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PAPER

Prospective association between obesity and depression: evidence from the Alameda County Study

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OBJECTIVE: To examine the temporal relation between obesity and depression to determine if each constitutes a risk factor for the other.
DESIGN: A two-wave, 5-y-observational study with all measures at both times.
SUBJECTS: A total of 2123 subjects, 50 y of age and older, who participated in the 1994 and 1999 waves of the Alameda County Study.
MEASUREMENTS: Obesity defined as body mass index (BMI)$\geqslant$30. Depression assessed using DSM-IV symptom criteria for major depressive episodes. Covariates include indicators of age, gender, education, marital status, social support, life events, physical health problems, and functional limitations.
RESULTS: Obesity at baseline was associated with increased risk of depression 5 y later, even after controlling for depression at baseline and an array of covariates. The reverse was not true; depression did not increase the risk of future obesity.
CONCLUSION: These results, the first ever on reciprocal effects between obesity and depression, add to a growing body of evidence concerning the adverse effects of obesity on mental health. More studies are needed on the relation between obesity and mental health and implications for prevention and treatment.
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Keywords: obesity; depression; epidemiology; prospective study; older subjects

Introduction

Is there an association between obesity and depression? There are four possible hypotheses. First, obesity increases the risk of depression. Second, depression increases the risk of obesity. Third, there is a reciprocal relation, such that the obese are at increased risk of depression and the depressed are at increased risk of obesity. Fourth, there is no association between obesity and depression. Which of these hypotheses does the empirical evidence suggest as more plausible?

In all, 11 studies have looked at this association using cross-sectional or prevalence study designs, with seven of these finding some evidence of greater risk of depression among the obese. But while seven have found support for the proposition that the obese are at a greater risk for depression, the evidence has not been uniformly robust.

Reed,[1] using data from the first National Health and Nutrition Examination Survey (NHANES I), found that obesity was related to worse mental health in both White and Black women. Regression analyses identified young, more educated, obese females as a subgroup with worse mental health (mostly symptoms of anxiety and depression). Istvan *et al*[2] also used baseline data from the NHANES I study and found that relative body weight was weakly related to elevated depression scores in women but not men.

Ross,[3] using data from a representative sample of 2020 adults 18 y and older, found no direct effect of being overweight on depression in most groups. Overweight persons were more likely to diet and to experience worse physical health, both of which were associated with depression. However, being overweight did increase depression among the more educated (but not among the less educated).

Han *et al*[4] found no overall association between obesity and mental health functioning using the mental health measure from the SF-36 in a sample aged 20–59 y in the Netherlands. However, men in the highest tertile for obesity were more likely to report not being happy and women in the highest tertile for obesity were more likely to report depressed mood.

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Carpenter *et al*[5] using data from a large national sample from the US, found that obesity was associated with an increased risk of depression and suicidal ideation among women but not men.

Roberts *et al*,[6] using data from the 1994 wave of the Alameda County Study, found an odds ratio (OR) of 2.29 for depression among the obese compared to those of normal weight. There was no association between underweight and depression. In a second paper, Roberts *et al*[7] found an association between obesity and depression (OR = 2.13), and also found that obesity was associated with worse perceived mental health (OR = 1.53), more pessimism (OR = 1.60), and lower life satisfaction (OR = 1.43).

What about prevalence studies which have not found any evidence for obesity as a risk factor for depression? In one of the early studies, Crisp and McGuiness[8] found that obesity was related to lower levels of anxiety in both middle-aged women and men and to lower levels of depression in men. This 'jolly fat' hypothesis was subsequently re-examined in a more rural sample and the same result was found for middle-aged men. However, the association was much weaker for women overall, obesity being related to lower anxiety in older, working-class women and to lower depression in younger, middle-class women. There was no association between obesity and depression in younger men.[9] Hallstrom and Noppa [10] studied women 38–54 y of age and found no association between obesity and present or past mental illness (including anxiety, phobias, depression, contact with psychiatrists, or use of psychotropic drugs). Palinkas *et al*[11] found that obesity was not related to risk for depression in women aged 50–89 y, but among men depression was inversely related to obesity, thus partially confirming the 'jolly fat' hypothesis.

