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OBESITY-ASSOCIATED MENTAL DISORDERS: NEUROBIOLOGICAL, PSYCHOSOCIAL AND CLINICAL-PSYCHOPATHOLOGICAL DETERMINANTS (NARRATIVE REVIEW)

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Abstract

Obesity is considered not only a metabolic disorder but also a condition closely associated with an increased risk of developing mental disorders. This article summarizes current evidence on biological, psychosocial, and genetic–epigenetic factors that increase the likelihood of psychopathology in patients with obesity. It is shown that chronic inflammation, insulin resistance, stress, stigmatization, emotional eating, body image disturbance, and early traumatic experiences form an interrelated system of risk factors. The review emphasizes the necessity of a comprehensive, multidisciplinary approach to the prevention and treatment of obesity and its comorbid mental disorders.

Keywords: Obesity, depression, anxiety, mental disorders, inflammation, insulin resistance, stigmatization.

Introduction

Obesity remains one of the major medical and social challenges of the 21st century and is characterized as a condition with a multifactorial pathogenesis. Alongside somatic complications such as metabolic syndrome, diabetes mellitus,

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Eureka Journal of Health Sciences & Medical Innovation (EJHSMI)
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and cardiovascular diseases, increasing attention is being paid to its association with mental disorders. Contemporary studies demonstrate that patients with obesity have a significantly higher risk of developing depression, anxiety disorders, and disturbances in eating behavior. However, the underlying mechanisms of these interrelationships remain a subject of ongoing debate. Most likely, the risk of mental disorders is shaped by the combined influence of biological factors (inflammation, hormonal regulation, insulin resistance), psychosocial factors (stigmatization, characteristics of coping strategies), and genetic–epigenetic determinants.

Aim of the Review

The aim of this review is to systematize current evidence on factors contributing to the development of mental disorders in patients with obesity and to identify directions for future research.

Methodology

The literature search was conducted using the scientific databases PubMed, PsycINFO, Scopus, and Google Scholar. The following keywords were used: “obesity,” “metabolic syndrome,” “depression,” “mental disorders,” “insulin resistance,” “inflammation,” “stigmatization,” and “early trauma.” Publications published between 2000 and 2025 were included in the review. Priority was given to meta-analyses, systematic reviews, and large cohort studies.

1. Neurobiological and Metabolic Factors

Obesity increases the risk of mental disorders through complex neurobiological and metabolic interactions that involve chronic stress, hormonal dysregulation, and components of the metabolic syndrome. The main neurobiological factors include dysregulation of the dopaminergic, endocannabinoid, and serotonergic systems, as well as a reduction in the activity and number of dopamine receptors,

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which may lead to pathological overeating. In addition, psychological characteristics such as social anxiety and avoidance of responsibility interact with neurobiological processes, further contributing to psychopathology [4,6,12].

Available evidence indicates a multifactorial interplay of biological, psychological, and social factors that may increase the risk of developing mental disorders. Nevertheless, the literature emphasizes the need for further research to definitively clarify cause–effect mechanisms, particularly through large-scale observational studies employing comprehensive regression analyses.

Chronic Systemic and Neuroinflammation

Other contemporary studies clearly demonstrate that obesity and its metabolic consequences—chronic inflammation and insulin resistance—not only coexist with mental disorders but also act as pathogenic mechanisms that induce structural and functional changes in the central nervous system (CNS). In patients with obesity, visceral adipose tissue functions as an active endocrine organ and synthesizes substantial amounts of proinflammatory cytokines, including interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), and C-reactive protein (CRP). This condition is characterized as chronic low-grade inflammation. Several large-scale studies and meta-analyses have shown that elevated levels of inflammatory biomarkers in patients with obesity are associated with increased severity of depressive symptoms and a higher likelihood of developing major depressive disorder (MDD) [33,35].

A **high-fat diet** induces neuroinflammation and depression-like behavior by activating inflammatory pathways in the brain. Numerous animal studies provide robust evidence for this mechanism: high-fat diets increase levels of proinflammatory cytokines (IL-1β, IL-6, TNF-α) and activate NF-κB signaling in the prefrontal cortex and hippocampus [35,51].

