

Understanding the Relation Between Obesity and Depression: Causal Mechanisms and Implications for Treatment

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Obesity is a serious and prevalent condition, with grave risks for morbidity and mortality. While the physical consequences of obesity have been well studied, the psychological correlates are less well understood. Theory and research suggest that obesity and depression may be causally linked. We propose a bidirectional theoretical model identifying the behavioral, cognitive, physiological, and social mechanisms that may be responsible for the pathway between obesity and depression, and vice versa. We investigate the research that supports this model, and identify areas of need for future research. In addition, we discuss the clinical implications of this literature, including the need for integrated care in this population.

Key words: depression, mechanisms, obesity, risk factors. [*Clin Psychol Sci Prac* 15: 1–20, 2008]

Obesity has become a serious public health problem, with approximately 32.2% of Americans classified as obese (Ogden et al., 2006). Obesity has been found to be associated with negative health outcomes (Must et al., 1999; Pi-Sunyer, 1993), functional impairment (Fontaine & Barofsky, 2001), and increased mortality (Allison, Fontaine, Manson, Stevens, & VanItallie, 1999). Similarly,

depression is one of the most serious mental health problems in the country with substantial consequences of human suffering, loss of life, and lost productivity (Klerman, 1989; Klerman & Weismann, 1992). Evidence suggests that individuals with major depression, or just elevated symptoms of depression, are also likely to experience impairments in multiple domains, including employment and physical and social functioning (Judd et al., 2000; Keitner & Miller, 1990; Wells et al., 1989; Whisman & Bruche, 1999). Individuals who suffer both obesity and depression may face particular risks to health and well-being. Not only may the presence of both conditions increase risk for loss of function, but also these conditions may perpetuate one another; obesity may increase risk for depression; and depression may promote obesity. Understanding and managing the mechanisms that link obesity and depression is crucial to the treatment of individuals who suffer from both conditions.

In an earlier review of the literature, Friedman and Brownell (1995) suggest that because of the heterogeneity of the obese population, it is not possible to generalize the relation between obesity and a psychological factor, such as depression. They suggest that in order to best understand depression among obese individuals, it is important to consider who suffers and, ultimately, the mechanisms that link these conditions. In order to understand the potential causal links between these conditions, we will first briefly examine the correlational data suggesting a link between obesity and depression, as well as potential moderators of this effect. Then, in order to elucidate these causal mechanisms, we will first discuss

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the ways by which obesity might contribute to depression, and then we will discuss the mechanisms by which depression contributes to obesity, with suggestions for areas for future study. Finally, we will discuss the future areas of research to test the model and further clarify the pathways by which depression and obesity influence each other.

Although primarily interested in the link between obesity and major depressive disorder (MDD), this review will include studies examining symptoms of depression and measures of mood in addition to those assessing MDD diagnostically. This decision will not only allow us to draw from a wider range of studies to inform our review, but also is consistent with evidence that even elevated symptoms of depression that do not reach the level of MDD can be associated with loss of functioning (Judd, Akiskal, & Paulus, 1997; Kessler, Zhao, Blazer, & Swartz, 1997). Furthermore, while there has been a call for more rigorous methodology to assess depression, including multiple assessment periods, multiple assessment methods, and interview instead of self-report when possible (Kendall & Flannery-Schroeder, 1995; Kendall, Hollon, Beck, Hammen, & Ingram, 1987; Tennen, Hall, & Affleck, 1995), few of the studies examined here meet all of these criteria, but particular strengths and limitations of each study are noted.

EVIDENCE OF A CROSS-SECTIONAL ASSOCIATION BETWEEN OBESITY AND DEPRESSION

Cross-sectional research has asked the question whether the obese individuals are more likely to be depressed than the nonobese by comparing samples of obese individuals to samples of nonobese individuals on measures of psychological functioning. In a review of the literature, Friedman and Brownell (1995) found conflicting results, due to methodological limitations of subject selection and measurement, and the heterogeneity of the population of obese individuals. Since Friedman and Brownell's (1995) review, more published studies have found evidence of an association between obesity and depression.

For example, Johnston, Johnson, McLeod, and Johnston (2004) analyzed data from the 1995 Nova Scotia Health Survey to examine the relation between body mass index (BMI) determined by measurement of height and weight and depression assessed with the Center for Epidemiologic Studies Depression (CES-D) scale. BMI

category was significantly related to increased risk for depression, and the odds ratio for the association between obesity and depression, after controlling for education and income, was 1.41 (Johnston et al., 2004). Although use of structured interview would enhance these findings, this study has the strengths of a large sample size and clinically measured height and weight. In another study, researchers examined a large cluster sample of Swedish men and found that self-reported BMI was associated with increased symptoms of depression on a study-specific self-report questionnaire (Rosmond, Lapidus, Marin, & Bjorntorp, 1996). Although this study is strengthened by a large sample size, it is limited by reliance on self-report for BMI and depressive symptoms.

One study examined a large cluster sample of young German women and found that obesity determined by self-reported height and weight was associated with increased affective disorders determined by structured interview (Becker, Margraf, Turke, Soeder, & Neumer, 2001). In addition to a large sample size, the strength of this study is its use of clinical interview, although it would have been helpful for the authors to report the particular diagnosis instead of grouping all affective disorders together.

Overall, these findings are suggestive, but not conclusive, that obesity is associated with depressive symptoms. When taken in the context of previous studies in the field that have found mixed or no results, there is still considerable question as to whether one can conceptualize the obese population as a homogeneous group with a consistent relation to depression. As a result, Friedman and Brownell (1995) suggest that it is more appropriate to examine which obese individuals suffer depression as this can help us identify potential causal mechanisms.

WHICH OBESSE INDIVIDUALS ARE MOST LIKELY TO SUFFER

The literature suggests that possible risks for comorbid obesity and depression include severe obesity, female gender, and high socioeconomic status (SES). These factors may not cause either obesity or depression, but their presence may cause certain obese individuals to be at greater likelihood for developing depression. Ideally, these studies would examine populations of obese individuals to determine which variables place them at greater risk for depression, but we consider any study that may suggest which obese individuals are at risk for depression

here, even if it was not based on population samples. Several reviewers have suggested that severe obesity puts individuals at greater risk for depression (Fabricatore & Wadden, 2004; Friedman & Brownell, 1995; Stunkard, Faith, & Allison, 2003). The Third National Health and Nutrition Examination Survey (NHANES-III) examined a large sample of men and women and found heterogeneity of prevalence of MDD, defined by the operational criteria of the *Diagnostic and Statistical Manual of Mental Disorders, Third Edition (DSM-III)* and assessed by the Diagnostic Interview Schedule (DIS), across classifications of obesity, such that the most severely obese individuals (BMI > 40) were significantly more likely to be depressed than those with BMI between 30 and 34.9 (12.51% versus 3.55%; Onyike, Crum, Lee, Lykestos, & Eaton, 2003). This is particularly strong evidence, as this study had a large sample size, assessed diagnoses of MDD with a structured interview, and measured height and weight instead of relying on self-report.