There is also an additional line of evidence bearing on the association between obesity and depression. A small number of studies have examined differences between obese individuals presenting to clinics for weight loss and general population controls on measures of depression. Based on a meta-analysis of such studies, Friedman and Brownell[12] found a moderate effect and this result was consistent across studies; the obese presenting for weight loss were more depressed. All of these case–control studies, while consistent in their findings, are based on cross-sectional data and do not clarify the direction of the obesity–depression relation.

We are aware of only two studies which have examined obesity as a risk factor for depression with a prospective design. Roberts *et al*[6] used data from the 1994–1995 cohort from the Alameda County Study and found a two-fold risk for depression in 1995 among those classified as obese in 1994. A second study by Roberts *et al*[7] then examined obesity as a risk factor for depression and seven other indicators of mental health functioning using the 1994–1999 Alameda County Study cohort. Again, those obese at baseline were at elevated risk for subsequent depression. The samples were 50 y of age and older at baseline.

Furthermore, we are aware of only one study which tested the effects of depression on subsequent weight, and this assessment focused on weight gain rather than obesity *per se*. Noppa and Hallstrom[13] followed up a sample of women for 6 y and found a positive association between depression at baseline and subsequent weight gain. Weight gain was not predicted by anxiety or phobias.

Based on the evidence thus far, it is unclear what the temporal relation is between obesity and depression. Only two papers have examined whether obesity predicts future risk of depression, and both find evidence that the obese have increased risk of future depression. However, both were based on the same study. We found only one paper which examined the reverse. In that paper, depression predicted future weight gain.

Given the paucity of data on the temporal association between obesity and depression, our purpose was to re-examine this question using two waves of data, collected 5 y apart, in which data on both obesity and depression were collected in both waves. The objective was to determine which of the four hypothesized relations between obesity and depression were supported by the data, for example, obesity is a risk factor for depression, depression is a risk factor for obesity, there is a reciprocal relation between obesity and depression, or there is no association between obesity and depression.

Method

The mental and physical health of a community sample in Alameda County, California, has been studied for over 30 y. In 1994, a fourth wave of data was collected. As part of this follow-up study, data on a diverse set of mental health indicators were collected, including symptoms of DSM IV major depressive disorders. In addition, extensive data on putative risk factors were collected, including data on social and physical functioning. In 1999, a follow-up survey was conducted, permitting examination of the reciprocal effects between obesity and depression with prospective data.

Using data from the 1994 and 1999 surveys, we examined the reciprocal relation between obesity and depression, and the contribution of other putative risk factors for mental health: gender, marital status, socioeconomic status, physical health and disability, life stress, and social support. In a previous paper, we used the 1994–1995 cohort data to examine the prospective association between obesity in 1994 and eight mental health outcomes in 1999, including depression. However, that paper did not examine reciprocal effects between obesity and mental health outcomes. To our knowledge, the current paper is the first to address this question empirically.

Sample

The sample was drawn from the Alameda County Study, a longitudinal study of physical and mental health and mortality that has followed a cohort of 6928 persons selected

in 1965 to represent the adult noninstitutionalized population of Alameda County, California. Subjects are followed regardless of subsequent location or disability status. Survivors have been assessed with the full questionnaire in 1974, 1983 (50% sample), 1994, and 1999 with response rates of 85, 87, 93, and 96% respectively. Detailed design and sampling procedures for this study have been reported elsewhere.[14,15]

The 1994 follow-up included 2730 subjects, of whom 2123 also responded in 1999. The analyses reported here are based on 1886 subjects who were 50 y or older in 1994 and who had complete data on the measures of depression, the BMI and on all the other risk factor measures. In a number of previous analyses based on the 1994 cohort, we focused on age effects and grouped the subjects into 10-y cohorts. For purposes of comparability, that strategy is employed here as well. There were only 75 subjects under 50 y of age in 1994.

