Factors predisposing to obesity: a review of the literature

Ali AT, PhD
Crowther NJ, BSc(Hons), DPhil
Department of Chemical Pathology, National Health Laboratory Service, University of the Witwatersrand Medical School, Parktown, Johannesburg
Correspondence to: Prof Nigel Crowther, e-mail: nigel.crowther@nhls.ac.za
Keywords: obesity, caloric intake, genetic factors, evolution

Abstract

The rising prevalence of obesity is a worldwide problem affecting not only the developed world but also developing nations such as South Africa. Excess body fat deposition is caused by an imbalance between energy intake and energy expenditure and there are many genetic and environmental factors that can influence this balance. The present article will describe these factors and discuss the complex interaction between the environment and the human genome that may underlie the current obesity epidemic.

JEMDSA 2009;14(2):81-84

Introduction

A famous ancient proverb states: eat breakfast like a king, lunch like an ordinary person, and your dinner like a beggar. These words of wisdom have long been discarded. Modern life has brought with it more food with high caloric density and better taste. New technology has made life easier and less active, and the result is a worldwide epidemic of obesity and its associated disorders.[1,2] Obesity involves both increased fat cell size and number[1] and occurs when energy intake is greater than energy expenditure. This balance between energy input and energy output can be affected by many factors including the quality and quantity of dietary intake, environmental and genetic inputs and physiological and psychological status.

Obesity is a common and serious medical problem all over the world[1,2] and South Africa is one of the developing countries that has been most affected by the current obesity epidemic. One of the possible reasons for the rise in prevalence of obesity in South Africa is the migration of populations from rural to urban areas, which has been shown to be associated with significant lifestyle changes particularly the increased availability and therefore consumption of calorie dense, fatty food.[3] Within South Africa older women (age range 45–54 years) have a significantly higher level of obesity (mean BMI of 29.4 ± 0.27) than their younger counterparts (age range 15–24 years, mean BMI of 23.4 ± 0.13) with middle-aged, urban African females having the highest prevalence of obesity.[4]

Obesity is not only a problem found in the adult population but is also occurring at an increased frequency in children in both the developed and the developing world. Thus, a national survey among South African school children showed that the prevalence of overweight in black, female students was 20.9% compared to 4.2% in males.[5] This study also noted that white males were heavier than males of other races while black and white females were heavier than coloured females.[5,6] In general, young females were heavier than young males[6] and among black teenagers these differences were attributed to overeating in females compared with undereating in males, in whom the prevalence of underweight (17% compared to 3.9% in black females) was higher than in all other groups.[5,6] It can therefore be seen that within all age groups in South Africa there are gender and ethnicity related differences in the prevalence of obesity (see Table I). These differences are probably a result of cultural, socio-economic and genetic factors which also underlie the worldwide obesity epidemic. This review will address all these influences with an emphasis on the most recent literature.

Table I: The prevalence of obesity in male and female adolescents and adults in different South African ethnic groups

Ethnic group	Age 13–19 years*		Age 15–95 years**	
	Male	Female	Male	Female
African	1.9	5.3	7.7	30.5
European	4.8	7.7	19.8	24.3
Coloured	2.8	3.8	9.1	28.3
Indian	–	–	8.7	20.2

Data given as % values. *Data taken from reference number 6; **data taken from reference number 4

Caloric intake

Food intake can be affected by many factors, including the price, portion size, taste, variety, and accessibility of foods. The method by which the food is prepared is also important. There are also strong cultural influences on the types of food consumed with some societies abstaining from particular types of food or only eating food if it has been prepared in a specific manner.