Multiple reviewers have suggested female gender as a potential risk factor for depression among obese individuals (Fabricatore & Wadden, 2004; Friedman & Brownell, 1995; McElroy et al., 2004; Stunkard et al., 2003). Carpenter, Hasin, Allison, and Faith (2000) analyzed data from 40,086 African American and Caucasian adults nationwide. Subjects self-reported height and weight and were assessed for MDD with the Alcohol Use and Associated Disabilities Interview Schedule (AUADIS). Obesity was associated with increased likelihood of MDD, defined by the operational criteria of *DSM-IV*, in the past year among women (4.7% for obese versus 3.6% of average weight), but not among men (1.2% for obese versus 2.9% of average weight) (Carpenter et al., 2000). The strength of this study is its diagnostic assessment of MDD, but it is limited in that it groups normal weight (BMI between 20 and 24.9) and overweight (BMI between 25 and 29.9) participants in the same category, thereby possibly obscuring differences between obese and normal weight participants. In a community sample of adults aged 50–69 years, depression, operationalized by Beck Depression Inventory (BDI) score greater than 13, was associated with overweight and obesity at trend level among women (4.4% normal weight, 9.0% overweight, and 7.5% obese), but not among men (4.8% normal, 1.8% overweight, and 0% obese; Palinkas, Wingard, & Barrett-Connor, 1996). This study

is limited, however, by its narrow age criteria and reliance on the BDI as a diagnostic tool. Among the 2,853 participants in NHANES-I, BMI was positively but weakly correlated with depressive symptoms measured by the CES-D, among women ($r = +0.065$), but not among men ($r = -0.01$; Istvan, Zavela, & Weidner, 1992).

Socioeconomic status and level of education have been suggested as potentially important risk factors for depression in obese individuals (Faith, Matz, & Jorge, 2002; Stunkard et al., 2003), but there is some disagreement about the direction of this association. There is an association between low SES and depression, as well as an association between low SES and obesity, suggesting to some researchers that low SES is an important risk factor in comorbid depression and obesity (Everson, Maty, Lynch, & Kaplan, 2002; Johnston et al., 2004). Other research, however, has demonstrated that among obese individuals, higher SES is related to increased relative risk for depression. In one study of African American women seeking a weight loss and fitness intervention, being above the median for pounds overweight was unrelated to depressive symptoms assessed with the CES-D in those with fewer than 12 years of education, but being above the median for pounds overweight predicted depressive symptoms in those with at least some college education (14.79 versus 9.95 on CES-D; Siegel, Yancey, & McCarthy, 2000). In a large nationwide sample of men and women, among those with a high school education or less, self-reported overweight did not predict depressive symptoms on CES-D, but was significantly associated with depressive symptoms in those with at least some college education (Ross, 1994). Both of these studies are limited by the fact that they assessed depressive symptoms, rather than MDD, but it is helpful that both used the same measures of depressive symptoms and education category. While those of lower SES may be more likely to experience depression or obesity, among the obese, high SES may put one at increased risk for depression.

Overall, these findings suggest that severe obesity, female gender, and high SES may confer risk for depression in the obese. In order to evaluate the evidence of a possible causal relation between obesity and depression, we will examine two types of evidence: longitudinal predictive studies and intervention studies. Specifically, we suggest that if obesity and depression are causally related, then

the presence of obesity should predict depression at a later time, and vice versa. Furthermore, change in either variable through intervention should result in a change in the other (e.g., weight loss should predict mood improvement; improved mood should predict weight loss). We evaluate each causal link independently (i.e., obesity as a cause of depression, and depression as a cause of obesity) by examining both longitudinal and intervention studies, and also propose mechanisms that explain each of these causal links.

OBESITY AS A CAUSE OF DEPRESSION

Longitudinal Studies

Reports from one large data set suggest that obesity predicts later onset depression in adults. Roberts, Kaplan, Shema, and Strawbridge (2000) examined data from the Alameda County, California, community sample of middle-aged and older adults and found that obesity and MDD, assessed by self-report on a 12-item scale based on symptom criteria from *DSM-IV*, were associated cross-sectionally, such that 7.4% of normal weight participants were depressed, compared with 15.5% of obese participants. Even more important, however, was the finding that among those not already depressed, obesity at baseline predicted depression one year later (odds ratio [OR] = 1.91). When controlling for demographic and psychosocial variables, the ORs for obesity and depression were reduced slightly but remained statistically significant (OR = 1.73; Roberts et al., 2000). Roberts, Strawbridge, Deleger, and Kaplan (2002) reported that among the same sample, obesity at baseline predicted increased unhappiness (OR = 1.70), pessimism (OR = 1.59), low positive affect (OR = 1.46), life dissatisfaction (OR = 1.39), and depression (OR = 2.16) five years later. Even after controlling for baseline mental health indicators, the effect for depression (OR = 1.77) remained significant (Roberts et al., 2002). In 2003, Roberts, Deleger, Strawbridge, and Kaplan reported more data from this sample, indicating that obesity predicted depression five years later, even when controlling for depression at baseline, but that depression did not predict obesity prospectively when controlling for obesity at baseline. The OR for between obesity at baseline and depression five years later was 2.09; when eliminating participants who were depressed at baseline, the OR was 2.01, indicating that obese participants were twice as likely as the nonobese to

become depressed five years later (Roberts et al., 2003). When controlling for demographic, psychosocial, and health factors, the association remained significant (OR = 1.53). These studies are strengthened by the large sample size, diagnostic assessment of depression, and prospective analyses controlling for baseline variables, making them good evidence for the prospective link between obesity and depression.

Intervention Studies

There is a long history of research that examines whether obese individuals who experience weight loss, either through traditional dieting and exercise or through surgical treatment, also experience improvements in depression. While there is evidence that weight loss treatment has a positive effect on depression, it does not seem that this relation is mediated by weight loss per se. In a review of the literature on weight loss treatment and mood, Wing, Epstein, Marcus, and Kupfer (1984) suggested that mood improvements (found in 6 out of 10 studies) were related to active participation in treatment (Wing et al., 1984). Bryan and Tiggemann (2001) found that among a small sample of middle-aged women assigned to a weight loss group or wait-list control, those in the weight loss group had scores indicating more positive mood on the depression subscale profile of mood states, but that these scores were not related to or affected by actual amount of weight loss.

