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An Analysis of the Relationship between Binge Eating Disorder and Metabolic Disorders in Adults

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Abstract. The incidence of binge eating disorder (BED) has dramatically increased in recent years, leading to increased rates of psychiatric and medical comorbidities such as obesity and diabetes. Metabolic disorders are a series of metabolic dysfunctions, represented by central obesity, insulin resistance, and dyslipidaemia. This literature review aims to explore the relationship between BED and metabolic disorders in adults, helping to raise early awareness of these issues. PubMed was used as the exclusive database for its comprehensive and authoritative medical content. The review includes 13 peer-reviewed articles from the past decade, excluding studies on animals and children due to significant differences in BED characteristics across age groups and species. The focus remains on adult populations, as studies on gender or age effects showed no significant impact. Overall, BED has a strong relationship with metabolic disorders; people with BED will have higher risks for visceral obesity and Type 2 diabetes, whereas the limitations of research, such as further research on the complexity of BED, should be improved, and some people have opposite opinions, indicating a need for further in-depth investigation.

Keywords: Binge eating disorder, metabolic disorders, type 2 diabetes, visceral obesity, metabolism.

1. Introduction

Binge eating disorder(BED) has been affecting more and more people in recent years, yet few are fully aware of the seriousness of this issue[1]. Approximately 8.8% of adults and 5.7% of adolescents suffered from BED between 2013-2018[2]. BED not only disrupts daily eating habits but also gradually impacts both physical and mental health. However, there is limited research on the relationship between BED and metabolic disorders, with most studies focussing on treatments or the neurobiological aspects of BED. For example, Juli et al. explored various available pharmacological therapies for BED[3]. In the absence of a well-established nursing environment for remote BED treatment, Shepherd et al. pointed out the effectiveness of a remote treatment program[4]. Additionally, an animal study by Alma Delia Genis-Mendoza et al. demonstrated that mice with BED exhibited neurological issues such as depression and anxiety[5]. However, fewer studies have addressed the metabolic complications associated with BED. This study aims to determine whether BED increases the risk of metabolic disorders in the hopes of raising awareness and encouraging earlier intervention. This article focusses on the consequences of BED and compares them with the symptoms of metabolic disorders, drawing on research from PubMed and comparing findings on BED and its related comorbidities. This review is intended to help patients

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recognize the severity of BED and prompt timely intervention to reduce both the physical and mental health impacts.

2. Method

This literature review exclusively uses PubMed as the database. PubMed was chosen because of its authoritative and comprehensive collection of medical articles, offering a wide range of peer-reviewed studies on eating disorders and metabolic disorders. The keywords for research included "binge eating disorder and metabolic disorders," "eating disorder and metabolic disorders," "binge eating disorder," "binge eating disorder and type 2 diabetes," and "binge eating disorder and visceral obesity." To ensure the relevance of the articles, the review focuses on 13 studies published in the past decade, with one reference for an image dating back no more than 20 years. Martin et al. highlighted differences in brain function development related to BED between adults and adolescents, indicating that the characteristics of BED reward networks vary between these groups, so findings from studies on children cannot be directly applied to adults[6]. Similarly, animal models do not perfectly replicate BED in humans[5]. However, there are no significant effects on gender or age[7]. Thus, this review mainly focuses on the relationship between BED and metabolic disorders in adults.

3. The Relationship Between Binge Eating Disorder and Metabolic Disorders in Adults

3.1. BED and Metabolic Disorders

BED, which is both psychological and non-psychological, was discovered over 300 years ago and global eating disorder prevalence increased from 3.5% to 7.8% between 2000 and 2018[8]. It can affect people of any gender, age, race, weight, or socioeconomic status[1]. BED is characterized by consuming large amounts of food rapidly while feeling a loss of control. Key behaviours include eating quickly, feeling overly full, eating when not hungry, eating alone due to embarrassment, and feeling nauseous or guilty afterward. These episodes typically occur at least three times a week for three months or longer[9].