Several studies have demonstrated that reactivation of astrocytes in the medial prefrontal cortex exacerbates depression-like behavior [47,54]. In addition, high-

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ISSN 2760-4942 (Online) Volume 2, Issue 5, May 2026

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fat diets have been shown to disrupt intracellular signaling and activate the innate immune system, which is associated with elevated inflammatory markers and anxiety-like behavior [25].

The convergent findings of numerous studies using various animal models provide strong evidence for a link between diet-induced inflammation and neuropsychiatric symptoms.

Effects on Neurotransmitter Systems. Inflammatory cytokines activate the kynurenine pathway of tryptophan metabolism, leading to a reduction in the synthesis of serotonin—the key neurotransmitter regulating emotional states. At the same time, the production of neurotoxic metabolites such as quinolinic acid increases; these metabolites can exert deleterious effects on neurons and enhance the risk of developing depressive disorders. Numerous studies have demonstrated that proinflammatory cytokines induce indoleamine 2,3-dioxygenase (IDO), thereby diverting tryptophan metabolism away from serotonin synthesis toward alternative pathways [35,40].

Additional evidence indicates that cytokines not only deplete precursor reserves for serotonin synthesis but also generate neuroactive metabolites that significantly affect neurotransmitter regulation [35,39]. This mechanism involves activation of indoleamine 2,3-dioxygenase (IDO), which reduces the availability of tryptophan for serotonin synthesis and increases the production of neurotoxic metabolites such as quinolinic acid. Further findings confirm that this inflammatory–metabolic pathway represents a key factor in the neurobiological mechanisms underlying depression [35,39,40].

Taken together, this body of evidence demonstrates the existence of a robust neurobiological pathway linking inflammation and depression through disturbances in metabolic processes.

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Eureka Journal of Health Sciences & Medical Innovation (EJHSMI)
ISSN 2760-4942 (Online) Volume 2, Issue 5, May 2026

https://eurekaoa.com/index.php/5

Brain Insulin Resistance (IR). Insulin resistance (IR) is a significant metabolic disturbance that markedly affects central nervous system (CNS) function. It extends beyond peripheral tissues and directly impacts signaling in neurons and glial cells. Numerous studies have demonstrated that cerebral IR disrupts insulin's neuromodulatory functions and profoundly affects neuroplasticity, cognitive processes, and mood regulation [16,42].

Notably, there is strong evidence that IR measured by the HOMA-IR index is a more reliable predictor of mental disorders—particularly depression and anxiety—than body mass index [34]. Additional research indicates that insulin signaling in astrocytes directly modulates behavioral responses, highlighting the complex neurobiological mechanisms underlying metabolic–mental interactions [21,28].

Indeed, HOMA-IR–measured insulin resistance has been confirmed as a stronger predictor of mental disorders than obesity itself, both in children and adults. Multiple studies have shown significantly higher HOMA-IR values in children with mental disorders [42,43,50].

Numerous researchers have reliably confirmed that depressive symptoms increase insulin resistance regardless of body fat levels, particularly in boys, and have demonstrated a statistically significant correlation between insulin resistance and depressive symptoms in the general population [32]. The evidence is especially robust in children: depressive symptoms during childhood can prospectively predict disturbances in glucose homeostasis [50].

Insulin resistance is closely linked to mental disorders and operates through neurological mechanisms that disrupt the brain's reward system and dopaminergic signaling.

Multiple neuroimaging studies converge on a consistent finding: patients with insulin resistance exhibit significant alterations in brain activity. Specifically, during reward processing, decreased activation is observed in subcortical and limbic structures, while cortical responses are heightened [55]. Insulin resistance

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Eureka Journal of Health Sciences & Medical Innovation (EJHSMI)
ISSN 2760-4942 (Online) Volume 2, Issue 5, May 2026

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also fundamentally alters dopaminergic systems, particularly in brain regions associated with motivation, such as the anterior cingulate cortex [52]. Neuroimaging data indicate reduced brain responses to positive stimuli within reward-related circuits, altered dopamine turnover, and mitochondrial dysfunction. Findings from several independent research groups collectively demonstrate a robust neurobiological link between metabolic dysfunction and depression [28].

Overall, these findings provide strong evidence for a neurobiological basis of motivational deficits in depression, encompassing diverse research approaches and participant groups.

