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IMPACT OF ADIPOSE TISSUE FAT IN BINGE EATING

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***Abstract*:** Binge eating is characterized as frequent episodes of consuming massive amounts of food in a short period of time. Obesity is thought to be a factor in binge eating. Obesity is a medical condition defined as excess body fat that causes a variety of health problems and it is frequently diagnosed with body mass index (BMI). However, BMI does not provide information about body fat percentage, bone mass, body cell mass, or body fluids. Adipose tissue is the most common type of fat storage tissue in the body. The main goal of this review is to determine how adipose tissue fat varies with binge eating. To discover that it is essential to find out how specific substances are derived from fat cells, such as leptin, and adiponectin. TNF α and IL-6 are linked to binge eating.

Index Terms: **Binge eating, Leptin, Adiponectin, TNF α, IL-6, Adiposopathy**

INTRODUCTION:

Eating disorders are defined as abnormal eating patterns that significantly interfere with a person's daily life (Kim, 2012). Eating disorders are complicated and serious mental health issues that can have serious consequences for a person's physical and mental health. Eating disorders are psychiatric conditions because they affect an individual's thoughts, feelings, and actions regarding food and eating (Singh, 2014). Eating disorders are more than just a person's food relationship. Eating disorders include anorexia nervosa, bulimia nervosa, binge eating disorder, night eating syndrome, pica, rumination disorder, orthorexia nervosa, and avoidant/restrictive food intake disorder (Zealand, 2019) These types can affect people of different ages, genders, and origins.

People believe that obesity contributes to eating disorders. Obesity, on the other hand, is a medical disease defined by excess body fat that can increase the risk of a number of health issues. Obesity is often diagnosed using the body mass index. People believe that having a high BMI is a health concern and that having a normal BMI is good, although this is not necessarily true. For example, an athlete with a high muscle mass may have a high BMI yet a low body fat percentage (Walls *et al.*, 2011). BMI does not give the idea about body fat percentage, bone mass, body cell mass, or body fluid. To understand how obesity affects eating disorders, it is critical to understand what role fat plays in mental disorders and how fat modifies the brain to cause psychiatric diseases such as eating disorders. To find that adiposopathy is important.

Adiposopathy is a new topic in medicine that focuses on changes in adipose tissue fat due to disorders (De Lorenzo *et al.*, 2016). Adiposopathy is also important to consider in the context of obesity because excessive adipose tissue expansion is a key factor in developing adiposopathy. Between eating disorders binge eating disorder is a unique eating disorder since most eating disorders affect obese or overweight people, but with binge eating disorder, it is possible to have normal or even underweight body weight as well as obese or overweight body weight (da Luz *et al.*, 2018).

Because of that, it makes it easier to discover how fat changes occur to produce mental disorders such as binge eating disorder to help with adiposopathy

Binge eating disorder is defined as recurring bouts of consuming enormous quantities of food in a short period of time (Zam, Saijari, and Sijari, 2018). This conduct can cause a feeling of shame, remorse, and sadness, as well as have harmful effects on physical and emotional health (Gagnon *et al.*, 2018). Overeating, struggling with body dysmorphia, utilizing the bathroom, or bathing after meals to vomit food after eating are all classic symptoms of binge eating disorder (Reichenberger *et al.*, 2021). A history of trauma or abuse may be present in a binge eating disorder. So, binge eating disorder is a sensitive topic. The main purpose of this review is to determine the potential role of adiposopathy in binge eating disorder.

3. Adipose tissue-derived molecules relationship for adiposopathy to binge eating disorder.

Adipose tissue is a form of connective tissue found all over the body. It is also referred to as body fat. Fat cells, immune cells, endothelium, fibroblasts, neurons, and stem cells are primarily found in adipose tissue (Cohen, Spiegelman and Drubin, 2016). Adiposopathy is typically defined as adipose tissue dysfunction. It is critical to understand how these adipose tissue components alter in order to examine the association between adiposopathy and binge eating disorder.

The structural unit of adipose tissue is the adipocyte [fat cell], which can be split into white, brown, and beige adipocyte (Gonzalez-campoy *et al.*, 2014). White adipocyte release adipocytokines. Adipocytokines are active substances generated from white adipose tissue. One type of adipocytokine is leptin (Mart, 2020). Leptin is a peptide generated from white adipose tissue. Lepin was identified in 1990 as a longer-term adiposity signal secreted in proportion to body fat reserves (Gale, Castracane and Mantzoros, 2004). Scientific research has demonstrated that leptin has a significant impact on apatite (Adamo *et al.*, 2014). The main apparent symptom of a binge eating disorder is a voracious hunger for food. As a result, there must be a connection.

New findings indicate that leptin can also influence neuroendocrine axes, the autonomic nervous system, brain plasticity, and memory (Havel, 2000). The hypothalamus pituitary adrenal axis is the neuroendocrine axis most closely connected with binge eating disorder (Geliebter *et al.*, 2004) The hypothalamus pituitary adrenal axis is a complicated network of interactions including the brain, pituitary gland, and adrenal glands. Researchers believe that dysregulation of the hypothalamus pituitary adrenal axis may play a role in the development of psychiatric diseases (Larsen and Ramshorst, 2009).

Binge eating disorder is associated with mental problems so, this adiposopathy signal molecule known as leptin must be paired with binge eating disorder.