Metabolic disorders are medical conditions that impact metabolism and body functions. They are marked by central obesity, insulin resistance, hypertension, and dyslipidaemia, and they increase the risk of cardiovascular disease and type II diabetes (T2D). The global incidence rate of metabolic disorders is almost parallel to the incidence rate of obesity. The primary causes of metabolic disorders include genetic, environmental, and lifestyle factors. Visceral obesity has been identified as the main trigger for the development of metabolic disorders, with high-calorie intake being the primary cause of visceral fat accumulation. Insulin resistance, adipose tissue dysfunction, and chronic inflammation are considered fundamental components of the pathogenesis of metabolic disorders[10]. Insulin resistance impairs glucose uptake, leading to elevated blood glucose levels and hyperinsulinemia. Visceral obesity exacerbates this by releasing free fatty acids, promoting insulin resistance and causing chronic inflammation. Adipose tissues also secrete pro-inflammatory cytokines like TNF-alpha, which increase insulin resistance in the muscles, liver, and adipose tissues by inhibiting insulin signaling. This contributes to complications like hypertension and thrombogenesis, further perpetuating metabolic disorders. Additionally, some behaviours associated with BED increase the risk of developing metabolic disorders; approximately 32%–60% of patients seeking treatment for metabolic disorders also have BED[7].

3.2. Relationship between BED and Type II Diabetes

According to Abraham et al., overeating is related to T2D, hypertension, and visceral fat accumulation. Their research found that individuals with BED carry a greater metabolic burden, including high rates of obesity, hypertension, hyperglyceridemia, and T2D compared to non-binge eaters (NBE). Even after weight loss, fasting blood glucose levels remain elevated in people with BED[7]. Another study by Succuro et al. demonstrated that people with BED still have higher insulin levels and greater insulin resistance than NBEs, even after adjusting for body mass index(BMI)[11]. Additionally, a study shows that overall insulin sensitivity has decreased by 28% compared to baseline levels in healthy people[7].

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Figure 1 illustrates the mechanism of the impacts of BED on insulin. Insulin helps cells absorb glucose from the blood for energy or fat storage. In insulin resistance, glucose remains in the bloodstream, resulting in elevated blood sugar levels, similar to the effects of reduced insulin sensitivity. In this condition, muscles, fat, and liver cells do not respond effectively to insulin, prompting the pancreas to produce more insulin. Prolonged elevated insulin and blood glucose levels can exhaust the pancreas, eventually leading to T2D.

Moreover, consuming large meals as opposed to frequent small ones can have adverse metabolic consequences despite similar calorie intake. Large meals can lead to higher fasting blood sugar levels and impaired leptin inhibition after meals of glucagon. Leptin and adiponectin, two important hormones produced by adipose tissues, reduce liver glucose production by inhibiting glucagon and enhancing insulin sensitivity. Abraham et al. have found that the BMI-independent increase in fasting blood glucose among BED individuals may be due to reduced leptin production caused by overeating[7]. Adiponectin helps increase glucose uptake in muscles and reduces glucose production in the liver, whereas women with BED had significantly reduced plasma adiponectin levels, as shown in a previous study[11].