More recently in another study of obese women undergoing weight loss treatment and with average baseline mood within population norms, weight loss in the first three months was correlated with improved mood on the mood adjective checklist, but at two-year follow-up average mood was no better than baseline regardless of weight change (Karlsson et al., 1994). These studies have the important limitation of assessing mood, not MDD or depressive symptoms. Cognitive or behavioral therapy for weight loss improved BDI scores among a sample of overweight and obese women from pretreatment to one-year follow-up, especially among those receiving cognitive therapy, regardless of weight loss success (Nauta, Hospers, & Jansen, 2001). It seems that the experience of being involved in active treatment may have a substantial but temporary effect on mood, which is not related to actual weight loss and may decline on cessation of treatment.

Studies of patients undergoing gastric bypass surgery are not as confounded by participation in an intensive therapeutic weight loss intervention as those of participants in behavioral treatments are. Patients undergoing gastric bypass surgery do, however, often receive weight loss counseling or other psychological support, so it may not be possible to determine what ingredient of the treatment caused changes in depressive symptoms or mood. There is evidence that gastric bypass surgery ameliorates depression in addition to reducing weight, both immediately posttreatment and at six-month follow-up (Bocchieri, Meana, & Fisher, 2002). One study examined a sample of morbidly obese (BMI > 40 or BMI > 35 with obesity-associated medical condition) patients undergoing Roux-en-Y gastric bypass surgery, and found that BDI scores improved significantly from pretreatment (14.6) to posttreatment (8.2), which is clinically meaningful (Dymek, Le Grange, Neven, & Alverdy, 2001). There was also evidence that mood continued to improve among the two-thirds of patients who completed follow-up evaluations six months later (2.7). These results do not, however, control for baseline BMI and depressive symptoms. In another study of patients undergoing gastroplasty for major obesity (BMI > 33), there was significant improvement in depression scores on the hospital anxiety and depression (HAD) scale from preoperation (7.9) to six-month follow-up (2.2; Kincey, Neve, Soulsby, & Taylor, 1996). Presurgery BMI predicted weight loss postsurgery, but not postsurgery HAD scores, and presurgery HAD scores predicted both postsurgery weight loss and postsurgery HAD scores (Kincey et al., 1996). A major limitation to these studies is that the type of behavioral or counseling intervention, if any, accompanying the surgeries is unknown, so it is not possible to speculate regarding the cause of improvement in depressive symptomology. There are further limitations to the generalizability of these findings, as there were no control groups in these studies, and weight loss surgery is only indicated for the extremely obese.

The longitudinal data are suggestive that obesity predicts later depression. The evidence based on intervention studies suggests that participation in weight loss treatment improves mood, but that this relation may not be related to actual weight loss. Taken together, there is a reason to believe that obesity can contribute to depression, but the relation is not a simple one. To further elucidate this

causal pathway, we next examine the possible mechanisms from obesity to depression.

Proposed Mechanisms

We propose that behavioral mechanisms, such as functional impairment and repeated dieting, cognitive mechanisms, such as body image dissatisfaction (BID) and poor self-rated health, and social mechanisms, such as stigma, may all play a role in the pathway from obesity to depression. We propose two causal paths based on the moderators of the link between obesity and depression: a “health concern” pathway by which severely obese individuals may experience functional impairment and poor self-rated health, resulting in depression, as well as an “appearance concern” pathway, whereby women and those of high SES may be more vulnerable to depression through a pathway involving BID and dieting. The severely obese may also be vulnerable to this pathway. While few studies have directly examined these factors as mechanisms that link obesity to depression, we review the existing evidence to inform future work.

Health Concern Pathway

Functional Impairment. Obesity may have a depressogenic effect by interfering with functioning. Functional impairment and disability are associated with obesity and depression (Wells et al., 1989). Obese individuals report worse health-related quality of life (Ford, Moriarty, Zack, Mokdad, & Chapman, 2001; Kushner & Foster, 2000), with impairment increasing as degree of obesity increases (Kolotkin, Meter, & Williams, 2001), particularly with regard to physical functioning (Fontaine & Barofsky, 2001). Obesity is also associated with both upper and lower body disability (Ferraro, Su, Gretebeck, Black, & Badylak, 2002), which interferes with basic activities of daily life (ADL; Han, Tjihuis, Lean, & Seidell, 1998). Furthermore, obesity at age 30–49 is associated with increased odds of ADL limitations later in life, and fewer years of life free from ADL limitations after age 50 (Peeters, Bonneux, Nusselder, De Laet, & Barendregt, 2004). These limitations may also impact participation in regular physical activity, which has been found to be related to the development of depressive symptomology (Camacho, Roberts, Lazarus, Kaplan, & Cohen, 1991).

The increased rates of chronic disease and functional impairment among the obese may help explain part of

the pathway by which obesity contributes to depression in certain individuals. There is evidence suggesting that the symptoms associated with other chronic diseases interfere with behavioral functioning, resulting in depressed affect. Diabetic complications (e.g., retinopathy) were significantly associated with diabetes intrusiveness (i.e., the extent to which diabetes interfered with life functioning), and diabetes intrusiveness predicted depressive symptoms among a sample of mostly obese type II diabetics (Talbot, Nouwen, Gingras, Belanger, & Audet, 1999). Similar findings have been demonstrated among individuals with rheumatoid arthritis (Devins et al., 1993; Neugebauer, Katz, & Pasch, 2003) and cancer (Williamson & Schulz, 1995). With weight loss, we would expect improvement in symptoms that accompany obesity, such as pain and ADL impairment. There is also evidence that weight loss leads to improved self-rated health-related quality of life (Fontaine & Barofsky, 2001). This improvement in quality of life may lead to improved mood as well.

Self-Rated Health. There is now mounting evidence that obesity is associated with an increased risk for poorer perceived health, as well as an increase in the presence of chronic disease (Mansson & Merlo, 2001). Based on results of the NHANES-III study, it appears that the relationship between self-rated health and obesity is linear (Fontaine, Redden, Wang, Westfall, & Allison, 2003). Poor self-rated health may contribute to depression through cognitive mechanisms in that individuals who believe their health to be poor may also hold other related depressogenic beliefs. They may believe that they are unable to engage in certain activities, or that they will not be able to have a long and fulfilling life. These beliefs may be strong and long-standing, similar to the schemata of Beck's cognitive theory (1967, 1983), and thus easily activated and global, making them especially depressogenic. Furthermore, poor self-rated health is related to reduced behavioral functioning, which can contribute to feelings of depression.

Poorer self-rated health may also be determined by public recognition of and debate over the obesity crisis. Sensationalist reports of an "obesity epidemic" and the proclamations about the potentially "fatal" consequences of obesity may unintentionally and prematurely perpetuate the assumption that "obesity" is a condition that is

inherently and uniformly dangerous in terms of physical health (Carr & Friedman, 2005). While many consider the negative health consequences of obesity inevitable, the debate over the inevitability of health problems among obese Americans has not yet been resolved. Some researchers have suggested that the overwhelming alarm regarding obesity has more to do with cultural and political factors than a real threat to public health (Campos, Saguy, Ernsberger, Oliver, & Gaesser, 2006).