A high fat diet enriched with saturated fatty acids is the common diet in developed countries whilst in poorer countries the majority of people derive their calories from a vegetarian diet.[7,8] Diet may affect

body weight by controlling satiety and metabolic efficiency, or by modulating insulin secretion and action.[9] Thus, the calorie dense diet common in the western world may predispose to obesity via elevated postprandial insulin levels resulting from the high carbohydrate intake which leads to increased triglyceride storage in the adipose tissue depots.[10] High insulin levels may also provoke a vicious metabolic cycle. Insulin induces hunger by depleting the glucose levels of the blood, and this promotes further food intake which leads to greater insulin secretion. Ultimately, this cycle will lead to weight gain and chronic hyperinsulinaemia.[11,12] It has also been observed that obese subjects have an increased preference for fatty foods[13] which will also enhance insulin output and triglyceride storage.

The modern diet of developed and developing countries contains more fat and considerably less fibre than the recommended levels. Thus, in one large epidemiological study, fat constituted 37.8% of the total energy intake compared to a recommended level of $< 30.0\%$, whilst fibre intake was 8.6g/1000 kcal per day compared to a recommended intake of 14g/1000 kcal.[14] Studies have shown that food containing saturated fat results in greater weight gain compared to food containing unsaturated fatty acids.[15,16] Fatty acids activate peroxisome proliferator-activated receptors delta and gamma (PPAR δ, PPAR γ), which promote adipogenesis, and expansion of adipose tissue depots.[17] Epidemiological studies have confirmed the positive correlation between a high-fat diet and the development of obesity.[18,19]

Socio-economic status and level of education

Obesity is a common feature in migrants, where a population with a common genetic heritage live under new socio-economic and cultural conditions. Pima Indians, who live in the USA, are on average 25 kg heavier than Pima Indians who live in Mexico.[20] Migration of Asian-Indians[21] and Australian Aboriginals[22] from rural areas to an industrialised environment is related to an increasing prevalence of obesity in these societies.

Studies have found that BMI is significantly higher among low socio-economic than middle and high socio-economic groups[23,24] with lower socio-economic status (SES) being associated with accelerated weight gain during adulthood.[25,26] Thus, data from the Whitehall study shows that over a five year period of follow-up, subjects with a clerical post had a two fold greater risk of an increase in BMI of > 3 units than subjects with an administrative post.[26] The effect of socioeconomic status (SES) on the prevalence of obesity may be mediated by low income which will limit the availability of the more healthy food options.

In many populations the level of education is inversely associated with obesity especially in women,[23,27] while husbands' education was found to be correlated negatively with the prevalence of obesity in their wives.[27] Conversely, in a national obesity survey in South Africa, a multivariate regression analysis demonstrated that women with greater than 12 years of education had higher BMIs than women with 1–12 years of education ($p < 0.0001$). A possible explanation for this phenomenon is that women in the latter group tend to perform higher levels of manual labour than the more educated women.[4]

Genetic factors

Genetic factors may act as determinants of BMI by affecting energy balance. More than 300 genes, markers, and chromosomal regions have been found to be associated with various human obesity phenotypes[28] and it has been estimated that 30–70% of the variance in BMI in humans can be explained by genetic factors.[29]

The first monogenic human obesity syndrome, congenital leptin deficiency was reported in 1997.[30] The discovery of the leptin gene has dramatically changed our understanding of the role that adipose tissue plays in the regulation of energy balance and appetite.[31] Leptin acts within the arcuate nucleus of the hypothalamus to decrease the expression of orexigenic signals and increase the levels of anorexigenic signals and thus reduce food intake.[30] A number of other forms of monogenic obesity have been discovered and each of the affected genes has been shown to be expressed in the hypothalamus and to play a part in the control of appetite.[32] However, these gene mutations explain only a very small proportion of cases of human obesity. The common form of obesity is a polygenic disease and it is thought that each of the polymorphisms involved contributes in only a small way to the phenotype and this may explain why it has been very difficult to unravel the genetic aetiology of human obesity. However, recent advances in gene screening techniques have allowed geneticists to perform high throughput, whole genome analyses and uncover a number of new gene loci that may play a part in causing increased adipose tissue deposition. Most of these genes are thought to be expressed in the CNS and to be involved in controlling food intake.[33] The genetic variant with the strongest association to the polygenic form of obesity lies close to the FTO (fat mass and obesity associated) gene.[34] This association has been confirmed in a number of large population studies, however the exact function of the FTO gene remains a mystery although expression studies have demonstrated that this gene is expressed in a wide range of tissues with high expression in the brain.[34]

