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Obesity, the Pandemic of XXI[st] Century

Abstract

A comprehensive review about the current situation in obesity and its consequences. The authors in the first parts of the paper define obesity, describe the methods for assessment of obesity and the amount of fat in the body and discuss the question if obesity itself is a disease or not. According to their view obesity is not a disease but a pathological condition caused by a disturbance of appetite regulation, as well as a gateway to many diseases. In the next part they present the most important data about the epidemiology of obesity in the world and in Europe.

The consequences of obesity depend on the distribution pattern of the fat in the human body. Upper body obesity associated with visceral accumulation of fat is more dangerous as compared with mild gluteal obesity in young women. The subsequent chapters copmprise a simple overview of the pathogensis of obesity from the point of view of the energy metabolism and the genetic, epigenetic and environmental factors involved in the development of obesity.

The consequences of obesity heavily increase health care and indirect economic costs in rich countries. The most important among these are type 2 diabetes mellitus, coronary heart diseas, hypertension, stroke and gout.

The treatment of obesity is often a troublesome and frutrating process both for health-care providers and the affected people. Therefore the psychological and social aspects of overeating are of crucial importance to understand obesity and to elaborate successful ways to prevent it.

Keywords: obesity, metabolic syndrome, energy metabolism, nutrition

1. Introduction

It is one of the tragic ironies of modern civilization that while roughly one third of the population in low-income countries suffers from various forms of food shortage [1, 2], the most serious nutritional problem in the middle- and

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high-income countries is overnutrition leading to obesity. Especially alarming is the increase of obesity in children and young adults. Obesity itself is not only a disease, but first of all a risk and causal factor of many diseases with a high rate of morbidity, invalidization and mortality [3–6].

The consequences of obesity heavily increase both the direct (health care) and indirect (social) economic costs in rich countries. According to pessimistic forecasts [7] there is a menacing danger of decrease of average lifespan in USA if the pandemic of obesity will proceed at the same rate as in the past 20 years.

2. Definition and assessment of obesity

Obesity is a condition in which the relative proportion of body fat is increased. Another term, **overweight** (sometimes used interchangeably with obesity) signifies only an increase of body weight above an arbitrary standard usually defined in relation to height. To determine whether a person is obese or simply overweight (due to increased mass of muscles in athletes or in manual workers) techniques for measuring and of body fat are necessary [8].

From anthropometrical measurements comparing weight with height (or more exactly with the body surface), the **body mass or Quetelet index (BMI)** is preferred. BMI is calculated from body weight in kilograms divided by the square of height in meters (kg/m^2). A desirable BMI for general population lies between 19 and 25. According to BMI, individuals can be classified as underweight, normal weight and three grades of obesity (Tab. 1). According to this classification persons with BMI from 25 to 30 kg/m^2 have a moderately increased risk of health problems, those with a BMI from 30 to 40 kg/m^2 have a considerably increased risk, and those with a BMI above 40 kg/m^2 have a very high risk. On the other side these cutoff values are arbitrary and the increased risk of consequences begins to increase around BMI 27 kg/m^2. From medical point of view the main class (30–40) is too broad because in a person of 1,72 m height BMI 30 is equivalent to 88,7 kg and BMI 40 to 118,3 kg. The health risk between these two values is considerably different and therefore it is rational to speak about morbid obesity not from BMI 40 but from already from BMI 35.

Table 1. Classification of obesity according to BMI

BMI, kg/m^2	**Classification**
< 19,0	Undernutrition
19,0 – 25,0	Normal body weight*
25,1 – 30,0	Overweight or obesity grade I
30,0 – 40,0**	Obesity (grade II)
> 40,0	Morbid obesity (grade III)

* The correct term is “body mass” but in everyday use we prefer the incorrect term “weight”.
** This class should be divided into two because in this form is too broad (see in text).

A simple method for assessment of body fat and obesity is the **skinfold thickness** because over half of the fat in the body is deposited under the skin. The skinfold thickness is measured on four sites with Best's caliper (over the biceps and triceps muscle, in the subscapular and suprailiac region). A more exact result is achieved by measurements on 10 sites with Holtain's caliper. The proportion of body fat is estimated by applying equations derived from correlations with the reference underwater densitometric measurements.

Underwater densitometry is based on the law of Archimedes and calculates the ratio between fat tissue (density = 0,9007 g/cm^2) and lean body mass (1,100 kg/cm^2). Another reference method is air displacement pletysmography.