In terms of age, 42.3% of the subjects were 50–59, 30.9% were 60–69 y, 21.3% were 70–79 y, and 5.6% were 80 y or older. The age ranged from 50 y to 94 y with a mean age of 63. Females comprised 55.7% of the respondents. Most of the subjects were married, with 26.1% reporting that they were divorced, separated, widowed, or never married. Only 12.3% of them had less than a high school education.

Measures

Every wave of the Alameda County Study has included items inquiring about height (without shoes) in feet and inches, and weight (without heavy clothes) in pounds. These are converted to kilograms in weight and meters in height to calculate the BMI (kg/m^2). We classify obesity using the 1998 guidelines from the National Heart, Lung, and Blood Institute.[16] Based on the NHLBI criteria, obesity is defined as a BMI$\geq$30 kg/m^2. The obese represented 17.3% of the subjects in 1994 using the criterion of BMI$\geq$30 kg/m^2. Scoring of obesity as a categorical measure also facilitates analyses of incidence.

The measure of depression was a set of 12 items which operationalized the diagnostic criteria for a major depressive episode (MDE) outlined in DSM-IV.[17] Designated the DSM-12D, the items or symptom queries were adapted from the PRIME-MD mood disorders section.[18] 'Cases' of MDE were subjects who experienced five or more symptoms of depression 'almost every day for the past two weeks', including disturbed mood or anhedonia. Use of this measure in the Alameda County Study cohort has been reported previously.[19,20] In 1994, 7.3% of the subjects met DSM-IV symptom criteria for depression. The items are presented in the Appendix.

Covariates (correlates) examined were age, gender, education, marital status, social isolation, social support, life events, financial strain, problems with normal daily activities, and chronic medical conditions. These factors can be categorized as status attributes, personal and social resources, and stressors, and are widely considered to be important determinants of risk for depression and other mental health outcomes.

Age was categorized as 50–59, 60–69, 70–79, and 80 y or older at baseline. Educational attainment was dichotomized: 12 y or less and more than 12 y. Marital status also was dichotomized: married versus other (divorced, separated, widowed, never married). Our measure of isolation consisted of six items: '(1) How many friends can you confide in; (2) How many relatives do you feel close to; (3) How many friends and relatives do you see at least once a month; (4) How many friends and relatives can you turn to for help; (5) How many friends and relatives can you talk to about personal measures; (6) How many friends and relatives do you have you can ask for advice or information?' A score of fewer than three on each question was considered an isolated response. The numbers of isolated responses were summed and coded into low (0), medium (1–2), and high social isolation (3+).

Our measure of social support asked 'How often is the following available: (1) someone to take you to the doctor; (2) someone to prepare meals for you; (3) someone to help you with your daily chores if you are sick; and (4) someone to loan you money if you need it'. Each question was scored from 0 (none of the time) to 4 (all of the time) and then summed into a total scale ($\alpha = 0.90$). The scale was divided into low (0–9), medium (10–15), and high (16) support.

We asked subjects about whether 17 life events had occurred in the current or previous year. The total number of recent events were summed. Financial strain consisted of five items which inquired 'How many times there was not enough money: (1) to buy clothes; (2) to fill a prescription; (3) to see a doctor; (4) to pay rent or mortgage; and (5) to buy food'. Not having enough money on any one item was coded as financial strain.

We asked about the occurrence of 12 chronic medical conditions in the last 12 months and whether a physician had been consulted. These conditions were heart trouble, high blood pressure, asthma, chronic bronchitis, arthritis, emphysema, diabetes, stroke, cancer, cataracts, osteoporosis, and circulatory problems. The numbers of conditions were summed and then categorized as none, 1, or 2 or more. We also asked respondents if they had difficulty with usual daily activities (ADL): (1) walking across a small room; (2) bathing; (3) brushing hair or washing face; (4) eating; (5) dressing; (6) moving from bed to a chair; and (7) using the toilet. Any difficulty on any item was classified as having a problem with ADLs.

Table 1 presents the distribution of the 1994 cohort on the measures included in the analyses.

