Obesity Biologycal and Psychological Aspect

Saida Fišeković*

Department of Psychiatry, University of Sarajevo Medical Center,
Bolnička 25, 71000 Sarajevo, Bosnia and Herzegovina

* Corresponding author

Abstract

There is no unique pattern to deal with obesity unless is presented as complex of biological and psychological factors. A lot of studies deal with only one side of it. This work shows both sides and discuses about all relevant factors, which are involved in pathogenesis of obesity. This is only way for finding better approaches for treatment and understandings for this issue.

KEY WORDS: obesity, BMI, Obesity and depression, NES, BED, leptin

Introduction

By ICD – 10 obesity is included and defined as general health disorder. Obesity is a disorder of body weight regulatory systems characterized by an accumulation of excess body fat (1). The prevalence of obesity increases with age and is more common among poorer persons and in individuals with only high school education or less. Risk factors for obesity are:

- Genetic factors
- Food intake
- Bad life behaviors
- Energy expenditure
- Behavioral and psychological factors

Assesment of Obesity

The amount of body fat is difficult to measure directly, and is usually determined from an indirect measure – the body mass index (BMI) that has been shown to correlate with the amount of body fat in most individuals (1).
BMI = (weight in kg) / (height in m^2)
BMI gives a measure of relative weight, adjusted for height. Healthy range for BMI is 19,5- 25,0, between 25 and 29,9 are considered overweight, and equal or greater than 30 are defined as obesity (2).

Anatomic and Biochemical Differences in Fat Deposition

The anatomic distribution of body fat has major influence on associated health risks. There are two types of fat distribution recognized as: Apple – shaped or upper body obesity, and is associated with a greater risk for hypertension, dyslipidemia, diabetes, insulin resistance and coronary heart diseases, and second Peer shaped or lower body obesity which is defined as relatively benign health wise, and is common in females. Abdominal fat cells are much larger and have a higher rate of fat turnovers than do lower body fat cells. The abdominal adipocytes are also hormonally more responsive than fat cells in legs and buttocks. Substances released from abdominal fat are absorbed via portal vein so have direct access to the liver. By contrast, free fatty acids from gluteal fat enter the general circulation, had had no preferential action on hepatic metabolism (Figure 1).

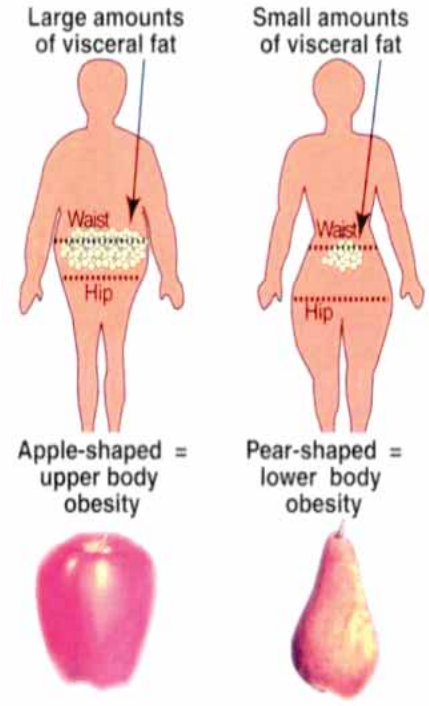

FIGURE 1. Anatomic differences in fat deposition

Set Point Theory

The observation prompted the theory that each individual has a biologically predetermined "set point" for body weight. The body attempts to add adipose tissue when the body weight falls below the set point, and to lose weight when the body weight is higher then the set point. This point is biological balance between factors influence food intake and energy expenditure, so body weight is stable as long as the behavioral and environmental factors that influence energy balance are constant.

GENETIC CONTRIBUTIONS TO OBESITY
Obesity behaves as complex polygenic disease involving interactions between multiple genes and environment. Classic twin studies have estimated high levels of hereditability of body weight, the percent of variance accounted for is about 80% (3). Even a study of identical twins separated at birth, a method that avoids the bias in classic twin studies, estimated hereditability at nearly this level (4). Environmental factors clearly influence the development of obesity, as shown by the powerful influence of social class and, strikingly, by the rapid, epidemic increase in obesity in recent years (5). These influences include the consumption of calorically dense, highly palatable foods and a deficit in physical activity (6).