In general practice the currently most employed methods for the assessment of lean body mass and fat content are based in **bioelectric impedance (BIA)** based on different high frequency conductivity of tissues with different water content. The measurement is quick and convenient but the results of simple equipments (with one frequency, two electrodes and without calibration) are rather imprecise but useful for "home monitoring" of people on weight normalization regimens. More sophisticated equipments of this type provide more exact results.

For research purposes **Dual energy X-ray absorptiometry (DEXA), computer tomography, magnetic resonance, ^{40}K isotope measurements** are employed but these are expensive and time consuming.

The normal amount of fat in human body is variable. A number of factors, including age, sex, environmental temperature, and the degree of physical activity determine the relative proportion of fat in the body. By age 18, the male body contains about 15 to 18% fat, and the female about 20 to 25%. Acceptable body fat content in the adult general population is 10–20% in men and 20–30% in women [9].

Although both the basic (increased fat content) and the numerical definition (BMI, proportion of fat) of obesity are correct, there are some open questions concerning a rational, patophysiology-based definition of obesity. The main question is if obesity is a disease or not? According to our view [10] obesity (with the exception of some monogenic, endocrine and hypothalamic diseases with morbid obesity) itself is not a disease, but

— a pathological condition caused by disturbance in appetite regulation and by positive energetic balance;
— a risk factor of many serious diseases and
— a phenotype based on combination of permissive genetic and triggering external factors.

Another good definition was presented at the congress of North American Society for the Study of Obesity [11]: where obesity was characterized as a **gateway disease** opening the door for type 2 diabetes mellitus, hypertension, coronary artery disease, and many other diseases.

3. The epidemiology of obesity

In 2008 an estimated 205 million men and 297 million women over the age of 20 were obese worldwide and the prevalence of obesity has nearly doubled between 1980 and 2008. The prevalence of overweight and obesity were highest in the regions of the Americas (36% overweight[1] and 26% obesity) In Europe the situation is better than the USA but notwithstanding serious and even more alarming are the trends and perspectives [12]. The average percentage of obese adult persons in Europe (2010) was around 17 % with significant differences among countries and in some regions between men and women. Some of these data are based on not very exact self-reports (e.g. Romania with only 8,9% prevalence of obesity). The data derived from measurements in epidemiological studies are more reliable and the worst situation is probably in Hungary (28,5%). A special situation is that of school-aged children. In the age group of 15 years old boys and girls the prevalence of obesity is already as high as 17 and 10%, respectively. These young persons should live with obesity (if they are not able to rid themselves of excess fat) the whole active period of life and suffer from its consequences around the age of 40.

Obesity is considered as a disease of affluence and this is true but on the other side the highest prevalence and incidence of obesity in high and middle-income countries is in the group of population with low level of education and income [3].

4. The distribution of the fat in the body

The pattern of fat distribution is a more important factor of morbidity and mortality associated with obesity than obesity per se. Fat distribution can be assessed simply from **waist to hip ratio (WHR)**, i.e. the ratio of circumferential measurement at the waist (midway between the iliac crest and lowest rib) to that around the hips (at the level of trochanters). In most populations normal WHR is distributed within the range 0,7 – 1,0. A WHR greater than 1.0 in men and 0.8 in women indicates upper body obesity.

Recently this relative value is replaced by the **waist circumference** and the normal values are as follows: less than 94 cm in men and less than 80 cm in women. According to our view body mass and waist circumference measurements should be standard procedures at each visit of patients and also of healthy persons at general practitioners' outpatient clinics.

According to fat distribution two basic types of obesity exist:

[1] In the source there is 62% overweight and 26% obesity, which can lead to false interpretation. We consider "overweight" as BMI between 25–30 and obesity as BMI > 30 kg/m^2.

1. upper body obesity, also referred to as abdominal, android or male obesity, and
2. **lower body obesity,** also known as **gluteofemoral, gynoid or female** obesity.

J.Vague was the first who in 1947 envisioned the importance of regional adipose tissue distribution as the most significant factor of the metabolic complications of obesity. He suggested that android obesity was associated with **accelerated atherosclerosis, diabetes mellitus type 2 and gout**. Vague's pioneering contribution has been confirmed by countless experimental, clinical and epidemiological studies and the condition caused by android obesity has been termed as **"X-syndrome", "metabolic syndrome"** or simply an correctly as "**insulin resistance syndrome"**. Excellent reviews on this topic are in [13, 14] and a more detailed description in Chapter 8.