Statistical analyses

Differences in the percentage of obese subjects with depression were tested with simple χ^2 statistics. Logistic regression models were used to assess the association between 1994 obesity and 1999 depression, then between 1994 depression and 1999 obesity.

Table 1 1994 Alameda County Study cohort characteristics (N=1886)

1994 Variables	*Value*	*No.*	*%*
Demographics			
Age	50–59	797	42.3
	60–69	582	30.9
	70–79	401	21.3
	80 or older	106	5.6
Gender	Female	1051	55.7
	Male	835	44.3
Years of education	⩾12	1654	87.7
	⩽12	232	12.3
Marital status	Married	1394	73.9
	Div/Sep/Wid/Never	492	26.1
Social factors			
Financial strain	Yes	303	16.1
	No	1583	83.9
Recent life events	None	703	37.3
	1	509	27.0
	2	367	19.5
	3 or more	307	16.3
Social isolation	Low	810	42.9
	Medium	588	31.2
	High	488	25.9
Social support			
	Low	398	21.1
	Medium	666	35.3
	High	822	43.6
Mental and physical health			
Depression	Yes	138	7.3
	No	1748	92.7
Obesity	Yes	325	17.2
	No	1561	82.8
Chronic conditions	None	864	45.8
	1	570	30.2
	2 or more	452	24.0
ADL	No problem	1745	92.5
	Problem	141	7.5

To examine the effect of obesity on subsequent depression, two sets of models were fitted. In the first, we computed ORs between 1994 obesity and 1999 depression, adjusting for depression status in 1994. In the second, we estimated the same OR, after eliminating all subjects with depression in 1994. To examine the effect of depression on subsequent obesity, two sets of models also were fitted. In the first, we computed ORs between 1994 depression and 1999 obesity, adjusting for obesity status in 1994. In the second, we estimated the same OR, after eliminating all obese subjects in 1994. Sequential logistic regression models were run. The first models were adjusted for age, gender, education, and marital status. Groups of variables were then added to the models to examine the influence of the different factors on the obesity/depression association.

All statistical analyses were carried out using SAS software (version 6.12).

We excluded 176 subjects from the analyses because of missing data on the variable of interest or covariates. Among them, 27 had missing data on depression and/or obesity status in 1994, 36 had missing data on depression and/or obesity status in 1999, six had missing data both in 1994 and 1999 for obesity and/or depression status, and 107 had at least one missing data on covariates used in the different sequential models.

Results

First, we examined the cross-sectional association between obesity and depression in 1994 (Table 2). The Pearson χ^2 test showed a significant association between obesity and depression in 1994 ($P<0.001$). The probability of depression among the obese was 0.126 and the probability of depression among the nonobese was 0.062. Thus the prevalence ratio (PR) for depression in 1994 among the obese in 1994 was 2.03 with a 95% confidence interval (CI) [1.44–2.87]. The probability of obesity among the depressed was 0.297 and among the nondepressed was 0.162. Thus the PR for obesity in 1994 among the depressed in 1994 was 1.83 with a 95% CI [1.39–2.41]. Obese people were then twice as likely to be depressed, while depressed people were 1.8 times as likely to be obese.

We repeated these analyses examining the association between obesity in 1999 and depression in 1999 (data not shown). The results were very similar to those for 1994, that is, the prevalence ratio for depression among the obese was 1.83 with a 95% CI [1.33–2.53] and the prevalence ratio for obesity among depressed was 1.65 with a 95% CI [1.28–2.13]. In both 1994 and 1999, we observed that the prevalence ratio for depression among the obese was somewhat larger than the prevalence ratio for obesity among the depressed. However, there still was an association between obesity and depression in both directions.

We then examined the prospective association between obesity and depression (Table 3). The OR of the association between obesity in 1994 and prevalence of depression in 1999 was OR = 2.09 with a 95% CI [1.44–3.03]. Eliminating those depressed in 1994, the OR between obesity in 1994 and incident depression in 1999 was 2.01 with a 95% CI (1.25–3.25) (data not shown). Thus, subjects who were obese in 1994 had twice the risk of becoming depressed in 1999 than subjects who were not obese in 1994.

