



Depression and Obesity

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Abstract

Purpose of Review To summarize the recent literature regarding the interaction between the public health concerns of obesity and depression and provide preliminary recommendations for assessment and management of patients with co-occurring obesity and depression.

Recent Findings Recent studies have reinforced that while there is a bi-directional link between obesity and depression, there is stronger evidence that obesity contributes to the development of depression. Recent research has also elucidated potential mediating factors in the association between depression and obesity (e.g., demographical, biological, and psychological) as well as a moderating effect of treatment of depression on outcomes for treatment of obesity.

Summary There is evidence to suggest that in co-occurring depression and obesity, it is beneficial to treat depression first. Cognitive behavioral therapies have demonstrated effectiveness in treating co-occurring depression and obesity and there is preliminary evidence suggesting that increasing emotion regulation and reducing weight bias internalization may be important treatment targets.

Keywords Obesity · Depression · Assessment · Treatment

Introduction

Obesity and depression are two highly prevalent medical conditions that independently have effects on mortality and morbidity [1–4]. Systematic reviews of the literature show a reciprocal and bi-directional link between obesity and depression, with stronger evidence suggesting that obesity contributes to depression [5]. Co-occurring obesity and depression are linked to morbidity, mortality, poor adherence to treatment recommendations, and poor quality of life [6–8]. Obesity and depression are also strongly associated with shared behavioral and psychosocial consequences. The following review summarizes recent findings regarding the interaction of depression and obesity. Adequate screening, assessment, and ongoing monitoring and treatment may help mitigate this risk of worsening depression in obesity. Future research should examine the efficacy of such risk management strategies.

Given the high prevalence and global impact of these chronic diseases, the body of literature is quite large. This brief review is limited to recent studies published in English and with human adult participants. Please see systematic reviews and meta-analyses conducted by Moradi, Mozaffari, Askari, Azadbakht [9], Rao, and colleagues [10] for discussion of recent studies examining depression and obesity in children and adolescents. There is also a growing interest in the association between depression and obesity in pregnancy and postpartum. Please see the recent review by Cattane and colleagues for discussion of this literature [11].

Obesity

“Obesity is defined as a chronic, progressive, relapsing, and treatable multi-factorial, neurobehavioral disease, wherein an increase in body fat promotes adipose tissue dysfunction and abnormal fat mass physical forces, resulting in adverse metabolic, biomechanical, and psychosocial health consequences.” [12]. Severity of obesity is categorized by body mass index (BMI), including Class I Obesity (BMI 30.0–34.9) Class II Obesity (BMI 35–39.9), and Class III Obesity (BMI ≥ 40).

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Recent age-adjusted prevalence of obesity ($\text{BMI} \geq 30$) in US adults was 42.4%, with no significant differences between men and women among of all age groups [13]. Class III Obesity was found in 9.2% of adults and is more prevalent in women than in men. This review does not discuss associations between depression and overweight/pre-obesity (BMI 25.0–29.9).

Depression

Major Depressive Disorder (MDD), as defined in the Diagnostic and Statistical Manual of Mental Disorders [14] is characterized by low mood and/or diminished interest or pleasure (i.e., anhedonia) for two weeks or longer; additionally five or more of the following symptoms should be present including significant weight or appetite changes, insomnia or hypersomnia, psychomotor changes, fatigue or loss of energy, feelings of worthlessness or inappropriate guilt, difficulty with concentration or indecisiveness, and recurrent suicidal ideation with or without a specific plan or suicide attempt. Symptoms must cause clinically significant distress or impairment in functioning and the depressive episode cannot be attributed to physiological effects of a substance or other medical condition. An additional qualifier of “with psychotic features” is added when delusions or hallucinations are present during a depressive episode. Other related diagnoses that will not be reviewed in this paper include Persistent Depressive Disorder (formerly known as Dysthymia), Disruptive Mood Dysregulation, Premenstrual Dysphoric Disorder, Adjustment Disorder with Depressed Mood, and Bipolar Disorders.

Twelve-month prevalence of depression in the general US population is approximately 7% with threefold higher rates among young adults (aged 18–29 years) in comparison to older adults (aged 60 and above) and 1.5 to threefold higher rates among women compared to men beginning in early adolescence. Many factors increase the risk of developing MDD, including personality factors, environmental factors/stressors, genetics, and physiology. The presence of a chronic or disabling medical condition may also increase the risk of developing MDD [14].