MOLECULES THAT INFLUENCE OBESITY
Afferent, or incoming neural signals, circulating hormones, and metabolites- that impinge on hypothalamus influence appetite. This diverse signals prompt release of hypothalamic peptides, and activate outgoing efferent signals (Figures 2,3).

HORMONES OF ADIPOSE TISSUE:

- leptin (is secreted by fat cells and act on hypothalamus to regulate the amount of body fat through control of appetite and energy expenditure).
- adiponectin and resistin (may mediate insulin resistance observed in obesity)

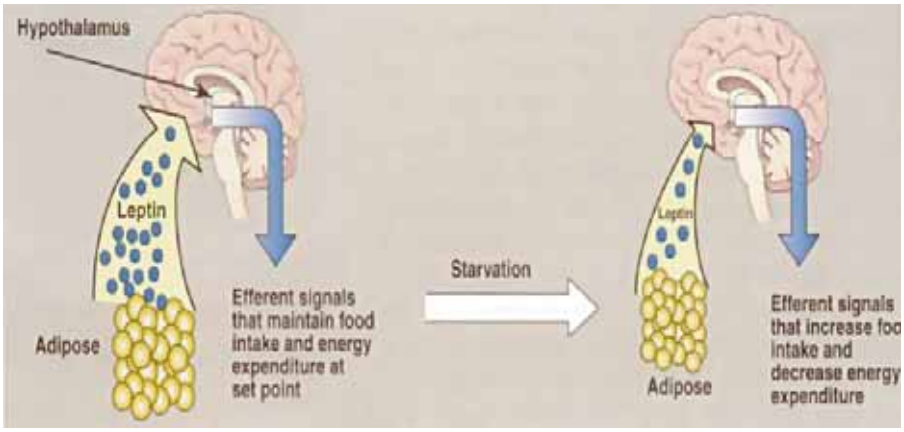

FIGURE 2. Action of leptin in maintaining adequate fat stores

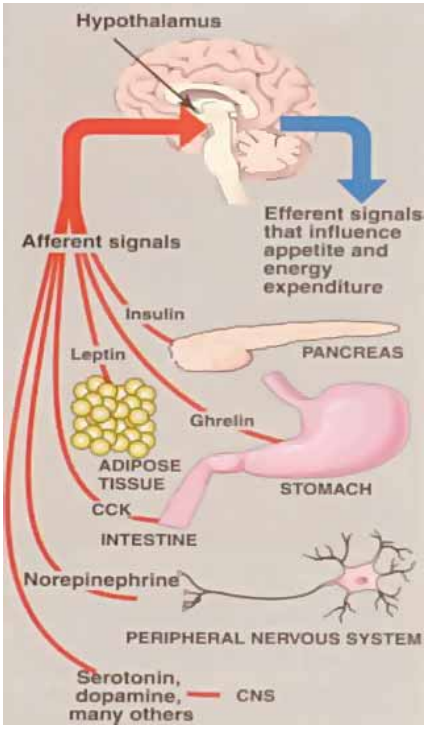

FIGURE 3. Afferent signals which reflect nutritional state of the body

Other hormones :

- ghrelin (secreted by stomach is the only known as appetite – stimulating hormone)
- CCK (cholecystokinin released from gut can act as satiety signals to the brain)
- insulin (not only influence metabolism, but also promotes decreased energy intake)

OBESITY AND HEALTH

Obesity is correlated with increased risk of death, and is risk factor for a number of chronic conditions including: diabetes, hyperholesterolemia, high plasma triacylglycerols, hypertensionh, heart disease, some cancers, gallstones, arthritis, and gout. Abdominal obesity is highly associated with syndrome X known as metabolic syndrome (glucose intolerance, insulin resistance, hyperinsulinemia, dyslipidemia – low HDL, high VLDL, and hypertension). The relationship between obesity and associated morbidities is stronger among individuals younger than 55 years. After age 74, there is no longer an association between increased BMI and mortality (Figures 4,5).