Appearance Concern Pathway

Stigma. Facing repeated discrimination and mistreatment, whether actual or merely perceived, can lead to lower self-esteem and increased negative affect (Kessler, Mickelson, & Williams, 1999). There is good evidence that obese individuals endure this type of treatment on a regular basis from strangers, acquaintances, and intimates (Carr & Friedman, 2005; Puhl & Brownell, 2003). Constant maltreatment is both a daily hassle that may negatively affect mood over time and a threat to one's self-concept, both of which can contribute to depression.

Multiple studies document that children, adults, and even healthcare professionals who work with obese persons hold negative attitudes toward them (Schwartz, Chambliss, Brownell, Blair, & Billington, 2003; Solovay, 2000). Consequently, overweight and obese persons may be subject to discrimination by peers, employers, and clinicians who hold antifat attitudes (Puhl & Brownell, 2003). Carr and Friedman (2005) examined data from 3,353 men and women in the National Survey of Midlife Development in the United States (MIDUS), assessed through study-specific questionnaires. Very obese subjects were significantly more likely to report that they had ever been treated rudely by the strangers, acquaintances, and professionals with whom they interact than normal weight individuals (Carr & Friedman, 2005). Although limited by reliance on self-report measures of height and weight, this study is useful in demonstrating the perceived discrimination experienced by the very obese on a daily basis. Actual and perceived mistreatment are associated with compromised self-esteem and heightened depressive symptoms (Kessler et al., 1999). Given the pervasive evidence that obese people are treated more negatively than normal weight persons, one might expect that such discriminatory experiences may account,

in part, for higher levels of depression among obese individuals. Moreover, given that obesity is more noticeable among severely obese individuals, it is possible that the stigma associated with being obese may be greater for these individuals, and that the associated psychological suffering may also be greater (Allon, 1982).

Body Image Dissatisfaction. Researchers have suggested that one mechanism by which obesity confers risk for depression is through BID (Friedman & Brownell, 1995). BID is linked to low self-esteem, which is linked to depression. Obese individuals are more likely to be dissatisfied with their body shape and size, and through the pathway of lower self-esteem, may thus be more likely to experience depression.

Body image dissatisfaction was related to depressive symptoms measured by the BDI and self-esteem measured by the Rosenberg self-esteem scale, and degree of obesity was correlated with BID in a sample of treatment-seeking men and women (Friedman, Reichman, Costanzo, & Musante, 2002). Degree of obesity was also correlated with depressive symptoms and self-esteem, and BID partially mediated the relation between BMI and depressive symptoms and self-esteem (Friedman et al., 2002). Another study compared treatment-seeking obese women with normal weight controls and found that BID was higher in obese women and was correlated with depressive symptoms on the BDI and lower self-esteem on the Rosenberg self-esteem scale (Sarwer, Wadden, & Foster, 1998). Some have suggested that childhood teasing may be a predictor of poor body image and depression in obese adults (Friedman & Brownell, 1995; Stunkard et al., 2003). Researchers have found that among obese treatment-seeking women, however, while adult teasing, self-esteem, and internalization of sociocultural appearance standards may be associated with BID, childhood teasing was not (Matz, Foster, Faith, & Wadden, 2002). Among a sample of obese treatment-seeking women, early-onset obesity was associated with increased BID and lower self-esteem in adulthood, independent of adult BMI, but the relation between early-onset obesity and adult BID was not mediated by childhood teasing or low self-esteem as hypothesized (Wardle, Waller, & Fox, 2002). There is also evidence that BID is heterogeneous among obese women (Hill, 1998). This suggests that BID, independent of childhood teasing, may be an important

mediator of depression in obese individuals, especially, but not exclusively, women.

Repeated Dieting. The evidence about the relation between weight and dieting history and depression in obese individuals is equivocal, and methodological differences in mood measurement may account for differential findings (Smoller, Wadden, & Stunkard, 1987). While some studies have suggested that no relation exists between repeated dieting or weight cycling and depression (Foster, Sarwer, & Wadden, 1997), recent evidence suggests that repeated dieting is associated with depression, and that repeated diet failure may have a particularly deleterious effect on mood (Demenkow, 1999).

This mechanism may act in two ways. Repeated efforts to lose weight may mean that the individual was unsuccessful in these attempts, leaving the person feeling demoralized and like a failure. The experience of taking off weight and then regaining it may also be experienced as failure, in that the individual fails to maintain his or her weight loss. Beck's well-researched cognitive theory of depression (1967, 1983) posits that individuals employ schemata, that is, mental representations of themselves and the world, which may be depressogenic. Dieting failure may activate a negative self-schema, such as "I am a failure," thereby contributing to depression. The dietary-restraint model posits that the reliance on cognitive mechanisms, instead of physical cues, to control eating behavior leaves dieters vulnerable to episodes of binge eating when these cognitive strategies are compromised (Polivy & Herman, 1985). If such a binge occurs in violation of strict dietary rules, the dieter may temporarily suspend all attempts to control food intake because of the abstinence violation effect (Marlatt & Gordon, 1985). Dieting failure, therefore, may put individuals at particular risk for both depression and obesity, as it can contribute to episodes of binge eating and activation of negative self-schemata. In addition to experiencing weight cycling as failure, there is also evidence that the experience of being on a diet may in and of itself worsen mood.

Obesity (determined by self-reported height and weight) was associated with increased dieting, and dieting was associated with depressive symptoms reported on the CES-D among a large nationwide sample of men and women (Ross, 1994). Among a sample of 149 obese

women, those with higher BMIs had made more attempts to diet in the past than those with lower BMIs, and memories of dieting attempts were usually negative (Ikeda, Lyons, Schwartzman, & Mitchell, 2004). Furthermore, reducing caloric intake may cause a worsening of mood. There is evidence that a very low-calorie diet is associated with irritability (Laederach-Hoffman, Kupferschmid, & Mussgay, 2002). Caloric restriction may have particularly negative effects on individuals with a history of depression. One study found that obese women with a history of depression showed impaired regulation of brain serotonin function in response to dieting (Smith, Williams, & Cowen, 2000).

Dieting may also be especially distressing for patients who use food as an emotional regulator, particularly binge eating disorder (BED) patients. Studies have suggested that weight cycling is associated with binge eating, particularly among the obese (Foster et al., 1997). In a sample of obese treatment-seeking individuals, those with BED were found to have higher BDI scores (15.5 versus 8.1) and eating in response to negative mood than non-BED patients (Kuehnel & Wadden, 1994). If a patient uses food to alleviate depressed mood, the deprivation of food in weight loss treatment might then contribute to dysphoria.