We then turned the question around, that is, we examined whether depression in 1994 predicted obesity in 1999 (Table 3). The OR of the association between depression in 1994 and obesity in 1999 was 1.92 with a 95% CI [1.31–2.80]. Eliminating the obese in 1994, the OR between depression in 1994 and incident obesity in 1999 was 1.32 with a 95% CI

Table 2 Percentage of obese subjects in 1994 reporting depression in 1994

			Depressed in 1994			
	Value	*No.*	N	*%*	*PR*[a]	*95% CI*
Obesity in 1994	Obese	325	41	12.6	2.03	1.44–2.87
	Nonobese	1561	97	6.2		

[a]PR, prevalence ratio.

Table 3 Cohort analysis of obesity in 1994 and subsequent depression in 1999, and depression in 1994 and subsequent obesity in 1999

	Value	*No.*	N	*%*	*OR*[a]	*95% CI*
			Depressed in 1999			
Obesity in 1994	Obese	325	44	13.5	2.09	1.44–3.03
	Nonobese	1561	109	7.0		
			Obese in 1999			
Depression in 1994	Depressed	138	43	31.2	1.92	1.31–2.80
	Nondepressed	1748	334	19.1		

[a]OR, odds ratio.

(0.65–2.70) (data not shown). Thus, subjects who were depressed in 1994 had less than twice the risk of being obese in 1999 than subjects who were not depressed in 1994, and eliminating the obese at baseline, the depressed at baseline were not at increased risk of becoming obese in 1999.

Next, we examined the prospective association between obesity and depression, first with 1994 obesity predicting 1999 depression adjusting for depression status in 1994 (Table 4) and then 1994 depression predicting 1999 obesity adjusting for obesity status in 1994 (Table 5). In both tables, we present results using sequential logistic regression models, first the model (Model 1) containing obesity and depression, adjusted on age, gender, education, and martial status, then controlling for financial strain, life events, social isolation, and social support (Model 2), and then controlling for chronic medical conditions and ADL (Model 3). In both tables, we present two sets of results, one set based on all subjects ($N=1886$) to study the prevalence of obesity and depression and the other set excluding subjects who were depressed in 1994 (Table 4, $N=1748$) or who were obese in 1994 (Table 5, $N=1561$) to study the incidence of both conditions.

As can be seen in Table 4, obesity in 1994 was a significant predictor of depression in 1999, using both prevalence and incidence data. There was a two-fold increased risk for prevalent depression in 1999 for those obese in 1994, in Models 1 and 2, and the OR with all covariates in the model (Model 3) was still 1.8 using incidence data. Thus, based on the incidence data from Table 4, obesity at baseline increased the risk of depression 5 y later, even when controlling for social and health covariates. Using prevalence data, obesity predicted depression in 1999 in Models 1 and 2, but not in Model 3 ($P=0.058$).

Table 5 presents a different picture. None of the ORs using either prevalence or incidence data are statistically significant. In fact, Model 3 based on incidence data yielded an OR of 1.01 and prevalence data yielded an OR of 1.17. In sum, the depressed at baseline were not at increased risk for subsequent obesity.

Several papers[3,5,11] have reported gender differences in the relation between obesity and depression. A gender–obesity interaction term was added to all models but failed to be significant, thus rejecting the hypothesis that the relationship between obesity and depression might be different for men and women. The *P*-value for the estimated interaction parameter was 0.73 in the prevalent depression model and 0.85 in the incident depression model.

Discussion

We began by asking what might be the association between obesity and depression. The question was whether obesity increased risk for depression, depression increased risk for obesity, there was reciprocal risk, or there was no associated risk for either obesity or depression. Using prospective data from the Alameda County Study, we found evidence only for the first hypothesis. Obesity at baseline was associated with being depressed at follow-up 5 y later. This was true in both bivariate analyses and in a series of multivariate analyses controlling for a number of covariates that affect risk for depression. We demonstrated here that obesity was able to predict subsequent depression. Our data did not demonstrate support for depression predicting subsequent obesity. Even if our data are not conclusive on this latter issue, our findings suggest a probable causal relationship between obesity and subsequent depression, and support the idea that depression does not predict subsequent obesity.