Psychological Aspect

At least one prospective study has shown that the presence of clinical depression predicts the development of obesity (7). Major depression among children and adolescents ages 6 to 17 was found to predict a greater BMI in adult life than that of adults who had not been depressed as children or adolescents (BMI of 26.1 versus 24.2, respectively). In a study of the predictors of weight change occurring during unipolar depression, Stunkard et al. assessed 53 unmedicated outpatients across two distinct episodes of severe depression. There were at least two notable findings from this study. First, there was a high correspondence in the direction of weight change across the two episodes. Thus, patients who had gained weight during the first depressive episode tended to gain weight during their second

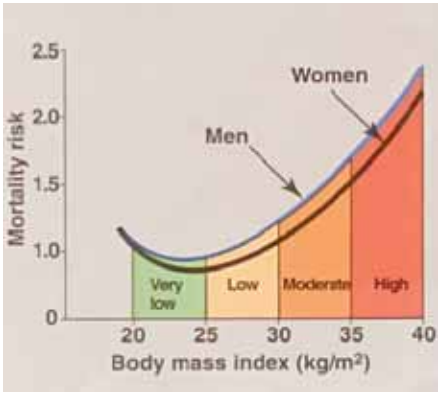

FIGURE 4. Body mass index and the relative risk of death

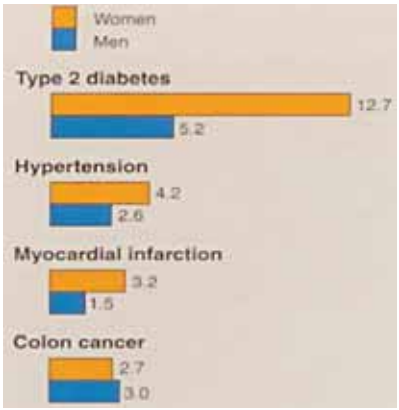

FIGURE 5. The relative risk of developing associated diseases in obese womwn and men compared with non -obese individuals in which the risk = 1.0

episode, whereas patients who had lost weight during the first depressive episode tended to lose weight again during their second episode. Second, BMI was positively associated with weight change during depressive episodes, such that heavier individuals tended to gain more weight (r=0,30, p<0,05), thereby suggesting that the association between obesity and depression may depend on the patient's initial weight status (8). Several studies report that the relationship between obesity and depression differs for men and women. Istvan et al., for example, showed a positive relationship between depression and obesity among women but not among men (9). Similarly, Faith et al. found a positive relationship between neuroticism and BMI in women but not in men (10).

Disordered Eating

Disordered eating may mediate the relationship between depression and obesity. The experience of binge eating (and its associated feelings of uncontrollable eating) may promote depression. From its first descriptions, BED has been strongly associated with depression (11,12,13). Among a group of obese people, 51% of those with BED had a history of major depressive disorder (MDD) compared to 14% of those without BED. Sherwood et al. showed that improvements in binge-eating status predicted greater weight loss in treatment through a pathway that involved a decrease in depression. (14). Night-eating syndrome (NES) (morning anorexia, evening hyperphagia, insomnia and nighttime awakenings to eat), may place people at increased risk for depression.(15) A particularly interesting characteristic of the depression exhibited by people with NES is its distinctive circadian quality. Thus, among these patients, depressive mood is minimal in the morning, rises during the afternoon and evening, and reaches its peak late at night in conjunction with the most intense hyperphagia. Finally, it should be noted that cognitive-behavioral therapies for depression are effective for many people (16).

Conclusion

Obesity is increasing in industrialized countries because of a reduction in daily energy expenditure, and an increase in energy intake. The weight is determined by genetic and environmental factors. Obesity is correlated with increased risk of death, and is a risk factor for a number of chronic conditions. Correlation between obesity and psychiatric disease still is not included in DSM- IV, but a lot of recent studies suggested that there are significant connection between some eating disorders, depression and obesity. Newly discovered eating disorders such as NES and BED confirm it. All new approaches for resolving this issue includes varies of psychotherapy, such as cognitive, behavioral, sociotherapy, help groups.

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