

Depression and Obesity: A Literature Review

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OUTLINE

I.	Abstract.....	3
II.	Introduction.....	4
III.	Theme 1.....	5
	- The trend in the association between depression and obesity.	
IV.	Theme 2.....	6
	- Obesity leading to later development of depression.	
V.	Theme 3.....	8
	- Depression leading to later development of obesity.	
VI.	Conclusion.....	10
VII.	References.....	12

ABSTRACT

The purpose of this paper is to review past research literature regarding the relationship between depression and obesity. Three common themes found in past research are discussed in depth, these include: investigating the trend in the association between depression and obesity, if obesity can lead to later development of depression, and whether depression can lead to later development of obesity. After analyzing and synthesizing past research results, new associations were discovered: there is a u-shaped trend between BMI and depression, and proper categorical BMI should be used regarding this relationship; there is a possible causal relationship between obesity and subsequent depression, especially in boys; and there is a possible causal relationship between depression and subsequent obesity, especially in girls. In conclusion, opportunities for further research were discovered. Future studies need to investigate the exact causal relationships between obesity and depression, the gender interaction on risk factors, and possible ethnic variations in depression and BMI. Further understanding the exact relationship between depression and obesity could have important clinical implications in terms of treatment and prevention; thus, decreasing the high prevalence of their comorbidity.

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INTRODUCTION

An estimated 10% of American adults suffer from depression; notably, 4.5% have been diagnosed with major depressive disorder (CDC, 2010). According to the DSM-5, major depressive disorder is a type of mood disorder that is characterized by at least two weeks of a major depressive episode (American Psychiatric Association, 2013). These episodes consist of persistent and pervasive symptoms that interfere with daily life; specifically, these episodes are characterized by depressed mood and loss of interest, among other cognitive and affective symptoms.

Interestingly, depression has a high comorbidity rate with obesity. Obesity is caused by the accumulation of excess body fat. The World Health Organization defines obesity as having a body mass index, (BMI), over 30; whereas, normal BMI is 18-24.99. BMI is calculated by dividing weight in kilograms by height in meters squared. Obesity in the United States is extensive, surpassing 30% in most gender and age groups (Flegal, 2010). Major depressive disorder and obesity have a high comorbidity rate due to various factors such as similar etiology and mechanisms of development.

Both obesity and depression have similar etiologies; particularly, their promotion and development is influenced by genetic and environmental factors, including socio-demographic factors. It is necessary to establish the exact association between depression and obesity, before causal pathways can be further investigated (De Wit et al., 2009). Once the underlying mechanism(s) of this relationship is/are established, causal pathways and treatment options can be studied to help reduce the prevalence of these comorbid disorders.

The author of this paper has identified three common themes in past research on the relationship between depression and obesity. These themes include:

1. The trend in the association between depression and obesity.
2. Obesity leading to later development of depression.
3. Depression leading to later development of obesity.

In the present paper, these themes will be discussed in depth by reviewing past research literature. With the objective of evaluating past research, discrepancies within these studies can be discovered as well as, opportunities for future research in this field of study.

THEME 1

The trend in the association between depression and obesity.

Only a positive association, negative association, or no association between depression and obesity had been studied until De Wit et al. (2009) studied the possibility of a u-shaped association between depression and body mass index. According to the DSM-5, changes in eating patterns (eating too much or too little) and physical activity (increase or decrease) both constitute core symptoms of major depressive disorder (APA, 2013). Assuming that eating patterns and physical activity can increase as well as decrease, De Wit et al. (2009) hypothesized a u-curved association in which people who are underweight and overweight/obese report more depressive symptoms, compared to people of normal weight.

Using a random, representative sample of 44,374 participants from The Netherlands, the criteria for this study were included in the annual Survey of Living Conditions by the Statistics Netherlands. The data included self-reported height and weight to calculate each participant's body mass index (BMI), and a Mental Health Inventory (MHI), which assesses psychological health. The inverse scores of the MHI were used as an indicator for the level of depressive symptomatology. Also, the World Health Organization's standards for BMI were used to separate the participants into appropriate categories: underweight (< 18.5), normal weight (18.5-24.9), overweight (25-30), and obese > 30. This is the first study related to depression to include all four categorical BMI's.