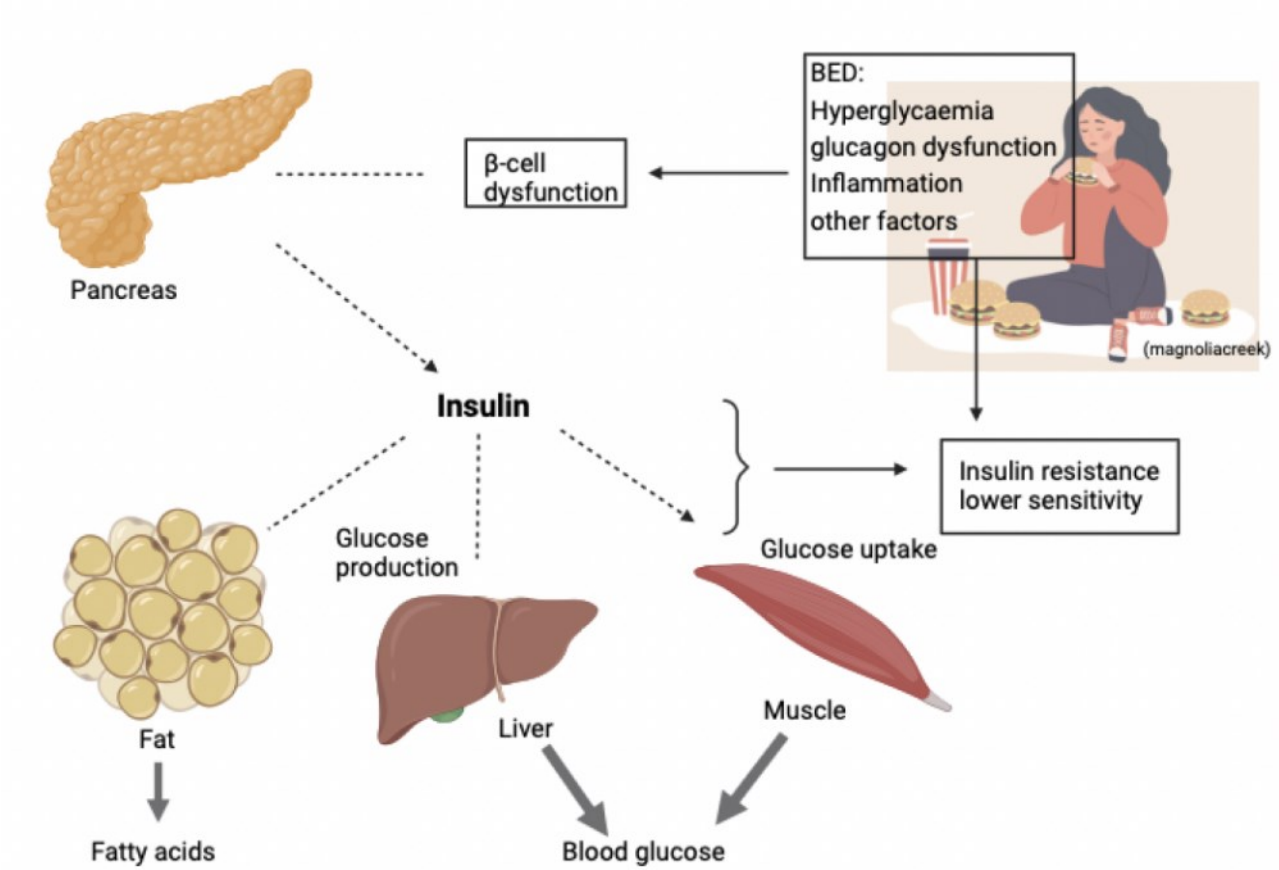

Figure 1. How BED affect insulin[12]

3.3. Relationship between BED and Visceral obesity

Visceral obesity is the primary cause of metabolic disorders, resulting from excessive intake of calories, carbohydrates, and fatty foods. According to Leone et al., they found a significant positive correlation between BED and waist circumference[13]. The abnormal calorie intake associated with BED accelerates the accumulation of visceral fat. Behaviours such as fast eating, nighttime eating, emotional eating, and a craving for carbohydrates in individuals with BED can lead to increased waist circumference, a higher visceral fat index, and insulin resistance, which may contribute to the production

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of inflammatory molecules[11]. Iceta et al. demonstrated that the visceral fat index(VAI) has a positive correlation with BED only in women, though no significant differences in BMI were observed. They also found women with high VAI experienced more frequent BED episodes[14].

In addition, the reduction of leptin and adiponectin in people with BED increases the risks of obesity. Adiponectin promotes the breakdown of fatty acids and lipid metabolism, which can help reduce the risks of visceral fat. Insulin resistance in individuals with BED may further contribute to visceral fat accumulation[11].

However, some studies suggest there is not always a strong relationship between BED and metabolic disorders. For instance, Leone et al. found no significant difference in body function between binge eaters and NBE. They used two ways to get these results and hypothesized that BED would increase the risk of obesity, but their results did not support this. In their research, body fat differed by only 0.3% between groups, despite differences in BMI[13]. Similarly, Iceta et al. found there is no substantial impact on the metabolism of visceral obesity in males with BED[14].

Given that some studies prove that the symptoms of BED and some metabolic disorders are not significantly different from other people, further research is necessary. In addition, because of the complexity of BED, it is not yet fully understood. To improve healthcare and enable earlier prevention and treatment, future studies should focus on the underlying mechanisms and potential treatment methods for BED.

4. Conclusion

In conclusion, there is a strong correlation between BED and increased risk of metabolic disorders in adults. This is because the consequences of BED, such as insulin resistance, weight gain, and lack of production of hormones, contribute to the development of metabolic disorders like T2D and visceral obesity. While most of the studies support the positive correlation between the degree of BED and the risk of metabolic disorders, some findings remain inconclusive. This review has certain limitations. First, the research methods have some shortcomings. The cited literature lacks animal experiments, thereby ignoring some ethically challenging but potentially effective research results that may not directly apply to humans. Some of the referenced studies are old, which could affect the findings due to technological and knowledge advances. To address these limitations in the future, this review could include relevant animal studies, despite their limitations in replicating human outcomes. Incorporating such studies could provide valuable foundational insights that complement human research.

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References

[1] Yu, Z. and Muehleman, V. Eating Disorders and Metabolic Diseases. International Journal of Environmental Research and Public Health, 20(3), p.2446. 2023. doi:https://doi.org/10.3390/ijerph20032446.

