Etiology Of Obesity- What A Family Practitioner Needs To Know

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Etiology Of Obesity- What A Family Practitioner Needs To Know

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**Abstract**

This article reviews the various etiological factor of obesity. In simple language it focuses on genetics, causes, and determinants.

**Introduction**

There is no single definition of obesity. It may be simply an extreme degree of overweight - but a person can be overweight without being obese. In contrast, a person in a normal weight range but with very sedentary habits could have a small muscle mass, be storing excess fat, and thus be classifiable as obese. Obesity has become epidemic in the U.S. and other developed countries. More than 58 million Americans are overweight, and that includes at least 1 in 5 children.

Obesity is a leading preventable cause of death worldwide, with increasing prevalence in adults and children, and authorities view it as one of the most serious public health problems of the 21st century.[1] Obesity is stigmatized in the modern Western world, though it has been perceived as a symbol of wealth and fertility at other times in history, and still is in many parts of Africa.[2, 3]

Obesity refers to an increase in total body fat. The easiest and most widely accepted method of determining whether you are obese is by measuring your Body Mass Index, or BMI. To calculate your BMI, follow these steps:

BMI is the initial step in the clinical assessment of the patient [7, 8]. This is calculate as the body weight (kg) divided by the stature [height (m)] squared (wt/ht²), or body weight (1b) X 703divided by the height (stature) squared [(wt (1b) X 703)/ [ht (in)] ²]. BMI correlates well with body fat and is relatively unaffected by height.

**Causes of obesity**

**Food systems causes of obesity**

The main problem has been the increased availability of high energy food, because of:

- Liberalized international food markets
- Food subsidies that “have arguably distorted the food supply in favour of less healthy foodstuffs”
- Transnational food companies have flooded the global market with cheap to produce, energy dense, nutrient empty foods.
- Supermarkets and food service chains encouraging bulk purchases, convenience foods, and supersized portions.
- Healthy eating often being more expensive than less healthy options, (despite global food prices having dropped on average).
- Marketing, especially “food advertising through television [which] aims to persuade individuals particularly children, that they desire foods high in saturated fats, sugars, and salt.”

**The local environment and obesity**

How people live, what factors make them active or sedentary are also a factor. For example, research, mainly in high income countries, indicates that local urban planning and design can influence weight in several ways.

For example, levels of physical activity are affected by:

- Connected streets and the ability to walk from place to place
- Provision of and access to local public facilities and spaces for recreation and play
- The increasing reliance on cars leads to physical inactivity, and while a long-time problem in rich countries, is a growing problem in developing countries.

**Social conditions and obesity**

Examples of issues the BMJ noted here include:

- “Working and living conditions, such as having enough money for a healthy standard of living, underpin compliance with national health guidelines”
- Increasingly less job control, security, flexibility of working hours, and access to paid family leave...undermining the material and psychosocial resources necessary for empowering individuals and communities to make healthy living choices.
- Inequality, which can lead to different groups being disadvantaged and having less access to needed resources and healthier foods.

**Antipsychotics and Weight Gain**

The second-generation antipsychotics vary in their propensity to induce weight gain; clozapine and olanzapine produce the most weight gain, quetiapine and risperidone produce intermediate weight gain, and ziprasidone and aripiprazole produce the least weight gain [10, 11]. Available evidence suggests that differences in weight gain associated with these agents...
reflect their order of risk for insulin resistance, glucoregulatory dysfunction, and dyslipidemia [9, 11]. The mechanisms by which antipsychotic medications produce weight gain may include stimulating appetite, reducing physical activity, and directly impairing metabolic regulation. The daily balance between calories consumed and expended determines an individual’s weight, and even small imbalances can cause significant changes in weight. On average, a 3% increase in daily caloric intake without an increase in energy expenditure results in a 10-lb. weight gain over a 1-year period [12]. Many drugs marketed to induce weight loss suppress appetite and hunger by enhancing the action of monoamine neurotransmitters (serotonin, norepinephrine, and histamine) in the CNS [13]. Conversely, many second-generation antipsychotics inhibit or reduce the activity of these sameneurotransmitters and thus may increase appetite.