As a result, De Wit et al. (2009) found a highly significant u-shaped association between depression and BMI, which supported their hypothesis. Interestingly, the underweight group had the highest level of depressive symptoms, followed by the obese group. The implications of this study are extremely important in understanding the mechanism between depression and obesity. Results demonstrate that it is necessary to properly define categories of BMI when conducting research regarding the association with depression. Simply grouping normal weight as having a BMI less than 24.9 and overweight as over 25, (which was a trend before this study), will skew the data due to underweight and obese persons being included in these brackets.

Some limitations of this study include the study design, and the use of the MHI and self-reported BMI. First of all, the study design is cross-sectional and only assesses the participants at one point in time. As both depression and obesity occur at differing magnitudes throughout life, a longitudinal study would be more accurate. Second, the sample in this study consisted primarily of Caucasians from The Netherlands. Including other races and ethnicities would create a more representative sample with possibly more generalizable results. Third, the Mental Health Inventory is a valid instrument to assess depressive symptoms, but it is not a valid diagnostic tool for depressive disorders. Fourth, in this study, height and weight measurements were self-

reported. This can possibly skew the BMI data because most people tend to over-estimate their height and under-estimate their weight. However, self-reported measurements can still be accurate and valid to use in research. A study conducted by Fonseca et al. (2010) determined that self-reported measurements are valid in epidemiological studies concerning the overweight and obese BMI categories.

Lastly, using the BMI as an indicator for adiposity may have been a limitation. While the World Health Organization's standards for categorical BMI are applicable to the general population, misclassification still occurs- although it is highly uncommon. Some persons that are overweight do not have excess body fat, (for example, body-builders). Others may have a normal BMI, but have a high percentage of fat relative to their weight. Despite these rare misclassifications, BMI is one of the best indicators of adiposity because no standard for correctly evaluating body fat ranges exists. All limitations considered, the results and implications of this study are still very important to the field of depression and obesity research. Moreover, the results stress the importance of implementing categorical BMI into research studies and establishing the exact u-shaped trend between depression and BMI.

THEME 2

Obesity leading to later development of depression.

There is limited information regarding the association between childhood obesity and particular types of psychiatric disorder (Mustillo et al., 2003). This is the basis for the study conducted by Mustillo et al. (2003) to examine the relationship between types of childhood/adolescent obesity and later developing psychopathologies, including depressive disorders. Two of the goals of this study were to determine distinct obesity trajectories in a representative sample of 991 Caucasian children ages 9 to 16, and to test the relationship between obesity trajectory membership and psychopathology trajectory- specifically depression for the purposes of this review (Mustillo et al., 2003).

Collecting the data consisted of two interviewers visiting the participants' home once a year to measure the child's height and weight and administer the Child and Adolescent Psychiatric Assessment (CAPA) to the child and a parent separately. The World Health Organization standards for BMI do not apply to children. In this study, growth charts from the Center for Disease Control and Prevention were used to define obesity in children. Age- and sex-specific 95th percentiles were the specification for childhood obesity (Mustillo et al., 2003). In addition to assessing if the child was obese, the CAPA was given to the child and parent separately to determine the presence of psychiatric symptoms according to the DSM IV. A symptom was considered present if reported in at least one of the assessments by the parent or child. These interviews measuring obesity and psychiatric symptoms were conducted annually for 8 years.

For the purposes of this study, 4 trajectories/categories of obesity were defined: 1) children who were never or rarely obese, 2) children who started in the normal weight range but became obese over time (adolescent obesity), 3) chronically obese children, 4) children who were obese and whose weight dropped over time (childhood obesity). As a result, this study found that of these four weight trajectories only chronic obesity was associated with a statistically significant increase in the risk of psychiatric disorders, followed by the childhood obesity group (Mustillo et al., 2003). Specifically, chronically obese boys had higher rates for depression. As a speculative conclusion, these results could be suggestive that obesity increases the risk of psychopathology- including depressive disorders.