One study found that weight cycling was significantly related to binge eating, and binge eating was significantly related to psychological distress among a sample of obese women seeking weight loss treatment. When binge eating was controlled for, however, there was no relation between weight cycling and psychological distress (Venditti, Wing, Jakicic, Butler, & Marcus, 1996). History of weight cycling and perceived experience of weight cycling were related to decreased self-esteem and life satisfaction, and increased body dissatisfaction among a large sample of people who had tried to lose weight in the past three years, but further analysis indicated that the perception of weight cycling was a much stronger predictor of all three psychological measures than actual history (Friedman, Schwartz, & Brownell, 1998). Another study of men and women found that perceived weight cycling was significantly related to negative affect in men and to binge eating in both genders (Womble et al., 2001). Weight cycling itself might be an important causal mechanism insofar as it relates to binge eating. The perception of oneself as a weight cyler (independent

of actual weight cycling) may, however, be an important mechanism causing depression in the obese, due to the sense of failure it might engender. Similarly, dieting itself may not cause depression per se, but repeated dieting failure may be a common and depressogenic experience in obese individuals (Ikeda et al., 2004).

DEPRESSION AS A CAUSE OF OBESITY

Although less work has been done examining risk factors and causal mechanisms that may cause depressed individuals to become obese, there are some data indicating that depression does confer risk for later obesity. More work determining the causal mechanisms of this association is warranted. As there are no intervention studies examining whether improved mood results in weight loss among the obese, we review longitudinal, but not intervention, studies here.

Longitudinal Studies

Data from samples of children and adolescents indicate that psychopathology in childhood may be related to increased risk for obesity in adulthood, although the data are mixed. Goodman and Whitaker (2002) assessed a large sample of adolescents with a one-year follow-up, and found that baseline depressive symptoms on the CES-D independently predicted obesity at follow-up among participants who were not obese at baseline, even when controlling for smoking, self-esteem, parental obesity, SES, conduct disorder, and physical activity. Although the large sample size, adequate control measures, and prospective nature of the study are strengths, it is limited by the use of self-reported height and weight. Another large study of adolescent girls found that negative affect assessed by the Expanded Form of the Positive and Negative Affect Schedule (PANAS-X), dietary restraint, radical weight control, and perceived parental obesity all independently predicted the onset of obesity at four-year follow-up (Stice, Presnell, Shaw, & Rohde, 2005). This study is limited by the use of a measure of negative affect instead of MDD or depressive symptoms, so it is not possible to draw conclusions regarding the impact of adolescent depression on obesity onset, but this limitation is counterbalanced by the strength of measuring actual obesity onset, rather than merely increase in BMI. One study compared a large sample of children being treated for MDD with controls of similar ages with no psychiatric

disorder, assessed with the schedule for affective disorders and schizophrenia for school-age children (K-SADS), and found that childhood MDD was associated with increased BMI (26.1 versus 24.2) in young adulthood (assessed 10–15 years later), which could not be explained by potential confounds (Pine, Goldstein, Wolk, & Weissman, 2001). Whereas an interesting result, it does not necessarily indicate that childhood depression predicts the onset of obesity in adulthood, as BMI of 26.1 is in the overweight range. In another study of children aged 6–12 at risk for obesity, either because of current overweight or having an obese parent, depressive symptoms on the Children's Depression Inventory did not predict the onset of obesity four years later (Tanofsky-Kraff et al., 2006). Scores on this measure were, on average, well below the cutoff for clinical depression, so this evidence does not necessarily indicate that childhood depression is not predictive of the onset of adult obesity, because these children were not depressed. It also appears that the relation between childhood MDD and later obesity may be partially moderated by gender. Among a cohort of children born in New Zealand, late adolescent girls with depression, assessed by the Diagnostic Interview Schedule for Children (DISC), had greater than a twofold increased risk for obesity in adulthood compared with their nondepressed female peers (Richardson et al., 2003). Furthermore, a dose–response relation between the number of episodes of depression during adolescence and risk for adult obesity was observed in females, but not males (Richardson et al., 2003). On the other hand, there does not appear to be evidence that depression in middle-aged and older adults increases the risk for later obesity. Among the Alameda County study sample, depression at baseline did not predict obesity five years later when controlling for obesity at baseline and demographic and psychosocial factors (OR = 1.17; Roberts et al., 2003). Although not entirely consistent, these data taken together seem to suggest that adolescent depressive symptoms, especially among girls, put individuals at risk for the onset of obesity later in life, although depression in middle and later adulthood may not confer similar risk.

Proposed Mechanisms

Overall, we suggest that there may be both a direct physiological and an indirect psychosocial pathway that link depression to obesity. Specifically, there may be

a direct pathway through the biological effect of increased stress reactivity with hormonal change, and an indirect pathway through which mechanisms, such as poor adherence, binge eating, negative thoughts, and reduced social support, make it more difficult for the depressed person to care for themselves effectively, leading to weight gain.

Direct Physiological Pathway

Hypothalamic–Pituitary–Adrenal Axis and Immunological Dysregulation. Previous research has demonstrated a significant relationship between depressive symptomology and hypothalamic–pituitary–adrenal (HPA) dysregulation (De Bellis, Gold, Geraciotti, Listwak, & Kling, 1993), as well as chronic elevations in cortisol in depressed individuals (Lupien et al., 1998). This situation is often manifested as an elevated reactivity to stress and challenges to the stress response in those with clinical depression, even after administration of dexamethasone (Modell, Yassouridis, Huber, & Holsboer, 1997). It has been suggested that antidepressants may exert their primary influence through regulation and normalization of the HPA response (Holsboer & Barden, 1996) and that failure to regulate the HPA response during treatment elevates the risk for depressive relapse (Holsboer, 2000). This may also suggest an important neuroendocrine link between depression and the development of obesity. Heightened levels of cortisol have been found to promote weight gain, with a particular impact on increased visceral adiposity (Ottoson, Lonroth, Bjorntorp, & Eden, 2000; Pasquali, Vicennati, & Gambineri, 2002). Furthermore, stress has been found to influence eating behaviors, body weight, and body composition (Greeno & Wing, 1994). The depressed individual may be more susceptible to these effects due to an already heightened activity of the stress system.