Computed tomography and the sophisticated methods described in chapter 2 allow exact measurement of visceral and subcutaneous adipose tissue amount at any site of the body and particularly in the abdominal area. Based on the precise estimation of the topography of the adipose tissue, C. Bouchard in 1991 [15] proposed four different types of human obesity:

1. **The first type** is characterized by excess total body mass or body fat without any particular regional difference.
2. **The second type** is defined as excess subcutaneous fat on the trunk, particularly in the abdominal area, and is equivalent to the **android or male** type of fat deposition.
3. **The third type** is characterized by excessive amount of fat in the abdominal visceral area and can be termed as **abdominal visceral obesity**.
4. **The fourth type** is defined as **gluteofemoral** obesity and is observed primarily in women (**gynoid obesity**).

Although this classification is more scientific as compared with the simple upper/lower classes, in public health care and general medical practice it is quite sufficient to use the simple classification because upper body obesity is always associated with visceral obesity. One should keep in mind that mild lower body obesity in young women is a positive feature of fertility but male type (abdominal + visceral) obesity is common also in women and has similar consequences as in men.

Recently modern methods allow also the estimation of fat content in different cells and the according to the results [16] the accumulation of fat outside adipocytes, namely in liver and muscle cells is also an important contributor of the consequences of obesity.

5. The growth of adipose tissue

The growth of adipose in the course of positive energy balance can occur by an increase in the **number of adipocytes (hyperplasia)** and/or by an increase in

the **size of fat cells (hypertrophy**). Although adipocyte number is partly determined in the early stages of life, the number of adipocytes formed from precursor cells (preadipocytes), may increase during later life, too.

The size of adipocytes responds to the balance between storage and release of triacylglycerols (TAG). Increased food intake and increased insulin secretion will favor lipogenesis; increased exercise and increased secretion of the lipolytic hormones (the catecholamines as the most potent regulators of lipolysis, as well as glucagon, growth hormone and ACTH) will promote fat utilization. That means that hypertrophic obesity is dynamic and reversible.

As the life span of the fat cells is very long, their size varies with the amount of stored TAG, but there is little or no change in their total number. It means that subjects with hyperplastic obesity have a lifelong increase in the total mass of fat cells and any weight loss occurs is by shrinkage of adipocytes without a decrease in their number.

Men and women with abdominal and visceral obesity (types 2 and 3) are characterized both by hyperplasia and by large fat cells. The cells have increased lipoprotein lipase (a key regulator of fat accumulation) activity, enhanced lipolysis, and low antilipolytic effect of insulin. In contrast, lower body fat deposition is the result of differentiation of fat cells from precursors (hyperplasia). This may explain the weight loss difficulties of many women with lower body obesity.

6. The etiology of obesity as viewed from the point of view of energy metabolism

Excess body fat is generated when the energy intake exceeds energy output. Output comprises of **basal metabolic rate** (usually about 75% of total energy expenditure), of **physical activity** (10–15%), and of **thermogenic response to food and cold temperature** (10–15%). The laws of physics are valid also in human physiology and **without positive energy balance the development of obesity is not possible.** Although this is true, there are at least three caveats in energy balance:

1. The ability to convert some portion of energy into heat (through mitochondrial uncoupling proteins) is strongly age-dependent. The same amount of ingested food in young people does not lead to weight gain but can cause obesity in middle aged and older adults regardless on their physical activity. Individuals with elevated function of thyroid maintain the ability to convert food-derived energy into heat also in higher age.
2. Weight gain is a slow process and already an imbalance of only 1% in energy balance represents weight gain of 1 kg every year.
3. The individual difference of basal metabolic rate is as high as 25–30%. If a difference in metabolic rate can be as high, it is clear that at a same energy

intake one individual may gain weight and another may lose it regardless on other ways of energy output.

Adipose tissue contains about 75% of fat, which is the main form of energy store. Overweight subjects store approximately 30 000 kJ (7000 kcal) energy per kilogram excess weight. Factors contributing to the energy imbalance are numerous and probably exist in different combinations among obese individuals.

Energy is required for muscular contraction connected with basic life processes and various forms of involuntary and voluntary **physical activity**. The efficiency of muscular work (the proportion of exercise-produced energy that is used for work) is about 30%, and is the same for lean and obese subjects, but obese subjects are usually less physically active as compared with lean people.

The expenditure of energy after usual food intake (former called specific dynamic action of food) and food-induced **thermogenic response** are relatively small and the differences between lean and obese individuals are small to nonexistent. However there are differences in energy expenditure after **overfeeding.** In lean people overfeeding (500 kJ extra calories daily) for 10 days or longer led to energy wastage, but no energy wastage has been found in already obese volunteers [17].