Interestingly, the childhood-limited obesity group, who became normal weight during adolescence, had high risks of developing psychopathologies. This begs the question- Why is their risk for psychopathology higher than the group that developed adolescent obesity? A study conducted by Levy and Pilver in 2012, helps to answer this question. They examined the psychological state of individuals who have transitioned from overweight to normal weight. Although formerly overweight individuals have made a socially approved transition, established effects from prior membership in a stigmatized group would continue to create psychological distress (Levy & Pilver, 2012). They hypothesized that formerly overweight individuals would be significantly more susceptible to any anxiety disorder, depressive disorder, and suicide attempt than the consistently normal weight group, but not significantly different from the consistently overweight group.

The sample for this study included 20,649 participants with a mean age of 49, and were recruited to reflect the demographics of the 2000 U.S Census in terms of race, ethnicity, sex, and socioeconomic status (Levy & Pilver, 2012). To assess childhood weight, the participant was asked if they were overweight before the age of thirteen. Current weight was assessed using body mass index (BMI); specifically, a participant was overweight if they had a BMI over 25 or normal weight if they had a BMI less than 25.

To assess psychiatric health, each participant responded to the National Epidemiologic Survey on Alcohol and Related Conditions. This survey evaluates many parameters of psychiatric health including the presence of anxiety or depressive disorders according to the criteria in the DSM-IV. Specifically, the presence of any anxiety disorders, depressive disorders, and suicide attempts were used to compare each weight group within this study. In conclusion, this study identified that formerly overweight participants had a significantly higher risk of any anxiety disorder, any depressive disorder, and suicide attempts than the consistently normal weight participants (Levy & Pilver, 2012). Furthermore, the risks of the formerly overweight group and the consistently overweight group were not significantly different. (Levy & Pilver, 2012).

The implications of these results are profound. Although a person may physically lose weight and transition from being overweight to the normal weight bracket, their self-image may remain constant and they may continue to define themselves as being part of a stigmatized group. This residual stigma of being overweight/obese can create psychological distress and greatly increase the risk of anxiety and depressive disorders.

The studies conducted by Mustillo et al. (2003) and Levy and Pilver (2012) provide more information regarding the relationship between obesity and depression. Mustillo et al. (2003) determined that chronically obese children had higher risk for expressing psychiatric symptoms or being diagnosed with psychiatric disorders; more specifically, chronically obese boys had higher risk for depression. Levy and Pilver (2012) confirmed that formerly overweight persons had a significantly higher risk for any depressive disorder than normal weight persons. Furthermore, there was not a significant difference between formerly overweight persons and consistently overweight persons for the risk of any depressive disorder. Although highly speculative, these studies seem to indicate a possible causal relationship between obesity and the later development of depression.

Even though the results of these studies are significant, there are some limitations to the study designs. In the research by Mustillo et al. (2003), the participants were mostly Caucasian, rural children from North Carolina. Including other races and ethnicities would create a more representative sample with possibly more comprehensive results. In the analysis by Levy and Pilver (2012), only two BMI categories were used to classify the participants: overweight was a BMI over 25 and normal weight under 25. As previously discussed, the study by De Wit et al. (2009), demonstrated the importance of using proper categorical BMI's in research. The results of Levy and Pilver's (2012) study could be greatly skewed due to any underweight or obese persons being placed in the wrong BMI category. Also, Levy and Pilver (2012) asked the participants if they were overweight before age 13. Without any accurate measurements from childhood height and weight, relying on a participant's recall threatens the validity of childhood weight data. Although these studies had several limitations, Mustillo et al. (2003) and Levy and Pilver (2012) helped to increase knowledge regarding the relationship between obesity in early life and later development of depression.

THEME 3

Depression leading to later development of obesity.

Depression has been suspected to be a risk factor for obesity in adults; although, longitudinal studies have had conflicting results regarding weight gain in depressed adults. Both depression and obesity have higher rates of occurring from adolescence to young adulthood (Richardson et al., 2003). Considering the increased prevalence during this developmental period, Richardson et al. (2003), performed a study to better understand the interaction between depression and obesity from adolescence to young adulthood.

