). Obese people are far more likely to die from CVD driven primarily by hypertension, loss of glycaemic control and hyperlipidaemia, combining to vastly accelerate atherosclerosis.

Obesity and mental health
Obese people appear to score less favourably on all of the psychological assessment scales, with symptoms ranging from sub clinical unhappiness to severe depression. Evident are more episodes of mood, anxiety, eating and personality disorders, often associated with or related to the obesity experienced by the individual (Pickering, Grant, Chou & Compton 2007). Obesity predicts the onset of psychiatric symptoms and vice versa and is associated with psychosocial impairment and weight-based stigmatization. This includes self-deprecation and low self-esteem, with the stigmatization as ‘voracious persons’ leading to isolation and loneliness. As such, many chronically obese people appear paradoxically to seek comfort in excessive or binge eating. So, the circle is complete - concern, shame and guilt related to low self-esteem, which is finally related to excessive overeating. (Roberts, Deleger, Strawbridge & Kaplan 2003; Herva, Laitinen, Miettunen, Veijola, Karvonen & Laksy 2006; Kasen, Cohen, Chen & Must 2008).

Obese people are discriminated against. They generally report poorer quality of life and functional health and well-being, collectively known as health-related quality of life (HRQOL)
(Puhl & Brownell 2001; Wadden & Phelan 2002). These relationships appearing especially strong for women (Fontaine 2001) and for individuals with more severe obesity (Hudson, Hiripi, Pope & Kessler 2007; Scott, Bruffaerts, Simon, Alonso, Angermeyer, de Girolamo et al. 2008).

In 2002 one expert said:

“In summary, the overall evidence that obesity impairs perceived health and quality of life is compelling and provides additional impetus for the already urgent need to develop better prevention strategies and treatments for this significant public health problem.” (Marcus 2002).

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<thead>
<tr>
<th>Relative risk (RR)</th>
<th>Metabolic consequences</th>
<th>Weight related</th>
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</thead>
<tbody>
<tr>
<td>Greatly increased</td>
<td>Type 2 diabetes</td>
<td>Sleep apnoea</td>
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<td></td>
<td>Gall bladder disease</td>
<td>Breathlessness</td>
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<td></td>
<td>Hypertension</td>
<td>Asthma</td>
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<td></td>
<td>Dyslipidaemia</td>
<td>Social isolation and depression</td>
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<td></td>
<td>Insulin resistance</td>
<td>Daytime sleepiness and fatigue</td>
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<td>Non-alcoholic fatty liver</td>
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<td>Moderately increased</td>
<td>Coronary heart disease</td>
<td>Osteoarthritis</td>
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<td></td>
<td>Stroke</td>
<td>Respiratory disease</td>
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<td></td>
<td>Gout/hyperuricaemia</td>
<td>Hernia</td>
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<td>Psychological problems</td>
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<tr>
<td>Slightly increased</td>
<td>Certain cancers</td>
<td>Varicose veins</td>
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<td></td>
<td>Reproductive abnormalities/impaired fertility</td>
<td>Musculoskeletal problems</td>
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<td></td>
<td>Polycystic ovaries</td>
<td>Lower back pain</td>
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<td>Skin complications</td>
<td>Stress incontinence</td>
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<td></td>
<td>Cataract</td>
<td>Oedema/cellulitis</td>
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Table 1. Total health consequences of obesity – Relative Risk (RR). Source: NICE GC43 Management of Obesity in Clinical Settings 2006

In June of this year in International Journal of Eating Disorders one discussion paper asked the question “Obesity: Is it a mental disorder?” (Marcus & Wildes 2009). The article defined mental disorders in order to test the question posed. The author cited the high rates of obesity among individuals with binge eating disorder, bipolar disorder, major depressive disorder, schizophrenia, and other psychological disorders. In this article they also looked at similarities among obesity, drug addiction, and other compulsive behaviours, and addressed suggestions that “compulsive food consumption” should be included as a mental disorder. The paper
concluded that there was insufficient evidence to claim that obesity is a mental disorder but did point out that there is evidence that obesity is “related” to mental disorder and many of the medications used to treat psychiatric illness.

What is clear is that there are only unfavourable outcomes from a health perspective from being overweight and this applies to both physical and psychological health.

The ‘Medicalization’ of Obesity

Although obesity has been claimed as being unhealthy for centuries, the medicalization of obesity really began in the 1950’s growing dramatically during the 60’s and 70’s (Maurer & Sobal 1995). Obesity was slotted into several medical models, within which it was considered as a genetic, endocrine, environmental, personality or addictive disorder, resulting in multiple medical strategies involving many health professionals in its treatment.

Obesity became officially classified as a disease in 1990 by the World Health Organisation, however this notion is now widely challenged and most obese adults have difficulty in accepting their weight as a disease. During this time however, obesity was transformed from a moral deviance the responsibility of which lay with the individual, to a disease with clinical legitimacy, thus providing a rationale for therapeutic intervention. As a consequence the importance of socio-cultural and environmental conditions was diminished as individual factors (lifestyle, personal choice and behaviour) became the focus of explanation, diagnosis and intervention. Even the mass public health campaigns promoting healthy lifestyles in America in the 1970’s and 1980’s directed action towards changes in individual behaviour, minimizing the significance of the broader socio-cultural and environmental determinants (Chang & Christakis 2003).