7. The genetic and epigenetic background of obesity and their association with the external factors

Variations among individuals in basal metabolic rate and fat accumulation after overfeeding are undoubtedly influenced by the **genotype**. This effect seems to be even more important for the determination of the sites of fat accumulation. The first genetic hypothesis termed as the "**thrifty genotype**" was based on the presumption that in the past starvation and famine was common and the survivors were individuals with "thrifty" genes able to cumulate as much energy from the meager sources as possible. 10–20 thousand years is a too short period to transform the genotype and in the XXth – XXIst individuals with „squandering" genotype are those able to remain lean and healthy. According to last achievements in prehistorical anthropology the validity of this hypothesis is questionable because our most of our ancestors did not die from undernutrition but from infections and wounds.

The role of **genetic** and **epigenetic factors** in the development of obesity has been supported by experiments on animals, as well as by studies on body weight in families of obese persons, studies of twins, of adopted children and their adoptees [18, 19]. The first actual gene involved in appetite and food intake regulation secreted from adipose tissue was **leptin**, a small protein coded on the chromosome 7 [20]. If the amount of fat in adipose tissue begins to increase, more leptin is secreted into the blood, binds to its receptor in the brain and in

healthy individuals decreases the appetite. After the discovery of the leptin and its mutated gene in mice the great expectations as to its role in human obesity was a disappointment because the mutations of leptin coding OB gene in humans are extremely rare.

Recently the number of genes involved in appetite regulation and energy metabolism is around 400. This makes the interpretation of genetic polymorphism in the pathogenesis of obesity extremely difficult but one thing is clear. In the human body there is no "**obesity gene**". All obesity-related genes are normal genes responsible for energy metabolism and energy metabolism is the basis of life. All polymorphisms and mutations of these genes are only **permissive** and without increased food intake and decreased output they do not make obesity [21].

Environmental influences are as important for the development of obesity as the genetic and epigenetic background. The difference in individuals lies only in the different speed of fat accumulation as a result of overeating and in the different ability to lose weight on a low-energy diet and in connection with increased physical activity. Some of the common sins promoting fat accumulation are as follows:

— **infant milk formulas instead weaning;**
— **eating huge amount of food once a day instead of 3–4 small servings;**
— **couch potato life-style;**
— **nibbling;**
— **fast & junk food;**
— **drinking too much sweetened beverage and beer;**
— **night eating, binge eating and many others.**

The social and psychological factors of increased food intake are summarized in Chapter 10.

Endocrine disorders and **hypothalamic diseases** are rare causes of obesity as well as the genetic **Prader-Willi syndrome**. In these cases the obesity is rather morbid (BMI > 40 $kg.m^2$) but always connected with overeating. Voluntary control of food intake in these patients is not working.

8. Health risks and diseases associated with obesity

Impaired social relationships, loss of self-esteem, anxiety, anger, self-doubt, etc. are not the only problems of obese individuals [22]. Since the times of Hippocrates it has been well known that obesity is associated with premature death, and since Vague it has been obvious that upper body fat accumulation (together with increased visceral and intracellular adiposity) has significantly worse prognosis than lower body fatness.

The risk of some diseases associated with obesity is summarized in Table 2. The causes of increased mortality of obese individuals include coronary heart

disease, cerebral hemorrhage, diabetes, digestive diseases, and cancer [23]. On the other side almost all health disorders associated with obesity improve after weight loss (24Hall et al., 2014).

Table 2. Increased risk of diseases in abdominal obesity with BMI > 27 kg/m^2

Disease	Relative risk*
Stroke	3,1
Hypertension	2,9
Type 2 diabetes mellitus	2,9
Myocardial infarction	1,9
Gout	2,5
Gallbladder disease**	2,0
Arthrosis	1,8
Malignancies (breast, colon)	1,2 – 1,3

* Compared to the risk of age-adjusted people with normal body weight.
** Common also in gynoid obesity.

The main pathogenetic factor behind most of the diseases caused by obesity is the **metabolic syndrome** described in 1988 by Reaven, which in addition to obesity includes

- **hyperinsulinemia and insulin resistance,**
- **impaired glucose tolerance or non-insulin dependent diabetes mellitus,**
- **dyslipidemia with elevated levels of triacylglycerols and decreased levels of HDL-cholesterol,**
- **disorder of uric acid metabolism,**
- **hypertension,**
- **accelerated atherosclerosis and its clinical consequences**
- **subclinical inflammation**
- **increased tendency to coagulation**
- **polycystic ovaries**

Although significant associations have been found between indices of abdominal obesity, insulin resistance and dyslipoproteinemia, the mechanisms underlying these interrelationships are not fully understood [25].