Insulin resistance, together with chronic stress, leads to hyperactivity of the hypothalamic–pituitary–adrenal (HPA) axis, resulting in persistently elevated cortisol levels. This disrupts neurogenesis in the hippocampus and exacerbates symptoms of depression and anxiety. Numerous studies confirm this mechanism: stress-induced glucocorticoids can impair brain glucose metabolism and reduce insulin sensitivity [16,24].

Results presented by V. Sharma and colleagues indicate that chronic stress increases glucocorticoid secretion, which contributes to insulin resistance and inflammation [49]. Research conducted in both humans and animals has confirmed a correspondence between metabolic disturbances (such as insulin resistance, diabetes, and obesity) and neuropsychiatric disorders, suggesting the existence of shared pathophysiological mechanisms [31].

The role of insulin in neurogenesis is particularly emphasized, with its effects on the proliferation and differentiation of precursor cells in the brain being well documented [16]. Evidence suggests the existence of a complex and interconnected pathway in which insulin resistance, stress, and cortisol dysregulation collectively contribute to the development of neurological and psychological dysfunctions.

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Eureka Journal of Health Sciences & Medical Innovation (EJHSMI)
ISSN 2760-4942 (Online) Volume 2, Issue 5, May 2026

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Thus, a neurobiological approach to the comorbidity of obesity and mental disorders presents the following model: metabolic stress resulting from excess adipose tissue and unhealthy eating (insulin resistance and inflammation) convergently disrupts key brain systems, including neurotransmitter homeostasis, neuroplasticity, and stress-response mechanisms.

2. Psychosocial and Behavioral Factors

Psychological factors contributing to the development of mental disorders in obesity primarily include negative self-perception, social pressure, and impaired emotional regulation. Studies confirm that individuals with excess body weight often have a markedly negative body image, low self-esteem, and higher levels of depressive symptoms, largely due to social pressures related to body appearance. Numerous studies support this view: respondents with overweight or obesity predominantly exhibit negative attitudes toward their bodies, reduced psychological well-being, and a tendency toward depressive states [14].

Sociocultural factors, such as the unrealistic beauty ideals prevalent in the media, significantly contribute to discrimination and stigmatization [20,37]. Evidence indicates that this is a widespread issue: obesity frequently leads to feelings of shame, distress, and social isolation. Importantly, these psychological consequences are not solely related to weight but are also influenced by societal criticism and internalized negative body image [36].

Modern society stigmatizes excess body weight, creating significant psychological stress for individuals with overweight or obesity. This has been confirmed by numerous studies, which demonstrate a substantial impact on mental health. Several investigations have documented the widespread nature of social stigmatization and its negative effects on both mental and physical well-being [8,33,36].

According to the available data, individuals with excess body weight often experience [14]:

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Eureka Journal of Health Sciences & Medical Innovation (EJHSMI)
ISSN 2760-4942 (Online) Volume 2, Issue 5, May 2026

https://eurekaoa.com/index.php/5

- Negative self-perception;
- Reduced psychological well-being;
- Difficulties in achieving personal goals;
- Low self-esteem.
- Furthermore, data indicate that psychological problems are particularly pronounced in children: nearly 50% of children with excess body weight exhibit internal psychological difficulties, such as anxiety and egocentrism [44,48]. The psychological impact is multifaceted, encompassing reduced autonomy, a tendency toward pessimism, interpersonal tension, and heightened sensitivity to depressive states, which are primarily linked to social pressure and unrealistic external appearance standards. Several contemporary studies emphasize the significant influence of excess body weight on mental health.
- Research conducted in 2022–2023 demonstrates a correlation between obesity and the development of major depressive and anxiety disorders, particularly during childhood [11,19]. Psychological maladaptation is largely dependent on both internal and external factors, with gender and social issues playing an important role in its intensification [3]. Psychosocial maladaptation is complex and multifactorial, with key contributing factors including high personal anxiety, social stress, and limited access to important resources [5]. Gender differences have been identified in social–psychological adaptation: boys tend to show higher levels of adaptation and employ different psychological resilience strategies compared to girls. Research indicates that maladaptation is not a simple linear process but rather a subtle interplay of personal traits, social environment, and mechanisms for overcoming individual challenges.