Interestingly a similar approach towards changing individual behaviour is now at the very heart of the current UK ‘Change 4 Life’ Social Marketing campaign. Although some individuals are able to make and maintain change, the
medical model largely ignores the external forces contributing to the development and maintenance of obesity. Patients leave the weight management programme only to re-enter the same environment that led to their weight gain in the first place. The commercial, social and structural environmental forces are still powerful.

Considering obesity as a disease in the traditional sense is probably unhelpful and an immediate challenge may be to re-define how we think about the ‘epidemic of obesity’. Whilst severe obesity is likely to confer clinical co-morbidities in later life, the causal factors behind population obesity are social, environmental and behavioural, not pathological. To medicalise obesity is to give it disease status, critically diverting attention and resources away from the omnipresent environmental pressures, and deflecting the onus away from the custodians of policy and planning that are ultimately responsible for shaping our obesogenic environments.

The public health model of obesity differs from the medical model in that it has an emphasis on prevention and that it considers a wider range of causative factors. A key step in addressing health problems in a public health model is identifying and modifying disease vectors. An effective public health approach to obesity would seek to modify identified causative vectors.

**The Obesogenic Environment**

Obesity is a normal response to an abnormal environment. The more urbanised the environment, the more obesogenic pressure is exerted on its population. Asking that population to make better choices whilst the current against them flows faster and faster, is futile as well as condescending. The environmental pressures that make the healthy choice the difficult choice mean that many people are growing up in a world where healthy behaviours are rarely encountered and they are slowly yet surely learning, practicing and reinforcing how to be unhealthy.

The term ‘obesogenic environment’ refers to the role environmental factors may play in determining both energy intake and expenditure. The term was first coined in the 1990s in the context of a hypothesis that might explain the current obesity epidemic. It has been defined in the Foresight Survey as: *The sum of the influences that the surroundings, opportunities or conditions of life have on promoting obesity in individuals and populations.* (King D 2007). The term embraces the entire range of social, cultural and infrastructural conditions that influence an individual’s ability to adopt a healthy lifestyle. The Foresight Authors state that understanding and preventing obesity is complex and that there is no example anywhere in the world where the obesity trend has been reversed.
Environmental influence on behaviour

Brian Wansink has investigated environmental influences that are beyond our conscious awareness. For example the “stale movie-popcorn study,” which showed that moviegoers ate more popcorn - even stale popcorn from a larger tub than from a smaller tub (Wansink & Cheney 2005). Wansink also devised the “Super Bowl chicken bone study,” which showed that leaving the chicken wing residue on the table (rather than quickly clearing the table) acted as negative feedback, so that those with residue on the table ate fewer chicken wings than did those without (Wansink & Payne 2007). Such examples illustrate the complexity of feeding and the subtle underlying environmental cues that can encourage passive overconsumption.

Environmental influences on diet also involve ease of access to food and drink. As eating habits become more unstructured, the availability of and access to ‘food on the go’ is an important consideration. Studies in the US suggest that the availability of high quality, reasonably priced ‘healthy’ food is in short supply for those who live in low-income neighbourhoods and such limitation is associated with poor diet and obesity (White 2007).

Opportunities for Physical Activity

The effect of technological development has been to continually engineer physical effort out of the environment. Cars, television and computer games are examples of technologies that have had such effects in recent decades. The built environment is now also coming under increasing scrutiny as a contributor to obesity. Whilst there is currently a lack of conclusive evidence on how and to what extent obesity is encouraged by the environment, some trends and themes are emerging. The evidence suggests that neighbourhood design and recreational environment does influence physical activity particularly where ‘Walkability’ is concerned. (De Bourdeaudhuij, Sallis & Saelens 2003; Saelens, Sallis, Black & Chen 2003; Fox K.R. and Hillsdon M 2007). Interestingly one study found that the mere inconvenience of owning a car in higher-density neighbourhoods encourages more walking or cycling than the actual urban structure (Lake & Townshend 2006).
Also how we perceive our environment has significant but modest associations with additional walking and physical activity (if you think your neighbourhood is safe and convenient you will walk around it more). Perceptions of social nuisances may increase the risks of obesity, while good access to leisure centres and living in a suburban environment reduce the risks (Poortinga 2006). However, it may be that these findings are affected by reverse causality, whereby those already engaging in higher levels of physical activity perceive their environment differently to people who are more sedentary in their lifestyles.

The Global Nutrition Transition

Cheaper processed foods tend to be more energy-dense and nutrient-poor (high macro / low micro) providing many calories but relatively few vitamins and minerals and other non essential but health promoting compounds such as phytochemicals and antioxidants. US studies show the cost of fruits and vegetables to have increased as a component of food budgets, while fats and oils, starches and sugars have decreased. Cheap non-perishable food commodities are now being mass produced, ignoring the old nutritional adage that: “Good food goes bad!”

