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Does Depression Cause Obesity?

A Meta-analysis of Longitudinal Studies of Depression and Weight Control

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Abstract

To evaluate the causal effects of depression on obesity, longitudinal tests of the effect of depression on follow-up obesity status were meta-analyzed. Combining data from 16 studies the results confirmed that, after controlling for potential confounding variables, depressed compared to nondepressed people were at significantly higher risk for developing obesity. The risk among depressed people for later obesity was particularly high for adolescent females (odds ratio: 2.57, 95% CI: 2.27, 2.91). These findings highlight the importance of depression screening and treatment programs, especially among adolescents, to assist in the prevention of adult obesity.

Keywords

- *depression*
- *meta-analysis*
- *obesity*
- *weight control*

MANY hundreds of studies have been conducted to examine the relationship between depression and obesity. The size of this research literature is noteworthy given that reviews find very small bivariate correlations between measures of depression and obesity (Faith & Allison, 1996; Friedman & Brownell, 1995). Recent national surveys confirm these conclusions. Data from the 2005 National Health Interview Survey, a study of over 30,000 American adults, found that depression and body mass index (BMI) correlated at .08 (National Center for Health Statistics, 2006). The 1995 National Longitudinal Study of Adolescent Health, a survey of over 20,000 youth, found similarly small correlations (.08 and –.01 for girls and boys, respectively) between depression and BMI (Needham & Crosnoe, 2005).

Despite the weak cross-sectional evidence linking depression and obesity, causal models linking the two constructs are theoretically appealing. One prominent causal model argues that obesity is a deeply stigmatizing attribute that prompts negative stereotyping and discrimination in others, which, in turn, causes depression and other negative psychological and social outcomes (Brownell, Puhl, Schwartz, & Rudd, 2005; Puhl & Brownell, 2006). Given the ethical and methodological issues associated with experimentally manipulating obesity, the best evidence for this model must come from controlled longitudinal studies. Several studies have found that obese compared with nonobese adults at baseline measurement are about two times more likely to be depressed at follow-up measurement (Roberts, Deleger, Strawbridge, & Kaplan, 2003; Roberts, Kaplan, Shema, & Strawbridge, 2000; Roberts, Strawbridge, Deleger, & Kaplan, 2002). Other research, however, found no longitudinal relationship between obesity and later depression (Hasler et al., 2005a).

A second, alternative causal model recognizes that depression can exert causal effects on obesity. This way of thinking about the relationship between depression and obesity is less intuitive than the previous model, in part because depression is traditionally considered an outcome rather than a causal variable. However, a large research literature attests to the causal effects of depression on an array of chronic illnesses, including hypertension and coronary heart disease (see Shulz et al., 2000). Theoretically, how does depression cause obesity? Eating and energy expenditure are often proposed as intervening constructs in the depression–obesity relationship. In this model depression is hypothesized to cause obesity indirectly through such behaviors as emotional eating, eating calorie-dense food, and decreased physical activity (see Dallman, Pecoraro, & la Fleur, 2005; Oliver & Wardle, 1999; Wise, Adams-Campbell, Palmer, & Rosenberg, 2006). Our concern in the present article is with evaluating the empirical evidence for the direct hypothesis in this model: that increases in depression explain later weight gain or obesity status.

Experimentation provides the best evidence of a causal hypothesis and, although depressive disorder cannot be ethically manipulated, studies that manipulate short-term depressive mood find that it does affect eating behavior. For example, depressed and nondepressed mood was experimentally induced in obese and nonobese dieters or nondieters (Baucom & Aiken, 1981). After the mood induction eating behavior was covertly measured. The effect of negative mood on eating was moderated by dieting status: dieters ate more when depressed than when nondepressed and the reverse was true for nondieters. In a more recent experiment, induced negative compared with neutral mood caused more self-reported emotional eating in college students (Bekker, van de Meerendonk, & Mollerus, 2004). Despite their ability to randomize potential confounding variables, these studies lack construct and ecological validity. Depressed mood following bogus failure feedback is a poor proxy for a depressive episode or disorder; likewise, a laboratory eating (e.g. taste rating) task does not generalize well to real-life eating behavior. We therefore turn to the next-best type of evidence—the controlled longitudinal study—to evaluate the hypothesis that depression causes weight gain. Longitudinal studies establish temporal priority, thereby ruling out the alternative causation that threatens cross-sectional studies. With regard to making causal inferences from longitudinal data, longitudinal research is strengthened to the extent that it measures and (statistically) controls for plausible confounding variables.

