

The Impact of Psychological Stress on Obesity Risk in Women: The Roles of Emotional Eating and Sleep Quality

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Abstract. Obesity in women is a serious global public health challenge closely linked to chronic psychological stress. The female hypothalamic-pituitary-adrenal (HPA) axis exhibits heightened sensitivity, predisposing women to unhealthy behaviors under stress. However, existing studies have examined individual behavioral pathways in isolation without integrating emotional eating and sleep disturbances into a unified framework. This narrative review searched PubMed, Google Scholar, and other databases for high-quality population-based studies published between 2020 and 2025. It examines gaps in the existing research on the multiple behavioral mechanisms through which stress may contribute to obesity in women, with a focus on the independent and combined mediating effects of three behavioral categories. Research confirms that chronic stress elevates cortisol levels, induces emotional instability and behavioral patterns such as emotional overeating and poor sleep, thereby increasing women's risk of weight gain; this behavioral pathway exhibits significant gender differences. Most existing evidence consists of cross-sectional data, with a lack of studies specific to women across their life stages. This review provides insights into combined behavioral interventions for the prevention and control of obesity in women and suggests directions for future epidemiological research.

Keywords: psychological stress, female, obesity, emotional eating, sleep quality, public health

1. Introduction

Obesity is a major global public health challenge and significantly increases the risk of type 2 diabetes, cardiovascular disease, and a decline in quality of life. In 2022, approximately 2.5 billion adults worldwide were overweight, of whom 890 million were obese, reflecting that obesity has become a widespread global health burden [1, 2]. For women, the risk of obesity may also be influenced by multidimensional factors at different life stages, such as work and family responsibilities, caregiving pressures, body image, and socioeconomic status [1].

A growing body of research suggests that psychological stress may contribute to the development of obesity; however, this effect does not necessarily manifest directly as weight gain but may instead operate through multiple interrelated processes, including alterations in the HPA axis and associated metabolic changes, changes in eating behavior, and impaired sleep [3, 4]. Emotional eating—that is,

eating in response to negative emotions rather than physiological hunger—has thus emerged as a potential pathway with strong biological and behavioral plausibility.

Sleep may represent another important pathway, as insufficient or poor-quality sleep can further disrupt appetite, energy intake, and emotional regulation [5, 6]. A systematic review of 50 randomized controlled trials found that sleep restriction was generally associated with higher energy intake, fat intake, feelings of hunger, and frequency of eating [7]. More notably, sleep and emotional eating may not be independent risk factors. Du et al. conducted a study of 1,392 college students, including 973 women, and found that emotional eating mediated the relationship between psychological stress and BMI, while higher sleep quality attenuated this relationship—an effect that was particularly pronounced among women [8]. Meanwhile, a systematic review noted that emotional eating may be involved in the relationship between sleep deprivation and obesity, suggesting that there may be more complex links among sleep, eating behavior, and body weight [9].

This narrative review aims to synthesize evidence from human studies published between 2020 and 2025 in journals indexed by PubMed and Google Scholar to examine the relationship between psychological stress and the risk of obesity in women. It further analyzes the potential roles of emotional eating and sleep quality in this process, focusing on answering three questions: How is psychological stress associated with the risk of obesity? Can emotional eating and sleep quality serve as potential mediating pathways? Do these two factors interact with one another? The need for a women-centered approach to integration stems from the fact that biological sex differences and stressors related to gender roles can alter stress responses and their behavioral consequences; yet existing research often combines men and women in its analyses or relies too heavily on samples of young women and college students.

2. Emotional eating as a potential pathway

Emotional eating typically refers to a situation in which an individual, when experiencing negative emotions such as anxiety, tension, or sadness, uses eating—rather than responding to physical hunger—as a way to regulate emotions or relieve stress. Clinical and behavioral reviews have consistently found that psychological distress, anxiety, or stress are associated with higher levels of emotional eating, which in turn is linked to poorer dietary patterns or higher body weight [5].

Meta-analysis evidence supports a small but detectable overall association between stress and eating behavior. After synthesizing 54 studies involving a total of 119,820 adults, Hill et al. found that stress was modestly positively correlated with overall food intake and was associated with increased consumption of unhealthy foods and decreased consumption of healthy foods [10]. Given the small effect sizes and high heterogeneity among studies, a more rigorous interpretation would be that stress may alter food choices and eating behaviors, thereby creating conditions for a positive energy balance over the long term, rather than necessarily leading to overeating or obesity.