Factors acting early in life, and during puberty, pregnancy and aging

In both genders rapid weight gain during infancy is an important risk factor for later obesity. Thus, children who showed rapid weight gain or catch-up growth between zero and two years of age have higher measures of adiposity at five years of age than children who did not undergo catch-up growth.[35] It is known that BMI falls in neonatal life and then increases in infancy. This increase in adipose tissue mass is known as adiposity rebound and children who experience adiposity rebound at an earlier age have a greater chance of being obese in adulthood.[36] Studies have also shown that the relationship between birthweight and adult obesity is U-shaped with low birthweight being associated with increased measures of adult abdominal fat deposition whilst high birthweight is associated with higher adult levels of overall body adiposity.[37]

Females have a higher prevalence of obesity than males and it has been suggested that this may be related to gender differences in the brain's response to hunger and satiety.[38] Furthermore, factors acting during puberty have been shown to influence the risk of obesity in females. Thus, a longitudinal growth study performed in Finland demonstrated that at the age of 31 the prevalence of obesity in females who reached menarche before the age of 11 was 15%

compared to 4% in those who reached menarche after 15 years of age.[39] The reason for this may be that fat accumulation during childhood increases the chances of early menarche[40,41] or that girls with early sexual maturation have a longer period of positive energy balance.[42]

A number of studies have shown that a positive relationship exists between gestational weight gain and postpartum weight retention.[43-45] However, the level to which weight is retained after parturition differs across societies. It has been demonstrated that American and Swedish women retained between 1.5 and 3.0 kg twelve months after delivery,[46,47] whilst in Brazilian females, 20% of mothers retained more than 7.5 kg nine months after delivery.[45] Another study showed that black women were twice as likely (odds ratio, 2.2 and 95% CIs of 1.5–3.2) as white women to retain more than 20 lb in weight postpartum, despite comparable weight gain during pregnancy.[44]

Body adiposity increases with age. This is because as people grow older their metabolic rate falls and energy expenditure decreases. Thus, older subjects do not require as many calories to maintain their body weight. If caloric intake remains constant or increases they will therefore gain weight. Men require more calories to maintain their body weight, because they have a higher resting metabolic rate than women. In postmenopausal women, obesity is a result of decreased metabolic rate and alterations in ovarian hormones, which accelerates the age-related increase in body fatness and decreases energy expenditure.[48] Thus, women have a higher BMI than men, especially after the age of 50 years.[49]

Psychological factors

Psychological status can influence eating habits, because most people eat in response to negative emotions. Stress for example, not only increases consumption of food but also shifts consumption toward high caloric foods that are normally avoided.[50] It is thought that the effect of stress on food intake is mediated via increased adrenal glucocorticoid (GC) output. Chronically elevated GC levels can give rise to increased intake of 'comfort foods' which in turn leads to abdominal obesity.[51] This hypothesis has been developed and tested successfully in rodent models,[51] however more studies are required in humans to confirm its validity.

Depression and some neurological problems can also promote overeating which will ultimately lead to increased fat accumulation.[52] A number of studies have shown a higher prevalence of borderline personality disorder in obese patients (ranging from 1.1 to 30.4%) compared to the general population (prevalence of 2%).[53] Such data suggests that psychopathology may have an impact on weight loss and weight maintenance, and may be an important factor that should be considered when devising intervention strategies in obese subjects. It is also noteworthy that one study has shown a higher prevalence of suicides in subjects following bariatric surgery (15 suicides out of 7 925 subjects) when compared to a control group of untreated, obese subjects (5 suicides out of 7 925 subjects).[54] This suggests that psychological disorders may be present in morbidly obese subjects and that these are not attenuated by surgical intervention. It has therefore been suggested that patients undergoing bariatric surgery are assessed for psychological disorders before treatment and are monitored after surgery.[54]

