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## Obesity and mental health

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## **Abstract**

**Introduction:** Obesity is defined as having an excess of body weight caused by a chronic caloric imbalance with more calories being consumed than expended each day. There are over 2.1 billion individuals with obesity around the world. Body mass index (BMI), which is weight in kilograms divided by height in meters squared, is used to identify individuals with obesity. For adults, a BMI of 25.0 to 29.9 kg/m<sup>2</sup> is defined as overweight and a BMI of 30 kg/m<sup>2</sup> or higher is defined as obese.

**The aim of the study:** The purpose of this systemic review was to collect and analyse current and new information on obesity, its impact on mental health and vice versa.

**Material and method:** Standard criteria were used to review the literature data. The search of articles in the PubMed database was carried out using the following keywords: obesity, mental health, diet, depression, anxiety.

**Description of the state of knowledge:** There are numerous studies about obesity and other factors. Obesity can be the cause of depression, often due to stigma and discrimination, however depression could be also a reason of development of obesity. There was found bidirectional relationship – obesity increased the risk of depression by 55% and depression increased the risk of obesity by 58%. Studies show that anxiety or post-traumatic stress disorder (PTSD) may be associated with obesity and considered as its risk factor. Another disorders, such as Seasonal Affective Disorder (SAD) and Premenstrual Syndrome (PMS) may also cause obesity.

**Summary:** Obesity has complicated and multifactorial etiology. Discovered dependencies and conducted research will allow to find new therapeutic paths and isolate people at risk. However more research is needed to fully understand problem of obesity and all its dependencies.

**Key words:** obesity, mental health, diet, depression, anxiety

## **1. Introduction**

Obesity is defined as having an excess of body weight caused by a chronic caloric imbalance with more calories being consumed than expended each day [1]. Body mass index (BMI), which is weight in kilograms divided by height in meters squared, is used to identify individuals with obesity. For adults, a BMI of 25.0 to 29.9 kg/m<sup>2</sup> is defined as overweight and a BMI of 30 kg/m<sup>2</sup> or higher is defined as obese [2]. BMI over 25 kg/m<sup>2</sup> increases:

- overall mortality increases by 29%
- vascular mortality by 41%
- and diabetes-related mortality by 210% [3,4]

Obesity is often associated with mental health problems. Women with obesity report worse mental health than women without obesity [5]. In addition obese people often experience stigma and discrimination, which can have numerous consequences for their psychological and physical health [6]. Studies have documented many weight-based stereotypes about obese people, such as obese individuals are lazy, weak-willed, unsuccessful, unintelligent, lack self-discipline, have poor willpower [7]. Therefore stigma and discrimination could deepen obesity and induce mental health problems in obese individuals. On the other hand there are evidence that obesity could be also induced by already existence mental health problems such as depression [8].

## **2. Depression, related mood disorders and obesity**

Coronary Artery Risk Development in Young Adults acknowledge that depression can be a risk factor for obesity. In 2010 Needham, et al. found that people who were depressed gained weight significantly faster than people without depression symptoms. Moreover, depression symptoms were positively correlated with a subsequent increase in waist circumference [9]. Depression in adolescents can be a predictor of obesity. The National Longitudinal Study of Adolescent Health found that depressed mood at baseline significantly predicted obesity 1 year later among those who were of normal weight at baseline [10]. Another longitudinal cohort study about the relationship between adolescent depression and adult obesity conducted by Richardson LP, et al. showed that depression in late adolescence is associated with later obesity, but only among girls [11]. Major depression occurred in 7% of the cohort during early adolescence (11, 13, and 15 years of age) and 27% during late adolescence (18 and 21 years of age). At 26 years of age, 12% of study members were obese [11]. There was found bidirectional relationship – obesity increased the risk of depression by 55% and depression increased the risk of obesity by 58% [12].