Weight-Related Stigmatization. Weight-related stigmatization is a widespread and harmful form of discrimination that significantly affects various aspects of life for individuals with obesity. Numerous studies document pervasive discrimination and identify common stereotypes: people with obesity are often

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Eureka Journal of Health Sciences & Medical Innovation (EJHSMI)
ISSN 2760-4942 (Online) Volume 2, Issue 5, May 2026

https://eurekaoa.com/index.php/5

described as “lazy, unskilled, and unproductive” in healthcare, employment, and interpersonal contexts [29,49]. These individuals are frequently perceived as “weak, lazy, and unsuccessful” within professional and medical environments [13]. The consequences are serious: the level of weight-based discrimination is now compared to racial discrimination, and weight-related stigmatization is positively associated with depression, anxiety, and physiological markers of stress [44,53].

Body Image Disturbance and Bodily Dysmorphism. Beauty standards promoted by mass media contribute significantly to body image disturbances and eating disorders, creating unrealistic expectations regarding appearance and leading to body dissatisfaction. Numerous studies indicate that social media platforms, particularly among adolescents, exacerbate anxiety related to body image [3,27].

Large-scale studies consistently show a significant association between social media use and increased body image concerns, with multiple meta-analyses providing robust correlational evidence. For example, Bonfanti et al. (2024) analyzed 83 studies involving 55,440 participants and found a moderate correlation between online social comparison on social media and body image anxiety [20]. In a sample of 1,331 individuals aged 15–35, a positive relationship was observed between the frequency of appearance-related comparisons and body dissatisfaction [13,30]. Young people are especially sensitive, with mediating factors including social comparison, internalization of thin ideals, and self-objectification [23]. The evidence indicates that social media use contributes to body image issues through mechanisms of continuous comparison and internalization of unrealistic appearance standards, leading to problems with self-acceptance [46].

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Eureka Journal of Health Sciences & Medical Innovation (EJHSMI)
ISSN 2760-4942 (Online) Volume 2, Issue 5, May 2026

https://eurekaoa.com/index.php/5

Using Food as a Coping Mechanism. Excess energy intake, particularly high-calorie "comfort" foods, often serves as a maladaptive mechanism to reduce emotional stress, anxiety, or boredom. This "stress–eating–guilt–stress" cycle contributes not only to weight gain but also to the exacerbation of underlying mental disorders.

Available evidence confirms a strong link between emotional state and eating behavior. Emotional overeating represents a paradoxical, self-reinforcing cycle in which high-calorie foods are used as a maladaptive stress-relief strategy, leading to increased psychological stress and potentially more serious mental health problems. Numerous studies support this connection: sweet and fatty foods can temporarily alleviate stress via neurotransmitter pathways [26], and emotional overeating has been shown to correlate with heightened psychological stress and depressive symptoms [22].

Furthermore, this behavior is classified as a "maladaptive conflict-resolution strategy," in which food symbolically compensates for emotional deficiencies [2]. The cycle of emotional consumption, guilt, and intensified stress forms a self-perpetuating closed loop, ultimately undermining both mental and physical well-being.

Reduced Physical Activity and Social Isolation. Physical difficulties and stigmatization associated with obesity can lead to avoidance of social situations, resulting in social isolation. Reduced physical activity deprives patients of the antidepressant and anxiolytic effects typically obtained through exercise. Numerous sources note that social stigmatization related to obesity negatively affects both mental and physical health [8]. Individuals with morbid obesity often experience severe physical limitations, reporting an inability to perform even minimal exercise and avoiding public spaces such as gyms [45]. Other researchers emphasize that physical exercise is crucial for improving the

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Eureka Journal of Health Sciences & Medical Innovation (EJHSMI)
ISSN 2760-4942 (Online) Volume 2, Issue 5, May 2026

https://eurekaoa.com/index.php/5

condition of patients, particularly those suffering from both obesity and depression [1].

There is an evidence-based connection between treating excess body weight and improvements in mental health: effective obesity therapy contributes to a reduction in depressive symptoms. Systematic physical activity represents a strong, multifaceted intervention that simultaneously supports mental health stabilization and weight management. Marconcin et al. (2022) consistently found across diverse population groups that higher levels of physical activity are associated with greater well-being and fewer depressive symptoms [38].