Studies conducted on families of patients with morbid obesity have suggested that anomalous eating habits of families, parental conflicts and parents' psychopathology may influence weight gain in children.[55,56] Familial influences on childhood obesity differ according to the gender of both the parent and child.[55] Dysfunctional patterns in these families which lead to overeating include regressive coping styles such as stress eating, lack of self-esteem, unsatisfactory personal relationships, and stigmatisation of the obese individuals.[57]

Other factors

The aetiology of obesity is obviously multi-factorial. The current article has discussed those factors that have received the majority of attention in the scientific literature but there are others that have received far less attention but may still be important. These factors have been the subject of a recent review[58] and therefore they will only be briefly discussed here.

Sleep duration: It has been shown in human studies that BMI is inversely correlated with sleep duration.

Smoking: A number of investigations have clearly shown that smokers are less obese than non-smokers and that cessation of smoking leads to weight gain.

Pharmaceuticals: A number of drug types lead to increased weight gain and these include antidepressants (e.g. serotonin re-uptake inhibitors), contraceptives, corticosteroids, antidiabetic agents (e.g. insulin, sulphonylureas and thiazolidinediones) and medications used for treating hypertension (e.g. beta adrenergic receptor antagonists).

Maternal age: Studies in humans and animals have shown that there is a positive correlation between maternal age at birth and BMI of the resulting offspring. Thus, one study has shown that for every five year increase in maternal age, the risk of obesity in the offspring increases by 14.4%.[59]

Increased life expectancy: BMI is known to increase with age and therefore as life expectancy for humans increases the relative frequency of older individuals within the population will increase and hence so will the prevalence of obesity.

Endocrine disruptors: These agents are by-products of industrial processes and leak into the environment and hence into the food chain. These molecules are able to elicit endocrine responses and include agents that have oestrogen-like effects (e.g. vinclozolin and bisphenol A) and also substances that are able to activate adipogenesis via interaction with transcription factors.

Environmental temperature: The advent of air conditioners has meant that humans spend less time in temperatures outside the thermoneutral zone (TNZ). The TNZ is the range of temperatures over which changes in metabolic rate are not required to maintain normal body temperature. Once a person leaves the TNZ, energy expenditure will increase in an attempt to maintain body temperature at the required level. Thus, in humans who are constantly within the TNZ, energy expenditure is reduced and this predisposes to weight gain at lower levels of energy intake when compared to subjects who do not spend large periods of time within the TNZ.

Reproductive fitness: The BMI of parents has been shown to be positively related to increased number of offspring for both mothers and fathers. Therefore, because of the strong genetic component to BMI this will lead to increased transmission of obesogenic gene variants.

Conclusions

Obesity is a multi-factorial disorder with major contribution from the environment and the genome. The maintenance of a large number of genetic variants within the genome that give rise to increased adipose tissue mass may be explained by the process of natural selection. It has been hypothesised that during human evolution there was selection for any genotype that favours energy storage because this would enhance survival during periods of famine. Famine is known to be an important and consistent occurrence during the evolution of the human species. However, this genotype is only advantageous under conditions of food scarcity and is deleterious in conditions where food availability is high and energy expenditure is low i.e. the prevailing environment! Thus, obesity is the result of an unfavourable interaction between our current environment and our ancient genome. The process of natural selection is not fast enough to modify our genome in response to rapid changes in environmental conditions. This genomic inertia has led to many mass extinction events during the life course of planet Earth. The only solution to the problem of the obesity epidemic is therefore a rapid change in environmental conditions to better match our present genetic make-up. Such changes must occur at the individual level and be encouraged by changes at the population level. However, societal inertia is a major stumbling block and it is therefore possible that the ultimate demise of the human species will be the result of a clash between a highly evolved genome, sculpted by millennia of fine tuning and a human-built, change-resistant environment crudely cobbled into existence over mere decades.

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