[2] Deloitte Access Economics. The Social and Economic Cost of Eating Disorders in the United States of America: A Report for the Strategic Training Initiative for the Prevention of Eating Disorders and the Academy for Eating Disorders. June 2020. Available at: https://www.hsph.harvard.edu/striped/report-economic-costs-of-eating-disorders/.

[3] Juli, R., Juli, M.R., Juli, G. and Juli, L. Eating Disorders and Psychiatric Comorbidity. 2023. [online] Psychiatria Danubina. Available at: https://www.psychiatria-danubina.com/UserDocsImages/pdf/dnb_vol35_noSuppl%202/dnb_vol35_noSuppl%202_217.pdf.

DOI: 10.54254/2753-8818/66/2024.18025

[4] Shepherd, C.B., Boswell, R.G., Genet, J., Oliver-Pyatt, W., Stockert, C., Brumm, R., Riebl, S. and Crowe, E. Outcomes for binge eating disorder in a remote weight-inclusive treatment program: a case report. Journal of eating disorders, 11(1). 2023. doi:https://doi.org/10.1186/s40337-023-00804-0.

[5] Alma Delia Genis-Mendoza, Isela Esther Juárez-Rojop, Yudy Merady Escobar-Chan, Carlos Alfonso Tovilla-Zárate, María Lilia López-Narváez, Nicolini, H. and Thelma Beatriz González-Castro. Increased Depressive-like, Anxiety-like, and Perseverative-like Behavior in Binge Eating Model in Juvenile Rats. Nutrients, 16(9), pp.1275–1275. 2024. doi:https://doi.org/10.3390/nu16091275.

[6] Martin, Elizabeth, et al. "Distinct Topological Properties of the Reward Anticipation Network in Preadolescent Children With Binge Eating Disorder Symptoms." Journal of the American Academy of Child & Adolescent Psychiatry (2024).

[7] Abraham, T.M., Massaro, J.M., Hoffmann, U., Yanovski, J.A. and Fox, C.S. Metabolic characterization of adults with binge eating in the general population: The framingham heart study. Obesity, 22(11), pp.2441–2449. 2014. doi:https://doi.org/10.1002/oby.20867.

[8] Galmiche, M., Déchelotte, P., Lambert, G. and Tavolacci, M.P. Prevalence of eating disorders over the 2000–2018 period: a systematic literature review. The American Journal of Clinical Nutrition, 109(5), pp.1402–1413. 2019. doi:https://doi.org/10.1093/ajcn/nqy342.

[9] McCuen-Wurst, C., Ruggieri, M. and Allison, K.C. Disordered eating and obesity: associations between binge-eating disorder, night-eating syndrome, and weight-related comorbidities. Annals of the New York Academy of Sciences, 1411(1), pp.96–105. 2017. doi:https://doi.org/10.1111/nyas.13467.

[10] Swarup, S. and Zeltser, R. Metabolic Syndrome. [online] Nih.gov. 2019. Available at: https://www.ncbi.nlm.nih.gov/books/NBK459248/.

[11] Succurro, E., Segura-Garcia, C., Ruffo, M., Caroleo, M., Rania, M., Aloi, M., De Fazio, P., Sesti, G. and Arturi, F. Obese Patients With a Binge Eating Disorder Have an Unfavorable Metabolic and Inflammatory Profile. Medicine, 94(52). 2015. doi:https://doi.org/10.1097/MD.0000000000002098.

[12] Stumvoll, M., Goldstein, B.J. and van Haeften, T.W. Type 2 diabetes: principles of pathogenesis and therapy. The Lancet, 365(9467), pp.1333–1346. 2015. doi:https://doi.org/10.1016/s0140-6736(05)61032-x.

[13] Leone, A., Vignati, L., Battezzati, A., Ramona De Amicis, Ponissi, V., Beggio, V., Giorgio Bedogni, Vanzulli, A. and Bertoli, S. Association of Binge Eating Behavior with Total and Abdominal Adipose Tissue in a Large Sample of Participants Starting a Weight Loss or Maintenance Program. Journal of the American College of Nutrition, 37(8), pp.701–707. 2018. doi:https://doi.org/10.1080/07315724.2018.1463184.

[14] Iceta, S., Dadar, M., Daoust, J., Scovronec, A., Leblanc, V., Pelletier, M., Biertho, L., Tchernof, A., Bégin, C. and Michaud, A. Association between Visceral Adiposity Index, Binge Eating Behavior, and Grey Matter Density in Caudal Anterior Cingulate Cortex in Severe Obesity. Brain Sciences, 11(9), p.1158. 2021. doi:https://doi.org/10.3390/brainsci11091158.