The value of longitudinal research for extending knowledge about the relationship between depression and weight control has been acknowledged in a recent review (Faith, Matz, & Jorge, 2002). Faith and his colleagues' review suggests that two variables might moderate the effect of depression on later obesity: whether the study controlled for background variables that could cause both baseline depression and later obesity, and the age of subjects. Sufficient numbers of these studies have been

published to merit a quantitative review. The present study reports a meta-analytic review of longitudinal studies of the relationship between depression and later obesity. The review estimates the overall population effect size for depression on later weight gain or obesity status, determines if that effect remains when controlling for potential confounding variables, and examines whether the effect is moderated by subject age.

Method

Articles were identified from searches of the MEDLINE and PsycINFO databases using the terms *obesity*, *depression*, and *longitudinal*. Articles were also traced from references in a review article (Faith et al., 2002) and from the literature reviews of identified articles. Articles were included if they employed a longitudinal design with a baseline measure of depression and a follow-up measure of weight change or obesity status. All identified articles were coded for the following variables: sample size, sample age (coded adolescent or adult), sample sex, mean age of subjects at baseline measure, duration of study (time in years between baseline and follow-up measures), analysis type (coded uncontrolled or controlled), and measure of depression used in the study. Effect size statistics were recorded from each study in the form of an odds ratio (the probability of depressed compared to nondepressed subjects at baseline having obesity status at follow-up) or converted to odds ratios from the particular inferential statistics or p levels reported in the article. The data conversions and meta-analysis were conducted with Comprehensive Meta Analysis Version 2 (2005).

Results

Sixteen (16) studies met the inclusion criteria and yielded effect size statistics from 23 samples for meta-analysis; the studies' descriptive statistics are presented in Table 1. The sample effect sizes are displayed in Fig. 1 for all studies. Effect size Z scores ranged from –3.35 to 20.22, with most (18 out the 23) samples providing data that depression leads to weight gain. Of the five samples in which depression led to weight loss (a negative Z score) rather than gain, the effect was significant ($p < .05$) in only one of those samples. In the analyzed studies, follow-up obesity measurement occurred an average of 7.6 years after baseline depression measurement (see Table 1).

A random effects model was used to assess the overall relationship between depression and weight control. This yielded a significant population effect size estimate of 1.47 (95% CI: 1.16, 1.85), indicating that depressed people at baseline measurement are about 1.8 times more likely than nondepressed people to have obese status or weight gain at follow-up measurement. There was considerable nonrandom variability across sample estimates, $Q(22) = 474.6$, $p < .001$, indicating that the relationship between depression and weight change was likely moderated by other variables.

To determine if the overall estimate was moderated by whether a study reported adjusted or unadjusted effect size statistics, a fixed effects analysis was conducted that generated population effect size estimates separately from those two types of studies. Table 1 lists the controlled variables for each study. The studies ($n = 17$) that estimated the effect of depression on weight change while controlling for baseline BMI and other control variables yielded an population effect size estimate of 1.18 (95% CI: 1.13, 1.23) whereas studies ($n = 6$) reporting unadjusted effect size statistics yielded an estimate of 1.25 (95% CI: 1.14, 1.38). In sum, controlling important confounding variables such as baseline BMI strengthened the causal analysis but slightly reduced the size of the effect of depression on weight change, although this meta-effect was still significant.

Subject sex and age were also analyzed as moderating variables. Using a fixed effects analysis, male ($n = 4$, odds ratio: 1.34, 95% CI: 1.14, 1.58) and female ($n = 11$, odds ratio: 1.26, 95% CI: 1.20, 1.32) did not generate significantly different estimates. Further, these results did not change when analyzing only studies with adjusted effect size statistics. Thus, depressed compared with nondepressed men and women do not differ in their greater likelihood of being obese or having gained weight at follow-up, controlling for baseline BMI and background variables.

A similar analysis investigated the moderating influence of sample age and found that adolescent ($n = 12$, odds ratio: 2.31, 95% CI: 2.06, 2.58) and adult ($n = 11$, odds ratio: 1.08, 95% CI: 1.03, 1.13) samples differed in the effect of depression on obesity status. This difference, as well as the odds ratios for each group, was essentially unchanged when only samples reporting adjusted effect size statistics were analyzed.