Recently, the contribution of inflammatory markers, such as the proinflammatory cytokines tumor necrosis factor- α , interleukin-1, interleukin-6, and interferon- γ , to the development and pathogenesis of depression has been examined (see Kenis & Maes, 2002). Increased inflammatory responses have been found in depressed individuals (Carney, Freedland, Miller, & Jaffe, 2002), and this may be particularly pronounced if obesity is also present (Miller, Stetler, Carney, Freedland, & Banks, 2002). It has been found that exogenous cytokine

administration, either centrally or peripherally, influences both mood states and eating behaviors (Konsman, Parnet, & Dantzer, 2002). Considering the link between elevated levels of these immunological mediators and activation of the HPA axis, it is not surprising that there has been some initial support for reductions in certain proinflammatory cytokines as a result of antidepressant administration (e.g., Basterzi et al., 2005). Due to different methodologies used to examine the cytokine/depression/antidepressant link (Kenis & Maes, 2002), considerable work remains to be done in order to determine the mechanism(s) underlying this relationship. Nonetheless, this appears to be a promising area of future inquiry, particularly if considered in conjunction with the responses of the HPA axis.

Indirect Psychosocial Pathway

Adherence. Poor adherence is one behavioral factor that may mediate the relation between depression and health outcome. Furthermore, depression has been shown to predict attrition from weight loss programs (Clark, Niaura, King, & Pera, 1996). It may be more difficult for a person experiencing clinically significant depressive symptomatology to engage in the meal planning and exercise recommendations necessary to lose weight.

Adherence may also be affected by the tendency of depressed patients to amplify physical symptoms of chronic medical conditions (Katon & Ciechanowski, 2002). If depressed patients experience symptoms related to their obesity, such as joint pain or breathing difficulty, this may mediate their adherence to weight-management programs, particularly exercise regimens. There is also evidence that depression is linked to increased functional disability and additively increases functional disability in those with chronic disease (Wells et al., 1989). This may further impair patients' ability to adhere to diet and exercise regimens.

Dysregulatory and Binge Eating. Binge eating may be an important mechanism by which depression confers risk for obesity. There is evidence that negative mood precipitates episodes of binge eating among obese women with BED (Arnou, Kenardy, & Agras, 1995), and that emotional overeating is associated with binge frequency and depression among overweight treatment-seeking BED patients (Masheb & Grilo, 2006). Binge eaters have been shown experimentally to eat in response

to negative mood (Chua, Touyz, & Hill, 2004), and therefore may be likely to gain weight when depressed. The use of food as a primary coping strategy for emotion regulation, particularly binge-size quantities of food, will almost certainly cause overweight or obesity for those who experience chronic depression, as they will frequently experience poor mood. Additionally, there is also prospective evidence that binge eating confers risk for later depression. It seems that depression may confer risk for obesity through the mediator of binge eating, but this link may function bidirectionally.

Dysregulatory eating, binge eating, and BED have been suggested as potential risk factors for depression and obesity by several researchers (Fabricatore & Wadden, 2004; Faith, Calamaro, Dolan, & Pietrobelli, 2004; Faith et al., 2002; Friedman & Brownell, 1995; Stunkard et al., 2003). Among obese females with BED, severity of binge eating was positively associated with BDI score (Telch & Agras, 1994). Furthermore, BED participants scored significantly higher on the BDI and measures of cognitive dysfunction, as well as preoccupation with thinness and eating in response to negative mood, than non-BED participants in a study of treatment-seeking obese women (Kuehnel & Wadden, 1994). There is evidence that the association between binge eating and negative affect begins as early as childhood. Binge eating has been shown to predict high levels of depressive and anxiety symptoms and low levels of self-esteem and body esteem on self-report measures among treatment-seeking obese adolescents (Glasofer et al., 2007; Isnard et al., 2003). Stice, Burton, and Shaw (2004) followed a large sample of adolescent girls for several years, and found that MDD assessed with the K-SADS predicted future bulimic symptoms (including binge eating) and bulimic symptoms (including binge eating) predicted future depression (Stice et al., 2004). Among samples of both treatment-seeking and non-treatment-seeking overweight and obese children, those with episodes of binge eating had lower self-esteem than those without (Decaluwe, Braet, & Fairburn, 2003; Tanofsky-Kraff, Faden, Yanovski, Wilfley, & Yanovski, 2005).

Additionally, there is evidence that obese BED patients use food to regulate mood. Kuehnel and Wadden (1994) found that those with BED were more likely to experience depression and eating in response to negative mood. Chua et al. (2004) demonstrated that, among a sample of

female binge eaters seeking treatment, sad mood induced increased food intake. The negative mood experienced in depression would then contribute to increased caloric intake in an effort to improve mood (Chua et al., 2004). There is also evidence that improvements in binge eating predict greater weight loss in treatment, mediated by reduction in dysphoria (Sherwood, Jeffrey, & Wing, 1999). Furthermore, interpersonal psychotherapy, a treatment that addresses social and interpersonal deficits, is effective in the treatment of BED, and results in small weight loss and improvement on depression scores (Wilfley et al., 2002). Accordingly, interpersonal factors likely play a role in the maintenance of both BED and depression. Binge eating appears to be an important link between depression and obesity. The evidence cited here suggests that binge eating occurs in response to negative mood, which sets the stage for a cycle of weight gain and further negative mood, such that this mediator functions bidirectionally.

Negative Thoughts. The negative thoughts and attitudes characteristic of depression may contribute to problems with weight management among obese individuals, particularly among women. Depressed individuals are likely to have lower self-efficacy, including about their ability to lose weight. Self-efficacy theory posits that self-efficacy, which is an individual's judgments about his or her abilities related to outcome attainment, directly affects outcome expectancy, which directly influences behavior (Bandura, 1997). Depressed individuals with characteristically low self-efficacy may therefore believe that they will be unable to lose weight, which may influence their weight loss attempting behavior. Because weight loss self-efficacy has been shown to predict weight loss, reduced self-efficacy may interfere with weight loss in individuals experiencing depression and obesity. Individuals experiencing depression likely lack the considerable resources and motivation necessary for planning and eating a healthy diet and engaging in regular exercise necessary for weight loss or maintenance.

There is some evidence that, among women, depression may be inversely related to self-efficacy about weight loss, which has been found to predict weight loss (Dennis & Goldberg, 1996). In a large sample of overweight and obese men and women, MDD operationalized by having been diagnosed by a medical doctor or having a

prescription for antidepressant medication was associated with lower weight self-efficacy and less weight loss at 6 and 12 months in women but not in men (Linde et al., 2004). Bryan and Tiggemann (2001) found that among a sample of obese women in a weight loss study, lower scores on scales of impression management and vulnerability predicted greater weight loss. The lack of optimism in depression may also contribute to obesity. There is evidence that subjective predicted weight is a good predictor of actual weight loss in women (Karlsson et al., 1994). Because depressed individuals are likely to assume the worst, they may believe that they will not lose weight, which may adversely affect any weight loss attempts.

Reduced Support. Depressed individuals likely have reduced social support from family and friends, which can make it harder to adhere to a weight loss program. There is evidence that weight loss programs employing help from important others in a patient's life are more successful than individual programs (Wing & Jeffrey, 1999; Wing, Marcus, Epstein, & Jawad, 1991). People with depression may not have these resources available.