## **3. Anxiety, post-traumatic stress syndromes and obesity**

There are several studies which show that anxiety or post-traumatic stress disorder (PTSD) may be associated with obesity and considered as its risk factor [13,14,15]. Study on 169 woman conducted by Pinaquy S, et al. suggests the involvement of alexithymia in eating disorders among obese women [13]. Another study was carried out using a questionnaire by Geliebter A and Aversa A. There were 90 questionnaires – 30 for underweight, 30 for normal weight and 30 for overweight. The results show that when experiencing negative emotional states or situations, overweight individuals eat more than normal-weight and underweight individuals. However, for positive emotional states the underweight individuals eat more than others [14]. Silva I in 2015 made a comprehensive review of eating behaviour and emotional states. According to this, we can found that anxiety and such other dysphoric mood states as anger, sadness, and stress are often coupled to obesity [16]. Another example for association between anxiety and obesity is seen in binge eating disorder. In this disorder huge amount of food are ingested over short periods of time by individuals who have anxiety or have experienced negative emotional stress or situations [17].

PTSD may have implications for the development of obesity-related diseases [18,19,20]. Study on US adults conducted by Pagoto SL shows that among individuals with past year PTSD nearly one-third (32.6%) were obese, compared to about one-quarter of individuals with history of PTSD (25.5%) and no history of PTSD (24.1%) [20]. In this study PTSD was associated with binge eating disorder (BED), BED did not lessen the association between PTSD and obesity. PTSD may influence body weight by psychological or biological pathways:

- PTSD is associated with alterations in the functioning of hypothalamic-pituitary-adrenocortical axis – this regulates the secretion of corticotropin-releasing hormone and the glucocorticoid hormone cortisol. Hypersecretion of cortisol, has been hypothesized to promote obesity
- Overeating increases the experience of positive emotions
- Overeating could be seen as an attempt to misattribute perceived stress to eating, in order to distract from the original source of stress [21,22].

In 2010 Pervanidou P. shows that individuals with PTSD have lower circulating cortisol levels and elevated basal cerebrospinal fluid corticotropin-releasing hormone concentrations relative to healthy controls [23]. Another study conducted by Lagarde G shows disruptions of inhibitory control in individuals with PTSD – they perform more poorly on behavioural tasks which measure the capacity to suppress inappropriate behavioural responses [24]. Inhibitory control seems to be critical to suppressing food intake [25]. There are evidences for association between impulsivity and deficits in inhibitory control which lead to obesity and poorer weight loss outcomes in behavioural treatment programs [26].

#### **4. Seasonal Affective Disorder (SAD) and Premenstrual Syndrome (PMS)**

Seasonal Affective Disorder is atypical form of depression, that occurs in November-December and is characterized by increased food intake, which leads to weight gain [27,28]. Study conducted by Krauchi and Wirz-Justice whose compared patients with SAD and healthy patients, shows that patients with SAD consumed more starchy or sweet carbohydrate-rich foods than healthy individuals. However, individuals with SAD decreased their food intake during remission [29]. Patients with SAD claim that eating carbohydrate-rich foods, their depression symptoms are lower [29]. Weight gain mechanism during Premenstrual Syndrome seems to be the same. Women with PMS eat more carbohydrate-rich foods and when their moods return to normal – the consumption of carbohydrate-rich foods returns to normal. Consuming carbohydrates increases synthesis of serotonin. It is known that serotonin is involved in mood and appetite and overconsume carbohydrates may be an attempt to improve their dysphoric mood state [30,31]. The effect of increased synthesis of serotonin does not occur after ingestion of protein [31]. However there are also studies which suggest that maintaining a healthy body weight may be important for preventing the development of PMS. It follows that PMS can be the cause and sometimes the result of obesity and an unhealthy lifestyle [32].

#### **5. Genetics and obesity**

There are numerous polymorphic gene products may also be a cause of obesity. Study conducted by Li, et al shows that 12 obesity-susceptible loci have been identified. There is association between these loci and BMI, waist circumference, weight, and height, as well as the predictive value for obesity risk [33]. In one cohort study of adult twins who were reared apart compared with a control group of twins reared together showed that body fat is strongly associated with genetic factors [34]. Another study of monozygotic and dizygotic twins suggest that adult body size, shape, and composition are highly heritable in both women and men, although a decreasing tendency is seen with advancing age [35].

#### **Summary**

All these findings show that obesity is an disorder with complicated etiology and can be caused by many factors. It can be a cause but also an effect of many of them. Further research may bring us new possibilities of therapy, earlier identification of people at risk of obesity or depression. Around the world 2.1 billion individuals are considered overweight or obese and due to the increase in obesity, more research is needed to understand the cause and to increase therapeutic options.

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