Longitudinal studies of women provide stronger temporal evidence for the "stress–emotional eating" relationship. Fowler et al. conducted a 49-day follow-up of 477 women and found that the higher the women's average stress levels, the higher their levels of emotional eating; meanwhile, at the individual level, when stress on a given day exceeds a woman's personal average, emotional eating on that day also increases [11]. Previous major life stressors further strengthen this daily association. Compared with cross-sectional studies, this type of design better supports the short-term temporal dynamics between stress and emotional eating; however, it still does not prove that emotional eating necessarily leads to long-term weight gain.

The association between emotional eating and obesity is also supported by systematic reviews. Vasileiou and Abbott found that obese adults exhibited higher levels of emotional eating compared to those of healthy weight, but the differences between overweight and healthy-weight individuals were inconsistent [6]. A more recent meta-analysis estimated that the overall prevalence of emotional eating among overweight or obese individuals was approximately 44.9%, although substantial heterogeneity was observed across studies [12]. Differences in definitions, questionnaires, study populations, and cut-off points limit the interpretive value of a single pooled prevalence estimate.

Furthermore, bidirectional relationships must be considered. Recurrent emotional eating may increase energy intake and promote weight gain, while higher body weight itself may increase psychological stress due to body dissatisfaction, weight stigma, or negative self-evaluation. Consequently, stress may contribute to emotional eating, while weight-related psychological distress may in turn sustain or exacerbate stress, potentially creating a feedback loop. Existing evidence is consistent with this model but is insufficient to prove the complete cycle. Therefore, emotional eating is best viewed as a clinically significant potential behavioral pathway rather than the sole established mediating mechanism.

3. Sleep quality as a potential pathway

Sleep may be another important pathway linking psychological stress to the risk of obesity in women, and it may also interact with emotional eating. There is a bidirectional relationship between psychological stress and sleep: persistent stress may disrupt normal sleep through physiological arousal, negative emotions, and cognitive rumination, while insufficient sleep or poor sleep quality may further amplify the stress response. Sleep is another potential pathway linking psychological stress to obesity risk. Psychological stress can impair sleep through cognitive arousal, rumination, and sustained physiological activation, while insufficient sleep may, in turn, amplify the stress response and weaken emotional regulation. Sleep duration and sleep quality should not be treated as interchangeable measures: two individuals with similar sleep durations may differ significantly in terms of sleep continuity, sleep efficiency, and subjective feelings of restfulness.

Evidence from randomized controlled trials and clinical studies suggests that sleep restriction can alter behaviors related to energy balance. A systematic review incorporating 50 randomized controlled trials found that sleep restriction was generally associated with higher energy and fat intake, feelings of hunger, and increased frequency and portion sizes of meals [7]. However, the review did not find consistent changes in circulating leptin, ghrelin, or cortisol levels; therefore, the phenomenon should not be explained solely by a simplistic "sleep deprivation—hormonal changes—obesity" model. Broader cardiovascular and metabolic evidence also indicates that both sleep duration and sleep quality are associated with cardiometabolic health [12]. The interaction between sleep and emotional eating warrants particular attention among women. Du et al. conducted a cross-sectional study of 1,392 college students, 973 of whom were female. The study showed that emotional eating played a statistically significant mediating role between perceived stress and BMI, and that better sleep quality could attenuate this relationship; sleep duration, however, did not exhibit the same moderating effect [8]. These findings suggest that sleep quality may modify the extent to which psychological stress influences eating behavior, rather than merely acting as an independent risk factor. However, because stress, sleep, eating behavior, and BMI were all measured at the same time point, the direction of the pathways cannot be determined based on these data.

Women-specific observational studies also support a link between sleep, anxiety, and emotional eating. Among 191 female undergraduate health science students, Díaz et al. observed a higher

prevalence of poor sleep quality and found associations between sleep-related measures and psychological factors [13]. Such studies help identify patterns of risk clustering but remain susceptible to self-report bias, common-method bias, and limited representativeness of the student sample.

Taken together, the existing evidence supports several possible models: stress may simultaneously and independently reduce sleep quality and increase emotional eating; poor sleep may amplify stress-related emotional eating by impairing emotional regulation and appetite control; conversely, emotional eating and irregular eating patterns may further impair sleep. Therefore, while the sequence "stress → impaired sleep → emotional eating → weight gain" is plausible, it remains a hypothesis that requires further validation. Few studies have directly tested this temporal sequence using repeated measurements of these four variables over time within the same cohort of women.