Depressed individuals may also cause strain and erosion of family functioning and social support (Coyne, 1976; Keitner & Miller, 1990). Considering that social support has been shown to be related to weight loss (Black, Gleser, & Kooyers, 1990; McLean, Griffin, Toney, & Hardeman, 2003), the lack of social support of the depressed individual may make it more difficult to lose weight. Additionally, social support is related to maintenance of weight loss (Bishop, 2002). Given the chronic nature of depression, even if an individual managed to lose weight when not depressed, we would expect this patient might have more difficulty maintaining this weight loss over time given the likelihood of reduced social support.

At this point, it is uncertain whether depression causes later obesity, but there is some evidence suggesting that there may be a link. We have suggested several mechanisms by which this pathway may occur, in addition to the mechanisms by which obesity may cause depression. In summary, there appears to be a bidirectional causal pathway between obesity and depression. Moderators, such as high SES, severe obesity, BID, and gender, make certain obese individuals more at risk for depression than others. Several proposed mechanisms, including behavioral factors such as binge eating and BED,

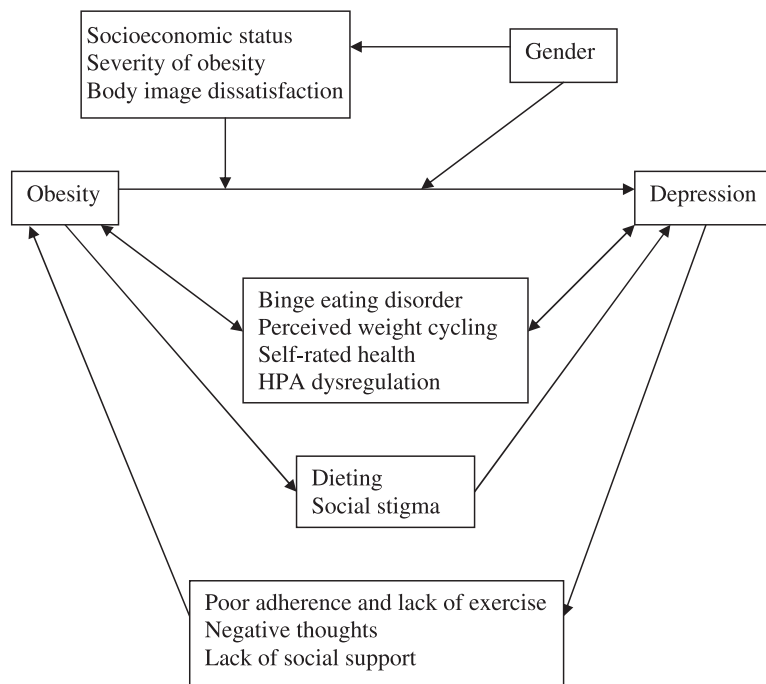


Figure 1. Model for bidirectional pathway of casual links between obesity and depression.

cognitive factors such as perceived weight cycling and self-rated health, and physiological factors such as HPA axis and immunological dysregulation, appear to function bidirectionally, such that they may have a role in how obesity causes depression, and vice versa. The proposed mechanisms by which obesity confers risk for depression unidirectionally include the behavioral factor of dieting and the social factor of stigma faced by the obese. Finally, there may be a causal pathway from depression to obesity that operates through behavioral mechanisms of poor adherence and lack of exercise, cognitive mechanisms such as negative thoughts, and social mechanisms such as reduced support (see Figure 1).

MANAGING OBESITY AND DEPRESSION

The treatment of depression and the treatment of obesity are similar in some important ways, suggesting that there is potential for the two treatments to interact synergistically, each reinforcing the other. There are, however, also many important differences in the treatment of each, which means that there is also the potential for the treatment of one condition to undermine treatment of the other. It is important for practitioners to be aware of these areas of

overlap and challenges, so that care may be integrated as effectively as possible. It is also imperative that researchers examine how to effectively integrate care to improve treatment outcome.

Overlap

Treatments for obesity and depression overlap in two important ways and can be informed by our identified causal pathways. Specifically, treatment of both conditions focuses on improved life functioning. For example, behavioral activation, which has demonstrated efficacy in the treatment for depression (Jacobson et al., 1996), can also be helpful in managing obesity, as it encourages individuals to engage in activities that give them a sense of mastery, which may include exercise and healthy eating strategies. Similarly, both treatments involve stress management; depression treatment such as cognitive-behavioral therapy addresses coping strategies for dealing with potential setbacks, such as avoiding all-or-none thinking and planning for difficult situations, and obesity treatments typically involve skills that help individuals choose alternatives to unhealthy eating behaviors in order to cope with stress.

Exercise and Lifestyle. Considerable evidence exists touting the benefits of exercise for the improvement of physical health and weight management (Avenell et al., 2004; Keim, Blanton, & Kretsch, 2004; Melzer, Kayser, & Pichard, 2004). However, there is also an expanding body of literature supporting the contention that exercise has a beneficial effect on both mood and stress reactivity (see Landers & Arent, 2001, for a review). The effect of exercise on depression has generated particular interest, with at least six meta-analytic reviews conducted on the topic since the 1990s (Calfas & Taylor, 1994; Craft & Landers, 1998; Kugler, Seelback, & Kruskemper, 1994; Lawlor & Hopker, 2001; McDonald & Hodgdon, 1991; North, McCullagh, & Tran, 1990). Across the meta-analytic reviews, moderate to large effects have consistently been seen for the reduction of depressive symptoms with both aerobic and anaerobic exercise, particularly for clinical depression (Craft & Landers, 1998), with the effects typically equivalent to those seen for more traditional psychotherapeutic interventions (Craft & Landers, 1998; Lawlor & Hopker, 2001). There is also evidence that inactivity predicts depression both cross-sectionally and longitudinally (Camacho et al., 1991; Farmer et al., 1988; Lobstein, Mosbacher, & Ismail, 1983). Experimental evidence also exists suggesting that exercise may produce effects equivalent to those of pharmacological treatment over 16 weeks of treatment (Blumenthal et al., 1999). This may be partly due to the common neuroendocrine effects shared by exercise and drugs commonly used in the treatment of depression (Dishman & Buckworth, 1997; Jacobs, 1994; Landers & Arent, 2001). Furthermore, consistent with the corticosteroid receptor hypothesis of depression (Holsboer, 2000), recent research has demonstrated that HPA and autonomic responses may be important mechanisms underlying the affective responses to acute bouts of resistance exercise (Arent, Landers, Matt, & Etnier, 2005). Given the available evidence, exercise may be a mutually beneficial adjunctive treatment modality for the management of both obesity and depression. It is not suggested that exercise alone replace other treatments for depression or obesity, but rather that exercise be included as a supplemental treatment. Chronic exercise and leisure-time physical activity have been found to reduce the inflammatory response associated with C-reactive protein (Ford, 2002), which may further impact the regulation of the HPA axis,

considering the link between inflammation and the stress response.