8. Treatment of obesity

The treatment of obese persons is one of the most troublesome and frustrating enterprises, particularly from the long-range standpoint, because the maintenance of a reduced body weight is a very difficult problem. The "success" of treatment may depend on the severity of obesity and the amount of

hypercellularity of the adipocytes in a given person. An individual who is modestly overweight with enlarged fat cells but little proliferation of extra adipocytes can more easily maintain weight loss. The adipocyte hyperplasia is a much greater problem in the maintenance of weight loss.

There is no single effective treatment for obesity [26, 27] Treatment methods include **nutritionally adequate weight loss diet, behavior modification, exercise, social support, pharmacological intervention** and in situations of marked obesity **surgical methods** [28].

9. The psychology of overeating and obesity

Not very long ago there was no big difference between the access to food between human beings and animals – it was connected with physical activity although humankind developed agriculture and animal farming instead of prey hunting and grazing. Hunger and famine was common also in Europe until the XIXth century.

In the past century it was a profound change in this situation. Food in countries with normal economic situation is easily accessible and relatively cheap. In the vast majority of population the access to food is not connected to physical work. According to our view and [28], the main factors attacking our senses to eat more than is necessary and at the same time decreasing our physical activity are as follows:

— Surplus of food in hyper- and supermarkets, together with their global advertisement and marketing policy;
— Sitting all day and watching television (advertisement and marketing once more) and/or peering on computer display instead of moving;
— Going to nearest corner by car instead of going anywhere on foot or cycling;
— Coca-colonization and Mcdonaldisation (pure energy without postprandial output);
— Full deep-freezers at home.

The result is that in the last 50 years there was a huge increase of per capita consumption of meat, fat, sweets and sugar-containing beverages with a similar decrease of physical activity.

From personal side of overeating, there is also an interesting classification of our eating habits [29]

Tranquil eating. A normal form of eating. It begins as weaning from the first hours of life and fulfills not only the nutrient needs of the baby but functions also as a very strong and important bodily and mental connection with the mother and the environment. Although later the human nutrition is more variable and not based on the bodily contact with the mother, this type of eating is the best prerequisite of normal growth of development of youngsters and in adult life the prevention of eating disorders including obesity.

Dominant eating. In the history of mankind the long periods of starvation were interrupted by occasional feasts (after successful hunts, and later – social and religious events). The head of the community was the first to take the best chomps of the goods. His big body was also a clear sign of his dominant position. This type of eating is common in animal kingdom, but in our society its role is clearly distorted. In our region, it is an outright discourtesy if the guests refuse the food offered by the host (and an impoliteness of the host not offering food immediately after the arrival of visitors).

Defensive eating. Not very common in our society, but it can occur in groups after prolonged starvation. When served enough food, people (usually children) eat all the food served on the table, because of the uncertainty about the future access to food.

Aggressive eating. Not very common, but if present, this is the first step to **bulimia**. However, its less pronounced form, the **stress eating** is very common. Most people in stress situations (e.g. before examination, having family and community problems) consume more food than is necessary, and although they usually realize that it is not healthy, they cannot stop it. In our opinion, this type of overeating is probably the basic factor of common obesity in our society.

The complex psychology of overeating is as important in the pathogenesis of obesity as its genetic and epigenetic background. However, there is a basic difference between them. Our genes are inherited and (relatively) stable. On the contrary, appetite and human eating habits are under control of the brain and therefore are amenable to reasonable arguments and education [30].

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Streszczenie

Otyłość – pandemia XI wieku

Tragiczną ironią współczesnej cywilizacji jest sytuacja, w której jedna trzecia światowej populacji (osoby żyjące w krajach słabo rozwiniętych gospodarczo) jest zagrożona niedożywieniem z powodu braku pożywienia, a równocześnie ludność zamieszkująca bogate kraje jest otyła w związku z nadmiernym spożyciem pokarmów.

Autorzy w pierwszej części definiują i klasyfikują otyłość jako problem medyczny oraz analizują dane epidemiologiczne dotyczące występowania otyłości w Europie Środkowej. Główna część pracy zawiera krótki przegląd zewnętrznych, a także genetyczych i epigenetycznych czynników rozwoju otyłości, której zasadniczą przyczyną powstawnia są zaburzenia regulacji apetytu na poziomie mózgowia. W związku z tym, genetyczne, epigenetyczne oraz metaboliczne czynniki związane z otyłością można uznać za wyzwalające w patogenezie choroby. W ostatniej części artykułu opisano krążeniowe i metaboliczne skutki i powikłania otyłości oraz możliwe sposoby jej zapobiegania i leczenia.

Słowa kluczowe: otyłość, nadmierne przyjmownie pokarmow, powikłania otyłości, profilaktyka otyłości, leczenie otyłości.