Not only leptin adiponectin and inflammatory cytokines are other molecules secreted from white adipose tissue that can affect binge eating disorder. Adiponectin has the ability to regulate the reward pathways involved in food intake and eating behavior (Reis, Bressan and Alfenas, 2010) The mesolimbic reward system refers to the brain reward pathway that involves food appetite (Roefs, Franssen and Jansen, 2018). If this mesolimbic reward pathway is active, our brain releases chemicals associated with pleasure and reward, such as dopamine, serotonin, and endorphins. That is why people eat food out of control without hunger to experience pleasure and reward. Adiponectin can reduce the release of dopamine, serotonin, and endorphins (Awofala *et al.*, 2019). As a result, adiponectin generated from white adipose tissue may be linked to binge eating disorder.

People who suffer from binge eating disorder consume a lot. They have eating cravings from time to time. As a result, they always have a surplus of nutrients in their bodies. When a person has an excess of nutrients in their body, this is referred to as nutritional excess. The majority of these surplus nutrients begin to accumulate in the body. Adipose tissue is important for storing food, but being constantly surrounded by an excess of nutrients to store in the body makes adipose tissue sick.

Due to an excess of nutrients in the body, white adipose tissue produces inflammatory cytokines (Dixit, 2008). Inflammatory cytokines are linked to obesity-related bad eating habits (Caldas *et al.*, 2022).Nutrient excess is not only a cause but also one of the primary causes of inflammatory cytokines in patients with binge eating disorder. Pro-inflammatory cytokines are a class of signaling molecules that play in the immune system (De Heredia, Gómez-Martínez and Marcos, 2012). The majority of cytokines are a type of signaling molecule that is also involved in the immune system. The majority of cytokines stimulate glucose absorption in adipose fat, while some induce inflammation (Coppack, 2001). Inflammation causes adipose tissue damage and dysfunction.

Interleukin-6 [IL-6] and tumor necrosis factor-alpha [TNF α] are cytokines associated with binge eating disorders because these two can operate directly on the neural circuits that control food intake in the hypothalamus (Butler, Perrini and Eckel, 2021). TNF α is a member of the adipokine family that has only recently been discovered (Erbaş *et al.*, 2022). TNF α is produced not just by white tissue in the body, but also by immune cells such as macrophages, T cells, B cells, and natural killer cells (Paouri *et al.*, 2017).TNF alpha can stimulate the recruitment of immune cells into adipose tissue, such as macrophages.

TNF alpha can sometimes hinder the development of pre-adipocytes into mature adipocytes. This lead reduces fat storage capacity. One of the essential properties of TNF alpha is its anorexigenic influence on eating (Amaral *et al.*, 2006). It has anorexigenic properties, which means it can suppress appetite and reduce food consumption. TNF α has the ability to directly alter the hypothalamic area, which controls hunger and food intake. TNF α demonstrates that it has merged with adiposopathy to generate binge eating disorder.

Apart from TNF α another type of inflammatory cytokine linked to binge eating disorder is IL-6, which might boost hunger in those suffering from binge eating disorder (Caroleo *et al.*, 2019). Hormonal and neutral processes regulate appetite. The hypothalamic arcute nucleus houses two conflicting sets of neuronal pathways (Kalra *et al.*, 1999) There are primarily two circuits that transmit signals to the paraventricular nucleus and other hypothalamic nuclei, which directly control feeding behavior. This mechanism is influenced by peripheral hormone signals capable of crossing the blood-brain barrier.

IL-6 can stimulate hunger by increased expression of neuropeptide Y [NPY] and Agouti-related protein [AgRP] (Caroleo *et al.*, 2019). NPY is a peptide neurotransmitter found in both the central and peripheral nervous systems, where it is released into synaptic clefts to regulate physiological activities such as hunger (Straub *et al.*, 2000).NPY has been linked to addictive behavior. This demonstrates that binge eating disorder has an impact on the neuroendocrine and immune systems, with alterations in inflammatory mediators in specific brain areas contributing to the establishment of eating addiction.

Not only does IL-6 raise NPY, but it can also raise AgRP. AgRP is a kind of neuropeptide. AgRP is largely produced in the brain's hypothalamus. Because AgRP regulates appetite and energy balance. It is significant in binge eating disorder (Wu, Boyle and Palmiter, 2009). AgRP is produced by AgRP neurons, a group of neurons in the hypothalamus. Hunger signals produced by AgRP activate this which acts on a specific sort of brain receptor known as melanocortin -4- receptor [MC4R]. MC4R can stimulate appetite. This shows that adipose tissue-derived molecule IL-6 has an impact on adiposopathy for binge eating disorder.

Conclusion:

Adipocytokine is secreted by white adipocytes. Adipocytokines are active substances generated from white adipose tissue. Leptin is adipocytokine. Leptin plays an important part in appetite regulation. The main apparent symptom of binge eating disorder is a various hunger for food. Leptin has the ability to influence the neuroendocrine axes. The neuroendocrine axis most closely connected with binge eating disorder is the hypothalamic pituitary adrenal axis. The hypothalamic pituitary adrenal axis may be dysregulated, which may lead to the development of psychiatric illnesses such as binge eating disorder.

Adiponectin is also an adipocytokine. Adiponectin has the ability to control the reward pathway, which is involved in food intake and eating behavior. The body contains an excess of nutrients in the case of a binge eating disorder. Adipocytokine releases pro-inflammatory cytokines as a result of an excess of nutrients in the body. Interleukin 6 and TNF alpha are the key pro-inflammatory cytokines related to binge eating disorder. Food is anorexigenic when exposed to TNF alpha. It has anorexigenic properties which means it can suppress suppress appetite and reduce food consumption. IL-6 can stimulate hunger

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