How do our results compare with those from other studies? This comparison is easy: This is the first study published to date that has used prospective data to examine reciprocal effects between obesity and depression.

The results are consistent with seven papers reporting cross-sectional data[1–7] and two papers reporting prospective data[6,7] that find evidence for an association between obesity and depression, such that obesity is associated with increased risk for depression. As noted in the Introduction, there have been negative results as well.[8–11]

Our results are not consistent with those reported by Noppa and Halstrom.[13] They found that depression at baseline predicted weight gain 6 y later. We also found that depression at baseline predicted obesity at follow-up 5 y later when no other adjustments were made. However, after adjusting on obesity status at baseline, or limiting the analyses to the nonobese at baseline, this association was nonsignificant, suggesting that the apparent association was attributable, in part, to greater obesity among those depressed at baseline as shown in Table 2.

Another possibility is that the association between depression at baseline and subsequent obesity is attenuated because of a subset of the depressed actually losing weight. Several

Table 4 Sequential logistic regression models showing relation between obesity in 1994 (BMI⩾30) and depression in 1999, with adjustment for other risk factors

	Adjusted[a] *(N=1886)*		*Incidence*[b] *(N=1748)*	
Depression, 1999	*OR*	*95% CI*	*OR*	*95% CI*
Model 1=Controlling for age, gender, education and marital status	1.69	1.11–2.59	2.06	1.26–3.37
Model 2=Model 1+financial strain, recent life events, social isolation, and social support	1.65	1.07–2.55	1.92	1.16–3.18
Model 3=Model 2+chronic conditions and ADL	1.53	0.99–2.38	1.79	1.06–3.02

[a]Adjusting on depression status in 1994.
[b]Excluding those depressed in 1994.

Table 5 Sequential logistic regression models showing relation between depression in 1994 and obesity in 1999 (BMI⩾30), with adjustment for other risk factors

	Adjusted[a] *(N=1886)*		*Incidence*[b] *(N=1561)*	
Obesity, 1999	*OR*	*95% CI*	*OR*	*95% CI*
Model 1=Controlling for age, gender, education and marital status	1.20	0.68–2.13	1.27	0.62–2.62
Model 2=Model 1+financial strain, recent life events, social isolation, and social support	1.43	0.79–2.61	1.36	0.64–2.90
Model 3=Model 2+chronic conditions and ADL	1.17	0.64–2.15	1.01	0.46–2.22

[a]Adjusting on obesity status in 1994.
[b]Excluding those who were obese in 1994.

epidemiological studies support the notion that both men and women who are depressed lose more weight than those who are not.[20,21] Depressed patients tend to weigh less than matched healthy controls, and unexplained weight loss is included in the DSM-IV criteria for major depression.

In the only other study which focused on subjects 50 and older, Palinkas *et al*[11] found no association between obesity and depression among women. But both overweight and obese men were at much lower risk of depression than men of normal weight. They argue that these results for men are consistent with the 'jolly fat' hypothesis. Thus, at this point, the only two studies of older samples have found very different results. The most likely explanation for these disparate results relates to differences in populations studied and procedures used.

Based on our results presented here, and results from the other studies we cite, we conclude that the obese appear to be at increased risk for depression. However, there has been sufficient diversity in results from epidemiologic studies to justify further examination of this issue. In particular, we need more data from long-term, prospective studies using contemporary diagnostic criteria for depression. To date, ours is the only study to employ DSM-IV diagnostic criteria to assess depression and the only prospective study looking at future risk of depression among the obese.