Existing evidence indicates a complex, bidirectional relationship: obesity can induce psychological stress, while psychological stress can, in turn, exacerbate obesity-related problems.

3. Genetic and Early-Life Factors

General genetic predictors: several genes may increase susceptibility not only to obesity but also to certain mental disorders, for example, by affecting dopaminergic regulation related to the reward system and addiction or by influencing stress regulation. This suggests that some genes may predispose individuals simultaneously to obesity and mental disorders through shared genetic vulnerabilities, particularly within neurotransmitter and stress-regulation mechanisms.

Within the framework of modern psychiatric genomics, attention has focused on the role of polygenic and poly-epigenetic factors in shaping mental health [10]. Research indicates that specific genetic polymorphisms may serve not only as predictors of particular psychological traits but also influence the development of pathological conditions [7]. In summary, the etiopathogenesis of obesity–mental disorder comorbidity is shaped by a complex synergy of genetic predisposition, epigenetic modifications, and environmental factors [17]. Although genetic

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Eureka Journal of Health Sciences & Medical Innovation (EJHSMI)
ISSN 2760-4942 (Online) Volume 2, Issue 5, May 2026

https://eurekaoa.com/index.php/5

overlap has been confirmed, the precise mechanisms remain incompletely understood.

Early Childhood Stress and Trauma. Experiences such as childhood abuse, neglect, emotional deprivation, or chronic family stress represent potent risk factors that program biological systems not only for metabolic disturbances but also for mental disorders.

Adverse childhood experiences can fundamentally alter stress-regulation systems in the brain, significantly increasing the risk of both metabolic dysfunction and psychiatric disorders. Research indicates that repeated stress during sensitive developmental periods reorganizes neuroendocrine system activity [41]. These neurobiological changes act as catalysts for subsequent metabolic dysfunctions [47].

A detailed neurobiological analysis confirms that the pathogenesis of post-traumatic stress disorders involves complex alterations, including dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis, impaired cortisol secretion, neurochemical imbalances, and structural brain changes. These interconnected neurobiological modifications provide direct evidence of the link between childhood trauma and heightened vulnerability to both metabolic and mental disorders [4].

Hypothalamic–Pituitary–Adrenal (HPA) Axis Dysregulation. The hypothalamic–pituitary–adrenal (HPA) axis—the body's primary stress system—serves as a common final pathway through which genetic predisposition and early-life stress exert their pathogenic potential, profoundly affecting both metabolic and mental health.

Chronic stress can lead to either hyperactivation or fatigue of the HPA axis, reflecting a complex physiological response to prolonged stress. Chronic HPA

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Eureka Journal of Health Sciences & Medical Innovation (EJHSMI)
ISSN 2760-4942 (Online) Volume 2, Issue 5, May 2026

https://eurekaoa.com/index.php/5

hyperactivation may transition from a physiological to a pathological state, particularly in conditions of sustained excessive cortisol secretion [9,15].

Feodorova et al. (2012) also noted that stress can differentially regulate gene expression, which may explain variability in HPA responses [18]. HPA dysfunction represents a critical convergence point, where genetic predisposition and early-life stress interact to produce deep neurobiological vulnerability.

Thus, genetic and early-life factors are not independent; they interact according to the "gene–environment" principle, creating profound neurobiological vulnerability in individuals that manifests as metabolic disturbances (obesity) and impaired emotional regulation (mental disorders).

Conclusion

This review demonstrates that factors exacerbating mental disorders in obesity operate at three levels:

1. Biological level — inflammation, insulin resistance (IR)
2. Psychosocial level — stigma, coping mechanisms
3. Developmental level — early-life stress, genetics

Future research should focus on the following directions:

1. Prospective cohort studies — to establish causal relationships between metabolic changes and the development of mental disorders.
2. Personalized, integrated approaches — designing therapeutic strategies that simultaneously target metabolic disturbances (e.g., anti-inflammatory diets, medications for IR) and psychological factors (e.g., cognitive-behavioral therapy, work with maladaptive coping, and stigma reduction).
3. Investigation of specific micronutrients — studying the role of omega-3 fatty acids, vitamin D, and other micronutrients in reducing inflammation and improving mental health in patients with obesity.

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Eureka Journal of Health Sciences & Medical Innovation (EJHSMI)

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