Figure 4: Increase or decrease in “real” cost of food items in US over 20 year period. Source: Institute of Agriculture and Trade Policy USA 2004.
The pervasive and powerful marketing of energy-dense foods, particularly to children, the composition, presentation and supersizing of cheap energy-dense food and the proliferation of fast-food outlets are driving the global adverse nutrient transition (Koplan, Liverman & Kraak 2005). This shift to processed foods is also illustrated by the trends in UK sugar utilisation where the substantial decline in household purchase of sugar has been matched by an increase in sugar being used in manufactured food and beverage products e.g. soft drinks, snacks, confectionery (Lobstein T. and Jackson Leach R 2007).

The conclusion of the Foresight Evidence Review on Obesogenic Environments was that environments do influence levels of physical activity and obesity. However, influences of the environment are probably small and the mechanisms remain unclear. Furthermore, it is certainly the case that changes to the environment alone are unlikely to solve the problems of increasing obesity and declining physical activity levels. (Jones, Bentham, Foster, Hillsdon & Panter 2007).

A Social and Cultural Perspective

Arguably once a problem has become as prevalent in Western cultures as obesity has, it is no longer a medical problem but a social one. The Western cultural environment is one of excess, with ever-increasing portion sizes, instant gratification and the ‘more for less’ mindset. Westerners may benefit from observing and listening to other cultural groups who have a greater respect for and more sophisticated relationships with food. Ironically though, some of those groups that emigrated away from such cultures and now live in the UK are experiencing the highest levels of obesity.

Considering the body within its social and environmental context may herald a new focus on the structural and environmental aspects of obesity. A deeper understanding of how the cultural and social aspects contribute to and support the ‘obesogenic’ environment could assist in devising more successful strategies to address obesity as a social issue. The parody of the diminishing perceived ‘ideal’ female body against the reality of the persistent expansion of the actual body, starkly illustrates the social dilemma of bodyweight in the UK.
The revenues generated by the plethora of weight reducing aids provided by the diet and weight loss industry, food industry, fitness Industry and pharmaceutical / herbal sector is testimony to the fact that people are trying to control their weight, and paying a hefty price for it. However, obese people are still held responsible for their body status, and the stigma of obese people as greedy and lazy individuals persists.

Not only is the prevalence of obesity increasing, so too is the severity, both in children and adults. It is apparent that society is now grappling with the uneasy relationship between personal choice and the abdication of responsibility. As health costs and the burden of taxation spirals, there will be inevitable unease surrounding health consequences which are considered to be self inflicted.

One press report recently was the latest in a number of recent reports of children being taken into care as a result of their weight. In the latest report, two children from Dundee aged only three and four years old, were controversially removed by social workers after concerns that they were beginning to become dangerously overweight. It was reported three weeks later that all seven children including the newborn child of these parents were removed for the same reasons (White 2009). This illustrates a new level of intolerance towards parents that fail to maintain their children within acceptable weight parameters. Sadly this case offers a glimpse of a potential new social chapter in the escalating challenges brought about by a burgeoning obese population.

**Economic consequences of an obese population**

A recent review found a positive relationship with overweight and obesity and long term sick leave in the workplace (van Duijvenbode, Hoozemans, van Poppel & Proper 2009). This confirmed the work of Suhrcke and colleagues which highlighted the importance of healthy people to an economy. The team examined the link between health and wealth in rich
countries and found that healthier populations have higher earnings (Suhrcke, McKee, Sauto Arce, Tsolova & Mortensen 2006). Professor McKee involved in the work said:

“The Treasury has identified the cost of obesity to the NHS as a major problem but our research shows how much healthy people contribute to the health of the economy. They remain in the workforce longer and are more productive while they are at work.”

The cost of obesity in the UK is estimated at up to £3.7 billion per year, including £49 million for treating obesity; £1.1 billion for treating the consequences of obesity; indirect costs of £1.1 billion for premature death and £1.45 billion for sickness absence. By adding similar costs for the overweight population the estimation is nearer £7.4 billion per year (Department of Health 2004). Unless the current trend in obesity is halted and reversed, the cost will be in the magnitude of £46 billion by 2050 (equivalent to four Olympic Games every year), with a seven fold increase in NHS costs alone (Foresight, 2007).

**Conclusion**

Obesity experts now agree that the epidemic of obesity is a public health crisis (Haslam & James 2005). It is causing an immense burden of morbidity and mortality resulting in enormous economic, social and human costs.

Obesity is probably the paramount public health issue facing the UK today. Considered in financial terms alone, if the projections of the Foresight survey for 2050 materialise (£46bn annually), obesity will render the UK insoluble long before this date, with unimaginable adverse consequences on public health and welfare.

This Government (and successive Governments) must act immediately to implement the wide scale, long term, social, educational and environmental policies required to arrest the current public health threat of obesity, in order to safeguard the future health and wealth of the nation.
References


Chakravarthy, M. V. & F. W. Booth (2004). Eating, exercise, and thrifty genotypes. Division of Endocrinology, Metabolism and Lipid Research. Department of Internal Medicine. Missouri 65211 Washington University School of Medicine, St. Louis 63110.