4. Limitations and public health implications

The most significant limitation of the current literature lies in causal inference. Because many studies use cross-sectional designs, mediation or moderation findings based on single-time-point data should be interpreted as statistical associations rather than evidence of temporal or causal relationships [8]. Even in longitudinal studies, researchers frequently capture only a subset of the variables involved in the proposed mechanisms. Future studies need to include repeated measurements of stress, sleep, emotional eating, and obesity indicators over a sufficiently long follow-up period, with pre-specified temporal sequence models.

The second issue is measurement heterogeneity. Stress may be defined as perceived stress, daily stress, major life events, or psychological symptoms; emotional eating is assessed using different scales and thresholds; and sleep is often reduced to self-reported sleep duration or overall sleep quality scores. Future studies should employ more standardized measurements and incorporate sleep diaries, wrist-worn activity monitors, and more objective measures of obesity. At the same time, a clear distinction should be made between sleep duration, sleep efficiency, sleep regularity, and subjective sleep quality, as these dimensions may have different relationships with eating behavior.

Third, women are not a homogeneous group. Much of the existing evidence comes from young women and college students, but women experience significant changes in their hormonal environment, social roles, sleep, and body composition throughout their lifespans. Pregnancy and the postpartum period, caregiving responsibilities, shift work, and perimenopausal changes can all alter stress exposure and susceptibility to weight gain. Therefore, future studies should stratify participants by life stage rather than treat "women" as a single category. Potential confounding factors also need to be controlled more systematically. Symptoms of anxiety and depression can simultaneously increase perceived stress, impair sleep, and promote emotional eating; physical activity, dieting history, socioeconomic status, chronotype, medication use, and baseline BMI may also influence several variables simultaneously. If these factors are not adequately controlled, the observed associations may partially reflect these common determinants.

Despite these limitations, this integrative framework remains relevant to public health. When women experience persistent stress, sleep disturbances, or significant emotional eating simultaneously, obesity prevention and treatment should not rely solely on calorie restriction and physical activity recommendations. Screening for these factors can help identify populations for whom traditional weight management advice may be insufficient. It is important to emphasize that improving sleep should not be promoted as a standalone weight-loss treatment but should instead be integrated with stress management, emotional regulation, healthy eating, and physical activity as part of a broader behavioral intervention strategy.

From a public health perspective, a tiered intervention approach may be appropriate. For the general population, basic stress management and sleep health content can be incorporated into health education on nutrition and physical activity; women experiencing persistent stress, poor sleep quality, or significant emotional eating may benefit from further targeted behavioral assessments and support; and for high-risk individuals who are obese and have multiple co-morbidities, multidisciplinary management involving nutritionists, mental health professionals, and sleep specialists may be necessary. This approach is consistent with current evidence and avoids the overgeneralization that "changing a single factor can prevent or reverse obesity."

5. Conclusion

Psychological stress may increase the risk of obesity in women through interrelated biological and behavioral pathways. Among these, emotional eating and sleep quality have received growing research support; however, neither can currently be considered an established single causal mediating mechanism. Existing research shows that stress is associated with a slight increase in overall and unhealthy food intake, and longitudinal data in women also indicate that elevated daily stress may coincide with increased emotional eating. Sleep restriction may promote higher energy intake and feelings of hunger, while observational studies primarily involving women further suggest that sleep quality may modulate the relationship among stress, emotional eating, and BMI.

Future research should first adopt a long-term longitudinal cohort design to simultaneously measure psychological stress, sleep, emotional eating, and weight/obesity indicators at multiple time points within the same female population, in order to clarify the temporal sequence among these variables. Second, further studies should evaluate combined behavioral interventions integrating stress management, emotional regulation, dietary behavior interventions, and sleep management to determine whether comprehensive interventions can produce synergistic effects.

Overall, the existing evidence is consistent with the "psychological stress–sleep health–emotional eating–obesity risk" pathway as a potentially interacting behavioral pathway, but it is not yet sufficient to establish a complete causal mechanism. From a public health perspective, the current evidence supports broadening obesity prevention and management in women beyond a sole focus on "eating less and exercising more" to include complementary measures such as stress management, sleep health, and emotional regulation, particularly for women with multiple coexisting risk factors. Future intervention studies should further examine whether comprehensive interventions are superior to single-intervention approaches, and whether intervention outcomes are influenced by baseline BMI, age, menopausal status, socioeconomic status, the severity of sleep problems, and the level of emotional eating. This will advance research beyond merely describing associations toward identifying "which women, at which life stages, are best suited for which combination of behavioral support measures."

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