Table 1 Descriptive statistics of reviewed studies

Study	*Sample size (n)*	*Sample % white*	*Sample age*	*Sample sex*	*Mean baseline age (yrs)*	*Study dur (yrs)*	*Analysis type*	*Control variables*	*Depression measure*
Pine et al. (1997)A	334	90.0	Adol	Male	14	8	Uncontr		DSM-III criteria
Pine et al. (1997)B	310	90.0	Adol	Female	14	8	Uncontr		DSM-III criteria
Pine et al. (2001)	90		Adol	Both	11.7	12.6	Uncontr		Schedule for Affective Disorders
Stice et al. (2005)	486	68.0	Adol	Female	13.5	4	Contr	Baseline obesity status, dietary restraint, perceived parental obesity, compensatory behaviors	Schedule for Affective Disorders
Richardson et al. (2003)A	490		Adol	Male	19.5	6.5	Contr	Baseline obesity status, SES, maternal obesity, maternal depression	DSM-III criteria
Richardson et al. (2003)B	439		Adol	Female	19.5	6.5	Contr		DSM-III criteria
Richardson et al. (2003)C	903		Adol	Both	13	13	Contr		DSM-III criteria
Goodman & Whitaker (2002)	9374	74.7	Adol	Both	15	1	Contr	Baseline BMI, childhood physical activity, delinquency, race, number of parents in home, parental education, baseline depression	CES-D
Tanofsky-Kraff et al. (2006)	146	67.1	Adol	Both	9	4.2	Contr	Baseline BMI, race, SES, dieting history, eating attitudes and behavior	Children's Depression Inventory
Bardone et al. (1998)	470		Adol	Female	15	6	Contr	SES, menarche age, presence/absence of father figure, parental smoking, childhood health, maternal health, maternal BMI	DSM-III criteria
Franko et al. (2005)	1554	50.1	Adol	Female	16.5	5	Contr	Baseline BMI, parental education	CES-D
Barefoot et al. (1998)	4726		Adult	Both	19	21	Contr	Baseline BMI, smoking, exercise	MMPI-D
Roberts et al. (2003)	1886		Adult	Both	45	5	Uncontr		DSM-IV criteria
Noppa & Hallstrom (1981)	712		Adult	Female	na	6	Contr	Baseline BMI, age, class	Hamilton Rating Scale

(Continued)

Table 1 (Continued)

Study	*Sample size (n)*	*Sample % white*	*Sample age*	*Sample sex*	*Mean baseline age (yrs)*	*Study dur (yrs)*	*Analysis type*	*Control variables*	*Depression measure*
Haukkala et al. (2001)A	41		Adult	Female	54	4	Contr	Baseline BMI, age, education, health behaviors	BDI
Haukkala et al. (2001)B	27		Adult	Female	54	4	Contr		BDI
DiPietro et al. (1992)A	594		Adult	Male	<55	4.3	Contr	Baseline BMI, age, education, health behaviors	CES-D
DiPietro et al. (1992)B	752		Adult	Female	<55	4.3	Contr		CES-D
DiPietro et al. (1992)C	448		Adult	Both	≥55	4.3	Contr		CES-D
Hasler, et al. (2005b)	299		Adol	Female	17	20	Contr	Baseline BMI, physical activity, SES, family weight history	DSM-III, -IV criteria
Forman-Hoffman et al. (2007)A	4253	72.5	Adult	Male	57	8	Uncontr		CES-D
Forman-Hoffman et al. (2007)B	4877	69.0	Adult	Female	57	8	Uncontr		CES-D
Hasler et al. (2005a)	479		Adult	Both	19	21	Contr	Demographics, health behaviors, physical activity	DSM-III criteria