A recent animal study reported weight gain associated with olanzapine, quetiapine, risperidone, and ziprasidone but increased food intake only with olanzapine and quetiapine [14]. Not all antipsychotic-induced metabolic disturbances result from increased adiposity. Some patients taking second-generation antipsychotics experience new-onset diabetes without changes in weight, and experimental studies demonstrate medication-associated insulin resistance independent of adiposity [15]. Metabolic disturbances related to second-generation antipsychotics may result from a direct alteration of insulin sensitivity and/or insulin secretion. Antipsychotic affinity at both histamine and muscarinic acetylcholine receptors correlates with weight gain and metabolic liability [16], and impaired parasympathetic regulation of beta cell activity may contribute to metabolic risk [17]. Insulin sensitivity may also be reduced as a result of alterations in gene products in the insulin-signaling pathway and/or elevated levels of circulating factors that alter insulin signaling. For example, there is evidence that certain antipsychotic agents may directly impair glucose transporter function. Glucose transporters are regulated by insulin and actively transfer glucose into peripheral tissues (e.g., liver, muscle, and fat). Direct attenuation of glucose transporter function by antipsychotic agents would result in elevations in circulating glucose and a compensatory hypersecretion of insulin, which over time may further reduce insulin sensitivity, triggering the cascade of events leading to metabolic syndrome and type II diabetes [18].

In addition to overweight and obesity, each element of the metabolic syndrome can be considered an independent treatment target to reduce the risk of cardiovascular disease [19]. Metformin reduces insulin resistance, reduces new-onset coronary heart disease in obese patients with diabetes, and prevents or delays type 2 diabetes in patients with impaired glucose tolerance [20, 22]. Insulin sensitizers of the thiazolidinedione class also prevent delay type 2 diabetes in at-risk patients [19]. Neither has been tested extensively in patients with impaired glucose tolerance induced by second-generation antipsychotics. Hypertensive patients who are taking second-generation antipsychotics and meet criteria for the metabolic syndrome should be treated with therapeutic lifestyle changes and medications in accordance with hypertension guidelines [21]. Finally, low-dose aspirin may be indicated to mitigate the prothrombotic state in patients with metabolic syndrome at elevated risk of coronary heart disease.

Genetics

Obesity is a major feature in several syndromes, such as Prader-Willi syndrome, Bardet-Biedl syndrome, Cohen syndrome, and MOMO syndrome. (The term “non-syndromic obesity” is sometimes used to exclude these conditions.) [4] In people with early-onset severe obesity (defined by an onset before 10 years of age and body mass index over three standard deviations above normal), 7% harbor a single point DNA mutation [5]. Studies that have focused upon inheritance patterns rather than upon specific genes have found that 80% of the offspring of two obese parents were obese, in contrast to less than 10% of the offspring of two parents who were of normal weight [6].

Genetic Dissection of Complex Diseases

Complex diseases such as diabetes or obesity have genetic components, which due to their polygenic nature can not easily be identified. Two basic approaches have been used to identify susceptibility genes for complex diseases: candidate gene approach and genomic approach (fig. 1). However, only limited success has been seen so far.

Fig. 1. Identifying genetic factors that underlie complex diseases.

(QTL Quantitative trait locus)

The Determinants of Obesity:

Obesity and mental health

Social determinants of obesity such as poverty and area deprivation are also associated with mental disorders such as depression and schizophrenia. Mental health problems are also risk factors for obesity in their own right, and there are strong associations between some of these disorders, such as depression [23] and schizophrenia [24, 25], and obesity [26].
contributing factor is that some of the medication prescribed for mental health problems can cause weight gain [27, 28].

There is also strong evidence that relates poor self-esteem to obesity [29], especially in children and adolescents [30]. Obese girls are more likely to suffer from serious emotional problems and hopelessness [31]. An association between depression and obesity has been described in clinical and community studies. Children and adolescents with major depressive disorder may be at increased risk of developing overweight, and obese people seeking weight-loss treatment may have elevated rates of mood disorders. Obesity is associated with major depressive disorder in females; however, most overweight and obese persons in the community do not have mood disorders [32].

Several surveys have noted that the great majority of food advertisements, especially those shown during children's television programmes, encourages the consumption of energy-dense foods and beverages. A systematic review of the scientific evidence, conducted for the United Kingdom Food Standards Agency in 2003 [33], concluded that sufficient evidence shows that advertising increases the overall consumption of food categories, as well as choices between brands. A review by the United States Institute of Medicine in 2006 [34] found strong evidence that advertising affects overall diet in the short term for children aged 2–11 years and moderate evidence of long-term effects on children aged 6–11 years. This review also noted strong statistical evidence linking higher exposure to television advertising and obesity among children aged 2–11 years and adolescents aged 12–18 years. Children’s exposure to television advertising of energy-dense foods is associated with an elevated prevalence of overweight, and exposure to the advertising of healthier foods is weakly linked to a reduced prevalence of overweight [35].