The implementation of an actual exercise regimen in this population is not without its problems. While exercise is important for management of both weight and mood, it may be challenging for patients to adhere to this recommendation. Considering that the affective response to an initial exercise experience is one of the best predictors of future adherence (Rejeski, 1994), recent findings on the impact of intensity and exercise mode on affect (Arent et al., 2005; Ekkekakis, 2003) can be used to guide prescription that may maximize both psychological benefit and likelihood of adherence. Practitioners should give patients practical suggestions for adding small amounts of exercise into their daily routine and stress the importance of lifestyle modification, as there is some evidence that it is as effective as prescribed exercise. Anderson et al. (1999) found that among a sample of 40 obese women, those randomized to a treatment encouraging lifestyle change to add activity lost the same amount of weight at one-year follow-up as those assigned to aerobics classes.

Stress Management. An important skill that may work to manage both depression and weight is stress management. Not only has stress been implicated as a potential cause of visceral obesity (Drapeau, Therrien, Richard, & Tremblay, 2003; Ottoson et al., 2000), but also patients who are unable to maintain weight loss over time often cite stress as a reason for their regain (Munnelly & Feehan, 2002). There is also evidence that stress reduction may be a helpful element in the treatment of depression and the prevention of relapse (Manber, Allen, & Morris, 2002; Teasdale, Segal, & Williams, 1995). This makes sense given the proposed role of the stress response in the etiology of depressed or anxious mood (Holsboer, 2000). Furthermore, stress has been suggested as a potential mediator between obesity and depression (Stunkard et al., 2003). Teaching patients to effectively manage stress should have a positive impact on their ability to control both mood and weight. Additionally, pharmacological agents targeting central stress hormone receptors may prove useful in the treatment of both conditions and warrant further examination (Holsboer, 2000). Another possibility is to incorporate nonpharmacological treatments that have been

shown to reduce stress reactivity, depression, and obesity. One such intervention is exercise.

Challenges

One major limitation in the traditional approach of managing *either* weight *or* depression without considering them as comorbid conditions is that there are some situations in which the treatment for one may actually exacerbate the other. For example, dieting may worsen mood, particularly if it is part of a pattern of repeated dieting failure. Conversely, the most common pharmacological treatments for depression have the side effect of weight gain.

Dieting. The most common intervention for weight loss typically involves some form of caloric restriction. Depending on the individual's experience, however, dieting may either improve or worsen mood. The actual experience of weight loss may increase self-esteem and self-efficacy, thereby having a positive effect on mood and ameliorating depressive symptoms. Actually being on a diet, however, can worsen mood, as caloric restriction is associated with irritability (Laederach-Hoffman et al., 2002). Because it is rare for individuals to maintain weight loss after dieting (Jeffrey et al., 2000), if they are able to achieve weight loss at all, it may be beneficial for practitioners to encourage lifestyle changes in lieu of dieting (Ikeda et al., 2004). There is some preliminary evidence that lifestyle change interventions, which emphasize improved nutrition and increased walking for long-term health rather than immediate weight loss, may be as effective as dieting for long-term weight loss (Rapoport, Clark, & Wardle, 2000), but the psychological effects of these types of treatment are less well explored. One study examined the effect of a lifestyle intervention compared with traditional behavioral weight loss treatment and a wait-list control condition among 62 obese women and found that both lifestyle intervention and behavioral weight loss produced modest but significant weight loss, whereas only the lifestyle intervention improved depression and anxiety symptoms on the BDI and State-Trait Anxiety Inventory (Tanco, Linden, & Earle, 1998).

Antidepressant Medication. Just as dieting may negatively impact mood, the pharmacological treatment of depression may negatively impact weight loss. There is a large

body of literature suggesting that antidepressants, particularly tri-cyclic antidepressants (TCA) and monoamine-oxidase inhibitors (MAOI), but also selective serotonin reuptake inhibitors (SSRI), cause weight gain (Fava, 2000; Golden, 2004; Gupta, Tiller, & Burrows, 2003; Sussman, Ginsberg, & Bikoff, 2001). This is a major reason for nonadherence to treatment in the population of depressed individuals not selected by weight (Deshmukh & Franco, 2003; Fava, 2000; Golden, 2004). While inconvenient and unpleasant in normal weight individuals, the side effect of weight gain is medically unsafe for obese individuals (Fontaine et al., 2003).

There is evidence that SSRIs have preferable side effect profiles with respect to weight gain in comparison to TCAs and MAOIs, and that some SSRIs are preferable to others (Fava, 2000; Fava, Judge, Hoog, Nilsson, & Koke, 2000; Sussman et al., 2001). In fact, although they are associated with weight gain in maintenance treatment, some SSRIs, such as fluoxetine, may induce weight loss in the acute phase of treatment (Fava et al., 2000; Sussman et al., 2001). There is also evidence that the SSRI sertraline may be useful as an adjunct to cognitive-behavioral weight loss treatment in obese individuals with and without mood problems (Ricca et al., 1996).

As of yet, no studies have examined the simultaneous treatment of both depression and obesity. So far, only changes in mood in the context of obesity treatment have been studied (e.g., Clark et al., 1996; Gladis et al., 1998; Karlsson et al., 1994; Linde et al., 2004; Nauta et al., 2001; Sherwood et al., 1999). Practitioners should encourage the patient to engage in behaviors that will improve both conditions, such as stress management, exercise, and potentially lifestyle modification in lieu of simply dieting or just taking medication.

SUMMARY AND FUTURE DIRECTIONS

Research has suggested important risk factors and causal mechanisms in the association between obesity and depression. We have suggested a bidirectional causal model by which each of these conditions may contribute to the other. Studies testing the differential effects of specific elements of this model will help clarify these associations. Longitudinal studies should be a high research priority in order to prospectively predict which obese individuals are likely to become depressed, and vice versa, and intervene prophylactically. In addition to this type of research,

there is a need for studies examining treatments intended for comorbid depression and obesity, rather than each condition individually. We have suggested reasons for which integrated treatment might be particularly useful in this population, but so far there is no research of which we are aware to directly support this hypothesis.

The field has advanced in the identification of important risk factors and causal mechanisms linking obesity and depression, but still has not reached the stage where optimal treatment resulting in long-term psychological and physical benefits is adequately employed. The development and testing of integrated treatment to simultaneously manage both depression and obesity should be a priority for further investigation.

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Received October 28, 2006; revised October 28, 2006;
accepted October 30, 2006.