Depression and Obesity

While depression and obesity are each pervasive and costly chronic illnesses, independent of one another, there is significant research establishing that they are frequently presenting together and likely interacting to worsen patient outcomes. For example, key findings from the National Health and Nutrition Examination Surveys indicated that 43% of adults with depression had obesity, while 33% of adults

without depression had obesity [15]. For adults treated with antidepressants, 55% of those with moderate or severe depression had obesity while 38% with mild or no depressive symptoms had obesity [15]. Further, reported rates of depression in patients with obesity tend to vary based on differences in study design, methodology (e.g., structured clinical interview vs screening questionnaire), and sample characteristics. Thus, when screening for depression, especially in populations with Class III obesity, it is important to differentiate between genuine symptoms of depression and those due to medical conditions (e.g., fatigue, changes in appetite, poor sleep, anhedonia vs limited physical functioning, etc.).

Previous research has investigated the role of depression in development of obesity, as well as the role of obesity in the development of depression. For example, meta-analysis of 15 studies supported the bi-directional association between depression and obesity, such that depression was found to be predictive of developing obesity and obesity was found to increase the risk of depression [16]. In a review of 25 population-based studies, where 15 tested the “depression-to-obesity” pathway and 10 tested the ‘obesity-to-depression’ pathway, there was stronger evidence for obesity leading to depression [5].

Recently, Liu and colleagues found that depression, as measured by the 9-item Patient Health Questionnaire (PHQ-9), increased the risk of incident obesity in a 10-year prospective cohort study of Chinese adults [17]. Meanwhile, Frank and colleagues determined that obesity preceded depressive symptoms, using meta-analysis of longitudinal data, in an international pooled sample of 57,532 patients aged 18 to 100 [18].

Contributing Factors

Previously discussed variables which moderate the relationship between depression and obesity include severity of depression, severity of obesity, gender, socioeconomic status, gene-environment status, and adverse childhood experiences, while mediating variables include eating behaviors (including disordered eating), physical activity, teasing, and stress [19].

New findings have highlighted the mediating roles of biological factors such as systemic inflammation and obesity-related morbidity [18], as well as the contribution of systemic inflammation [20] and obesogenic medication [12], in the association between depression and obesity. This review focuses largely on the psychological and behavioral constructs which potentially drive the relationship between depression and obesity. For additional review of the biological links of importance in this interaction, please see the

works of groups lead by Milaneschi [21] and Schweinfurth [22].

Emotional Eating

Kontinen [23] recently reviewed studies examining emotional eating, as eating behaviors, including disordered eating, have been previously identified as a mediating factor between depression and obesity. Based on the literature, emotional eating is defined as “a tendency to eat in response to negative emotions with the chosen foods being primarily energy-dense and highly palatable ones.” The article summarizes multiple cross-sectional and prospective studies which establish emotional eating as a mediator by which depression leads to weight gain and increases in both BMI and waist circumference. In particular, the author highlights that this effect is especially prominent among women [24].

Maternal Warmth and Conscientiousness

As adverse childhood experiences are an established moderator in the depression-obesity relationship, Gerhardt, Feng, and Chan [25] sought to examine the mechanisms by which parental warmth may impact the development of both depression and obesity over the lifespan. Using the Midlife in the United States (MIDUS) national longitudinal data set, the authors tested a moderated mediation model to conclude that trait conscientiousness (collected at T2 via self-report of adults aged 25–75) mediated the beneficial effect of early maternal warmth (collected at T1 as perceived by adult participants based on their recollection of growing up) on development of both depression and obesity (collected at T3, with each time interval being 10 years) and the effect varied by age. The authors interpret this finding to mean that maternal warmth may augment a child’s conscientiousness, thereby reducing levels of both depression and obesity in adulthood.

Weight Bias Internalization

In a systematic review of the literature regarding weight bias internalization, Pearl and Puhl [26] define this construct as “the internalization of negative weight stereotypes and subsequent self-disparagement.” The authors assert that while several reviews and meta-analyses have established that experience of weight discrimination and stigma are pervasive and detrimental to health, less is known about the prevalence and impact of weight bias internalization. Weight discrimination and stigma, similar to teasing, may mediate the relationship between depression and obesity [26]. As treatment and patient behaviors may act on weight bias internalization more readily than discrimination, stigma, and teasing, it stands to reason that weight bias internalization

may be an important treatment target for behavioral interventions treating co-occurring depression and obesity.