Psychomotor retardation, the slowing-down of thoughts and physical movement, has been shown to increase over time in depressed youth (Carlson & Kashani, 1988). Richardson et al. (2003) hypothesized that the increase in psychomotor retardation over time in depressed youth would lead to a positive association between depression in late adolescence and the development of obesity in young adulthood. Childhood and early adolescent depression were not hypothesized to have a positive relationship with young adulthood obesity. Depressed younger children have a higher risk of agitation and anxiety, and lower risk of psychomotor retardation compared to depressed youth (Richardson et al. 2003).

The data for Richardson et al.'s (2003) study was acquired from the Dunedin Multidisciplinary Health and Development Study, or DMHDS. Participants underwent an evaluation every 2 to 3 years starting at the age of 3 up until age 21, and then again at 26. Richardson et al.'s study used all data from the DMHDS for participants who had a mental health interview any time from 11 to 21 years of age and who had never been pregnant. The final sample was primarily Caucasian and consisted of 881 participants for the investigation of early adolescent depression and young adult obesity, and 884 participants for the investigation of late adolescent depression and young adult obesity.

To diagnose depression, mental health interviews were administered to each participant following the DSM-III criteria for major depression. Early adolescence was defined as ages 11, 13, or 15, and late adolescence was 18 or 21. Obesity in adulthood was measured at age 26 and defined by having a BMI of over 30. After evaluating these parameters every 2 to 3 years, a positive association between late adolescent depression and young adult obesity was found only in girls. However, there was a negative association for late adolescent depression and subsequent obesity in boys, and a negative association for early adolescent depression and subsequent obesity in all participants.

Furthermore, girls that met the criteria for being depressed during late adolescence were more than twice as likely to develop obesity at age 26 than girls who did not experience late adolescent depression (Richardson et al., 2003). This interaction between gender and depression as risk factors for obesity was observed in a

subsequent study conducted by Hasler et al. (2005). The longitudinal study on BMI and depression administered by Hasler et al. (2005) was the first to continue collecting data from childhood into middle age. Based on prior studies, they tested the hypothesis that depression during childhood increases risk for weight gain and obesity in adulthood.

Participants were acquired from a cohort study of young adults resulting in a total sample of 591 primarily Caucasian members. Between the ages of 19 and 40, participants were screened for depression and BMI every 3-5 years for a total of 6 assessments each. To diagnose depression, the Structured Psychopathological Interview and Rating of the Social Consequences for Epidemiology (SPIKE) was administered which assesses symptoms of major depression according to DSM-IV-TR criteria. Subjects were asked about the existence of past depressive symptoms and when they first occurred. Childhood depressive symptoms were defined as occurring before age 17. Obesity was defined as having a BMI over 30.

Based on these measures, a significant positive association was found between childhood depressive symptoms and adult BMI. More specifically, childhood depressive symptoms were associated with increased occurrence of adult obesity in women. These results reinforce the study conducted by Richardson et al. (2003) that found a positive association between late adolescent depression and young adult obesity in women. It is apparent that there is a gender interaction regarding depression as a risk factor for obesity. Even still, Hasler et al. (2005) found that childhood depressive symptoms increased adult weight overall. Theoretically, a causal relationship might exist between depression and the later development of obesity.

Furthermore, not only do these studies have significant results and important implications, they have few limitations to their study designs. Both Richardson et al. (2003) and Hasler et al. (2005) had primarily Caucasian participants in their studies. To create a more representative sample with more generalizable results, other races and ethnicities should have been included. Also, Hasler et al. (2005) asked the participants if any depressive symptoms occurred before the age of 17. Without any accurate measurements of childhood depressive symptoms or diagnoses of depressive disorders from childhood, relying on a participant's recall threatens the validity of the childhood depressive symptoms data. Considering the limitations, both studies still have significant results and have helped to establish knowledge concerning the relationship between depression and later development of obesity.

CONCLUSION

Evaluating past research regarding depression and obesity, has revealed three main associations:

1. There is a u-shaped trend between BMI and depression, and proper categorical BMI should be used in research regarding this relationship.
2. There is a possible causal relationship between obesity and subsequent depression, especially in boys.
3. There is a possible causal relationship between depression and subsequent obesity, especially in girls.