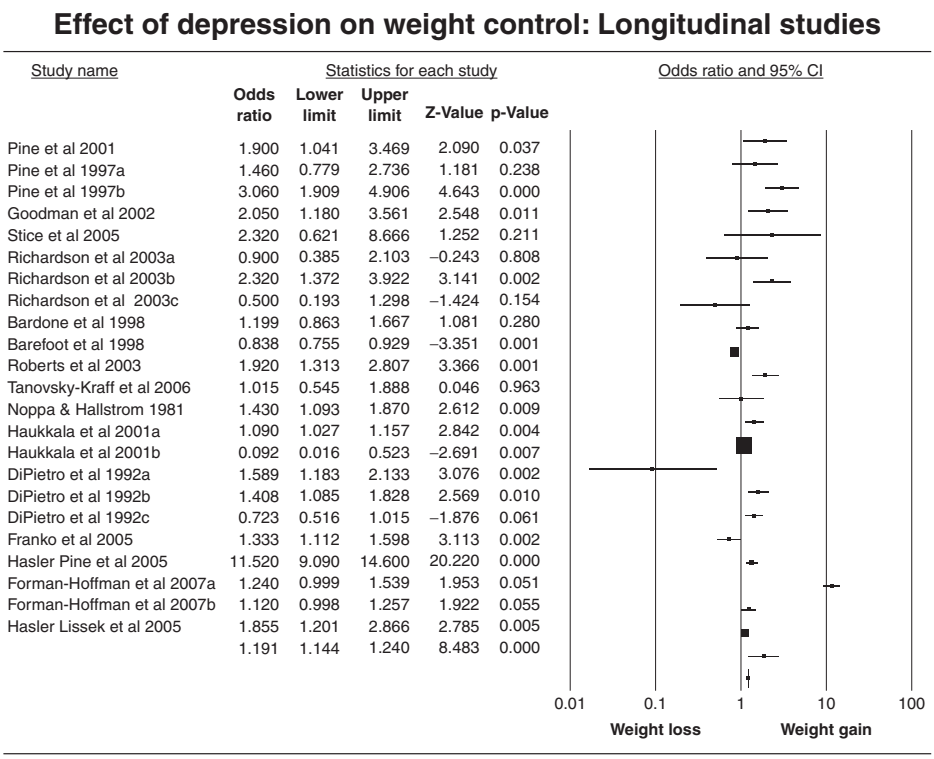

Figure 1. Effect size estimates in the form of odds ratios (with 95% confidence interval) and forest plot for all samples in the meta-analysis.
Note: Forest plot line thickness indicates the weighting of each sample in the analysis

Does the risk posed by depression for developing later obesity among adolescents differ for girls and boys? Analyzing only adolescent samples found evidence of significant risk among girls (n = 6, odds ratio: 2.57, 95% CI: 2.27, 2.91) but none among boys (n = 2, odds ratio: 1.23, 95% CI:.74, 2.04). These results indicate that, overall, adolescents with depression are about 2.5 times more likely than nondepressed adolescents to have obesity status or weight gain at follow-up measurement. However, the risk was further qualified by sample sex: after controlling for baseline BMI and other variables, depression predicted later obesity only in girls. The apparent lack of risk in boys is based on just two samples and should be interpreted with caution.

The small risk posed by depression on obesity in adult samples is misleading. Of the eleven (11) samples of adults in the analysis, three provided estimates that depression lowers the risk of obesity status, whereas five samples provided estimates that depression increases the risk of obesity status. It appears that the age of the adult subjects may help account for these different estimates. The samples in which depression was found to reduce the risk of obesity, or produce weight loss, have markedly older subjects than those reporting the opposite effects. These results show that the implications of depression for later weight control are more dependent on other factors (i.e. chronic illness) in adults, especially older adults, than in adolescents.

Discussion

In this report, longitudinal research was meta-analyzed to see if depression accounts for later obesity after plausible confounding variables are controlled. The results, combining data from over 33,000 subjects, showed that depressed compared with nondepressed people are significantly more likely to be obese at follow-up measurement. This general vulnerability is heightened in female adolescents such that depressed female teens are about two-and-a-half

times more likely to be obese at follow-up measurement than are nondepressed female teens. To put this effect in context, the effect of adolescent depression on obesity in the young adult years is larger in size than the effect of passive smoking (inhaling second-hand smoke) on the development of cancers (Vineis et al., 2005). Whereas bans on smoking in public places have been enacted across the USA in response to the health dangers of second-hand smoke, little attention has been given to the long-term health consequences of adolescent depression.

These conclusions mesh with other research that is similar to the reviewed studies but did not meet the inclusion criteria for the review. For example, in a large community sample of young adults depression predicted later weight cycling after controlling for possible confounding variables (Hasler et al., 2005a). Other research has found that, after controlling for potential confounds, depression in adolescent girls accounted for binge eating 20 months later (Stice, Presnell, & Spangler, 2002). In that study, depression was the sole risk factor for later binge eating among girls who did not overemphasize the importance of appearance for relationships and achievement. Finally, in a study of 2400 weight losers from the National Weight Control Registry, those subjects who regained weight from baseline to one-year follow-up measurement also had significant increases in depression during that time while the weight maintainers' depression levels did not change (Phelan, Hill, Lang, Dibello, & Wing, 2003).

Research identifies trycyclic antidepressant medications (e.g. amitriptyline) as particularly likely to cause weight gain (Brady, 1989). However, a recent review finds that for most antidepressants the effect on subsequent weight change is unclear or unknown (Vanina et al., 2002). Nevertheless, it is possible that some of the relationship observed in this review may be an artifact of antidepressant use. Because the articles in this review, with one exception (Hasler et al., 2005a), do not report subjects' antidepressant use or control for it in analyses, we cannot rule out the confounding influence of antidepressant use.

In sum, there is mounting evidence that adolescent depression is very likely a cause of later weight gain and/or obesity status. The main clinical implication of this meta-analysis is the observation that adolescent depression screening and treatment programs, alongside nutritional and exercise-based interventions, are an important tool in the prevention of adult obesity. The need to treat root causes of adult obesity is emphasized by the fact that, even in the context of supervised treatment programs, large-scale weight loss among adults is unusual and difficult to sustain (Blaine, Rodman, & Newman, 2007). This review provides the empirical support for the direct effect of adolescent depression on later weight gain. What is still not clear is how adolescent depression promotes weight gain; this study did not test a mediating mechanism. Mediating explanations that surface in the literature include the impact of depression on emotional eating and exercise. Future research must sort out the possible mediators and indirect effects.

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