Differences by Gender, Race, and Depressive Presentation

Silva, Coutinho, Ferriani, and Viana [27] found, in their meta-analysis of eight studies, that patients with atypical depression (symptom profile including qualities such as mood reactivity, hypersomnia, leaden paralysis, and interpersonal rejection sensitivity) had significantly higher BMI on average compared to patients with melancholic depression.

Similarly, Blasco, Garcia-Jimenez, Bodoano, and Gutierrez-Rojaz [6] concluded that the presence of atypical depression increases the risk for development of obesity, particularly among African-American adolescent males. The authors also posited that obesity is a risk factor for recurrent depressive disorder among women.

Behavioral Treatment

While obesity treatment is not itself an evidenced-based treatment for depression, combination cognitive behavioral therapies (CBT) for depression and behavioral lifestyle treatment for weight loss has been shown to further enhance mood outcomes but not necessarily boost weight loss outcomes [28, 29]. Furthermore, recent review of seven randomized control trials suggests that not only is there benefit to integrating treatment for depression and weight loss intervention, but that a sequential approach focusing first on depression may be especially advantageous in terms of overall outcomes and engagement [30].

Suicidality

Suicide, or the act of taking one’s own life voluntarily and intentionally, is a complex global public health burden that is associated with psychiatric and medical illnesses. Recurrent suicidal ideation, with or without a specific plan or attempt to take one’s life, is a serious symptom within those that comprise the diagnostic criteria for depression [14]. Common risk factors for suicide that coincide with obesity include alcohol and other substance use disorders, impulsivity, history of trauma or abuse, major physical illnesses, lack of social support and a sense of isolation, stigma associated with asking for help, and lack of healthcare [31].

Depression in Patients with Obesity Seeking Metabolic and Bariatric Surgery (MBS)

Psychiatric comorbidities are prevalent among MBS patients, with estimates ranging from 23 to 68% of patients affected preoperatively [32, 33]. Depression is prevalent in bariatric surgery candidates with approximately 40% of patients reporting pre-operative treatment for depression [32, 34] and 11.6% currently meeting criteria for an affective disorder. Prevalence of depression has also been shown to increase further as class of obesity increases. However, these may be an underestimate as patients seeking bariatric surgery may also under-report psychopathology due to social desirability and wish to be “cleared for surgery.”

Initial improvements in depression are typically observed within the first 6 to 12 months following MBS. These findings suggest that over time, some patients may return to baseline levels of depression, underscoring the importance of continuing to monitor MBS patients’ mood and adjustment long-term [34].

Despite the high rate of comorbidity between mood disorders and obesity, unstable depression, suicidal ideation, and recent suicide gestures, attempt, hospitalization are often considered contraindications for MBS. The 2019 AACE/TOS/ASMBS Guidelines [35] asserted that potential contraindications to MBS include “substance abuse or poorly controlled psychiatric illness.” While the relative risk for suicide attempt is higher among individuals who have

undergone MBS compared to those who have not undergone MBS [36], for the majority of MBS programs, patients who pursue and engage in treatment to effectively manage or improve their symptoms of depression may be appropriately referred for MBS.

Sample Guidelines: see Fig. 1. Depression & Suicidality Assessment and Recommendations.

- Patients presenting with obesity should be assessed for depression using a validated and generalizable measure such as the BDI-II, PHQ-9, etc. and/or a semi-structured clinical interview which includes the DSM-V-TR criteria for depression.
- Assessment of suicidal ideation is recommended for patients presenting with depressive symptoms. If active ideation (e.g., planning thoughts or intent) is present, it is recommended that providers complete/document a thorough assessment of risk and protective factors (e.g., VA Suicide Risk Assessment Guide: Reference Manual; informed by APA 2004 and Rudd et al., 2006) to determine the appropriate level of care (e.g., creating a safety plan, involving patient’s supports such as family and medical providers, and/or engaging in established institutional imminent risk protocol).
- It is recommended that establishing psychiatric/psychotherapeutic care is prioritized for patients with moderate to severe depressive symptoms, who are seeking treatment for obesity.