Synthesizing these implications can create questions for future research. For example, why is there a significant sex interaction in obesity being a risk factor for depression, and vice versa? To answer this question, comparative studies need to be conducted that examine the causal relationships between obesity and depression across genders. Also, most of the studies reviewed in this paper used primarily Caucasian participants (De Wit et al., 2009; Mustillo et al., 2003; Richardson et al., 2003; Hasler et al., 2005). Conducting a comparative study including other races and ethnicities would create a more representative sample with possibly more generalizable results. Also, it could reveal any possible ethnic differences in the relationship between BMI and depression.

A study conducted by Hicken et al. (2013) observed ethnic differences in the association between obesity and depression in adult women. This study determined a positive association in Caucasian women, no association in Mexican American women, and results suggested obesity was inversely associated with depression in African American women (Hicken et al., 2013). These results indicate ethnic differences in the association between obesity and depression; however, this study only included adult women. Further research in this area needs to include males, as well as, different age groups.

In conclusion, after evaluating and synthesizing past research, ambiguities have been discovered. Future studies need to investigate the exact causal relationships between obesity and depression, the gender interaction on risk factors, and possible ethnic variations in depression and BMI. More specifically, this could be achieved via a longitudinal study that includes males and females, various races and cultures, and proper categorical BMI to classify participants. Being that both obesity and depression are treatable and preventable disorders, understanding their exact causal relationship could have important clinical implications in their treatment and prevention. Also as a result of future research, the high prevalence of comorbid obesity and depression in the United States could be vastly reduced.

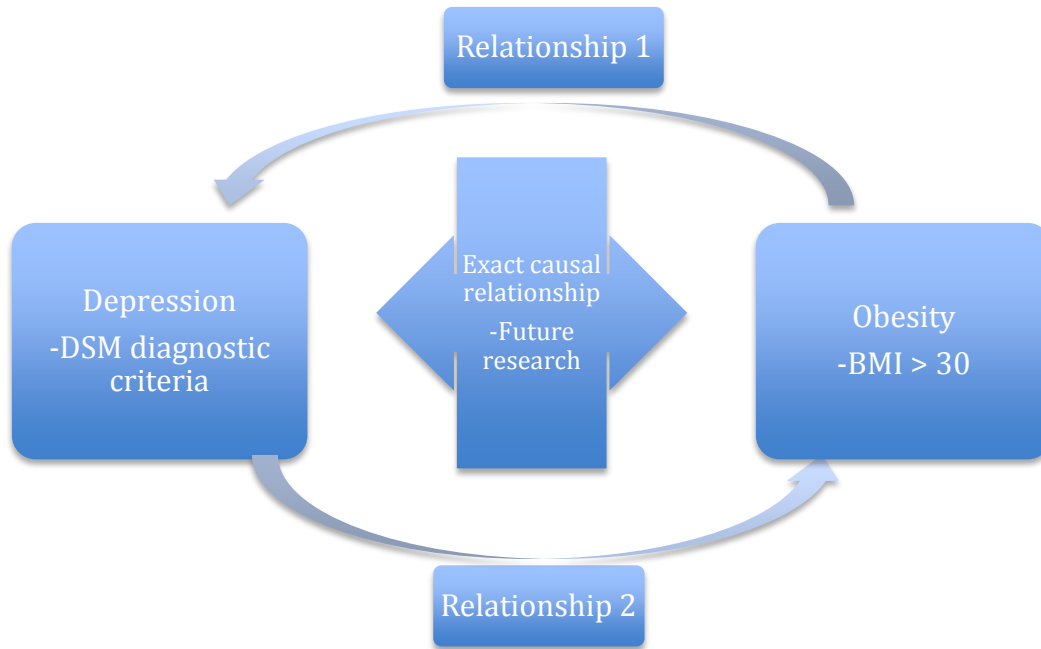


Figure 1. Schematic model of the relationship between depression and obesity adapted from a literature review of past research. Relationship 1 is a possible causal relationship between obesity and subsequent depression, especially in boys, (Mustillo et al., 2003; Levy & Pilver, 2012). Relationship 2 is a possible causal relationship between depression and subsequent obesity, especially in girls, (Richardson et al., 2003; Hasler et al., 2005). Knowing the exact causal relationship requires proper future research in this field (*elaborated above in conclusion).

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