Various drugs can cause weight gain, including a variety of psychotic agents [36] and hormones. Some phenothiazines and many of the ‘atypical’ antipsychotic are particularly prone to causing weight gain. This increase in weight is primarily fat and is associated with an increase in respiratory quotient, there is an increase in carbohydrate utilization, which might stimulate food intake. Metabolic rate does not change. [37]

Some anti depressants also can cause weight gain. The tricyclic antidepressant amitriptyline is a common culprit and may also increase the preference for carbohydrate. Lithium also has been implicated in weight gain. Two antiepileptic drugs, valproate and carbamazepine, which act on the glutamate receptor, cause weight gain. Insulin stimulate appetite, probably through hypoglycemia. Weight gain occurs not only in patients with diabetes treated with insulin but also in patients treated with sulfonlyureas, which enhance endogenous insulin release, and with glitazones, which act on the peroxisome proliferators-activated receptor γ (PPAR-γ) receptor to increase insulin sensitivity. [38]

**Dietary influences on obesity**

The modern food environment provides a wide range of opportunities to consume food and drink products. These are then are readily consumed, which inadvertently leads to what has been described as “passive over-consumption”, where the individual has no way of recognizing that he or she is eating a particularly energy-dense food or drink. The recent analyses of different studies on individual responses to food, assessing spontaneous intake both in carefully controlled environments [39, 40] and in everyday life, all point to two dietary factors that are particularly conducive to inadvertent overeating: a) the consumption of diets which are very energy-dense, i.e. high in calories per unit weight because extra fat and/or sugars have been added, because the food has been refined to limit its water-holding and bulking properties, or because fruit and vegetables are marginally present; and b) the consumption of energy-rich drinks, such as sugary drinks, between meals. These two factors seem to evade the normal biological short-term regulation of appetite and food intake, so children and adults tend not to adjust their intakes when these foods and drinks are constantly offered. This problem is then accentuated in sedentary societies, where people need to eat less in general and where it is therefore more difficult to maintain an energy balance when energy-dense foods and drinks are consumed. Conversely, diets low in energy density, with lower proportions of fat, more complex carbohydrates and more fibre, protect against weight gain [41]. Intervention studies also show that a high intake of dietary fibre may assist in losing weight [42].

Such low-energy diets, however, should have an adequate density of micronutrients and bioactive compounds to supply the required micronutrients while keeping the energy intake low. Given this perspective, it is not surprising that sweetened beverages [43] and “fast food” [44] emerge as specific risk factors. In addition, large portion sizes of energy-dense foods increase the risk of excessive consumption [45], whereas the frequency of eating itself has not been shown to contribute specifically to weight change, when the type of food is the same. Unsurprising therefore, are the findings that higher intakes of fruit
and vegetables are linked to lower weight gains [46], while a high intake of meat (together with its associated fat) is linked to a greater risk of weight gain [47]. There is some evidence that alcohol contributes to obesity in men, but there is no consistent association. Some recent evidence links weight gain to foods with a high glycaemic index, but longer-term studies are needed to confirm this association. Although there are many cited reports which seem to contradict these conclusions, care needs to be taken with their interpretation, because many studies rely on self-reported intakes and even weight gains, where both measures are subject to large errors. Thus there is marked underreporting of total energy, fat and sugar intakes, especially in those most overweight [48].

Conclusion(s)

One very easily burn off one’s excess weight and keep it off. It is just a matter of knowing the right things to do and doing them. It does not involve radical diets or strenuous exercise regimes. Everything in moderation except for moderation, so the saying goes. It is very important that the case of fat psychological stable and balanced, and that for obese or overweight real desire to get rid of excess weight, and must be ready to develop the way we eat and the implementation of the recommendations of the physician carefully. Obesity Treatment plans are customized for the individual and will depend on clinical evaluation, existing treatment modalities and most importantly the patient’s desires.

References

Illustrations

Illustration 1

Fig. 1. Identifying genetic factors that underlie complex diseases. (QTL Quantitative trait locus)
Illustration 2

BMI: Body Mass Index: BMI = Kilograms/meter²

<table>
<thead>
<tr>
<th>WHO Guidelines</th>
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<tbody>
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<tr>
<td>18.5–24.9</td>
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</tr>
<tr>
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<tr>
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<td>35.0–39.9</td>
<td>Class 2 obesity</td>
</tr>
<tr>
<td>&gt; 40.0</td>
<td>Class 3 obesity</td>
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A normal BMI = 18.5-24.9; overweight = 25.0-29.9; obese = 30 or greater; and morbidly obese = 40 or greater.

Health impacts

With obesity comes increasing risks of

- Cardiovascular disease (mainly heart disease and stroke), already the world’s number one cause of death, killing 17 million people each year.
- Diabetes (type 2), which has rapidly become a global epidemic.
- Musculoskeletal disorders, especially osteoarthritis.
- Some cancers (endometrial, breast, and colon).

In addition, childhood obesity is associated with a higher chance of premature death and disability in adulthood.
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