Depression/Suicidality Assessment and Recommendations

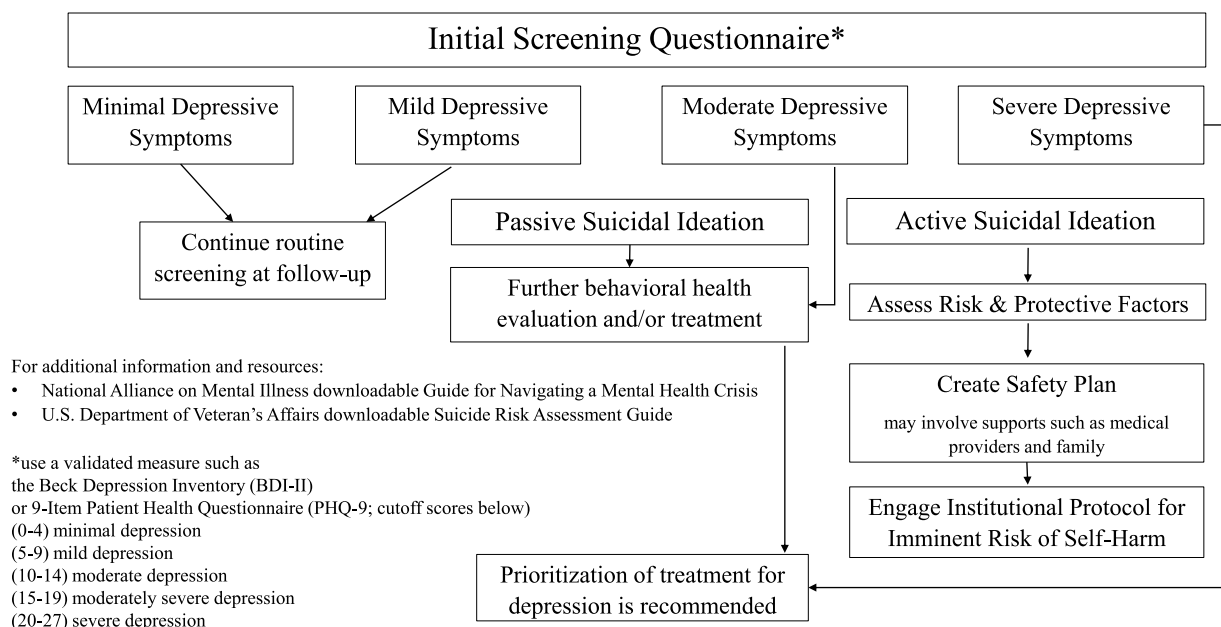


Fig. 1 Note: Suggested flowchart for screening, assessing, and treating depression and suicidality in the context of obesity

- Medical providers treating depression in individuals with obesity should avoid obesogenic medication (e.g., amitriptyline, dosulepin, doxepin, imipramine, paroxetine, citalopram, venlafaxine, isocarboxazid, phenelzine, mirtazapine, brexpiprazole, and trazodone) and consider antidepressants which may contribute to weight loss (e.g., Bupropion and Fluoxetine).
- Cognitive behavioral therapy, especially those focusing on increasing emotion regulation, facilitating conscientiousness, and decreasing weight bias internalization, may be particularly effective for treating comorbid depression and obesity.

Conclusion

Obesity and depression are highly prevalent, often co-occurring, and negatively impact morbidity, mortality, and quality of life. This brief limited review summarized recent findings regarding the interaction of depression and obesity. While a reciprocal and bi-directional link between obesity and depression has long been established, evidence suggests that obesity more strongly contributes to development of depression versus depression contributing to the development of obesity. In addition to previously identified moderators (e.g., severity of depression, severity of obesity, gender, socioeconomic status, gene-environment status, and adverse childhood experiences) and mediators (e.g., include eating behaviors, physical activity, teasing, and stress) in the relationship between depression and obesity, systemic inflammation, obesity-related morbidity, and obesogenic medication may be important new mediating factors. Moreover, emotion regulation, conscientiousness, and weight bias internalization may be important treatment targets when addressing comorbid depression and obesity using cognitive behavioral therapies. Also, when treating comorbid depression and obesity, there may be benefit to addressing depressive symptoms prior to obesity in order to facilitate initial engagement and support improved outcomes overall. Adequate screening, assessment, and ongoing monitoring and treatment may help mitigate the risk for worsening depression and suicide in individuals with obesity. Future research should examine the efficacy of such risk management strategies.

Author Contributions G.G. wrote the main manuscript text and prepared Fig. 1. N.P. and K.G. contributed to portions of the written manuscript. All authors reviewed the manuscript.

Data Availability No datasets were generated or analysed during the current study.

Declarations

Competing Interests The authors declare no competing interests.

Human and Animal Rights and Informed Consent All reported studies/experiments with human or animal subjects performed by the authors have been previously published and complied with all applicable ethical standards (including the Helsinki declaration and its amendments, institutional/national research committee standards, and international/national/institutional guidelines).

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