We should also note that other significant predictors of incident depression, other than obesity, were: older age (OR=1.95; 1.07–3.53); female (OR=1.99; 1.22–3.24); less than high school education (OR=2.15; 1.23–3.77); financial strain (OR=2.30, 1.37–3.83); recent life events (OR=2.08, 1.10–3.92); social isolation (OR=2.01, 1.12–3.61); social support (OR=0.53, 0.31–0.92); chronic conditions (OR=1.92, 1.14–3.23); and ADL (OR=2.07, 1.06–4.07). These findings are consistent with earlier studies based on the Alameda County Study,[22,23] as well as other studies of older samples.[24–27]

One limitation of our results was that, although we had data from a prospective study, we had only two waves of data on obesity and depression. Both obesity and depression are, in many respects, health conditions that are the product of the life-long interaction of risk and protective factors, and two observation points over 5 y capture only a small interval in the lives of people 50–94 y of age.

Another limitation is that BMI was calculated using self-reports of height and weight rather than measured values. The literature on this point is consistent. Self-reports of height and weight are not as accurate, with studies showing that subjects tend to overestimate height and underestimate weight thereby underestimating obesity.[28–31] The correlation between measured and reported obesity (BMI) is high in general, but prevalence of obesity using self-reports is lower than using measured values.[31] Sensitivity of reported obesity declines with age, but specificity is little affected by age.[31] Our measure of BMI almost surely underestimates obesity, perhaps as much as one unit (see Kuczmarski *et al*[31]). At the same time, our self-report measure of depression is almost certainly an overestimate, since we did not have clinical diagnosis. Given this, the net effect is probably small, although of course, the true effect is unknown since we do not have independent measures of either the risk factor or the outcome. It is unlikely that the magnitude of the observed association between obesity and depression would

be substantially reduced in our study and extremely unlikely that the direction of the association would be reversed.

The data from the Alameda County Study do not include data on the use of psychotrophic medication. The relation between weight and depression may be confounded by the greater tendency for the depressed to be taking antidepressants. However, it is unclear from the available literature whether and in what ways medication use might affect the observed association between depression and obesity. It has not been unequivocably demonstrated whether taking antidepressants results in weight gain, weight loss, or has no effect on weight.[32–36] Measures of obesity, depression and use of psychotrophic drugs collected periodically over the lifespan ideally are needed to address the question of reciprocal effects. Major depression can be a chronic, intermittent disorder, and to understand more fully its association with another condition, such as obesity, ideally requires data on lifetime episodes. We did not have data on lifetime prevalence of MDE.

Several studies also have examined obesity and anxiety.[8–10,13] These results have been mixed as well, with some supporting the jolly fat hypothesis and others providing no support. The Alameda County Study did not include a measure of anxiety *per se*, so we could not examine the effect of obesity on that outcome. Given the inconclusive results thus far, more research certainly is warranted on the association between obesity and anxiety.

A number of explanations for a relation between obesity and mental health, particularly depression, have been offered, including the possible role of psychological, sociological, and biological factors.[3,11,12] Ross,[3] for example, has outlined two possible explanations for an association between obesity and depression. One, the reflected self-appraisal perspective, argues that the stigma toward and devaluation of the obese may cause overweight individuals to suffer from lower self-esteem, have more negative self-images, think others dislike them, and have higher levels of depression. The less common, normal, and acceptable it is to be overweight in a group, the greater would be the psychological impact. The second, the fitting norms of appearance perspective, argues that for those who are obese, fitting the norm for weight is stressful because dieting is stressful rather than obesity *per se*. This may be particularly true when weight control is not successful, which is commonly the case.[37] Ross[3] presents data supporting the fitting-appearance-norms hypothesis, but found little support for the reflected appraisal hypothesis. These competing perspectives offer plausible explanations for sociocultural processes linking obesity with psychological dysfunction. However, to date there have been no attempts to replicate or extend the research by Ross.

Palinkas *et al*[11] note that obesity also might be associated with depression through differential consumption of nutrients affecting depression, in particular, carbohydrates. Consumption of carbohydrates appears to affect the vegetative symptoms of depression via central serontogic activity while also affecting weight *per se*.[17–19,38] We did not have data on carbohydrate consumption and so were not able to assess this association. Obese people also are less likely to exercise, and physical activity reduces the risk of depression by increasing the levels of endorphins, improved regulation of norepinephrine, improved fitness, and enhanced self-esteem.[20,39]

There is also evidence, albeit limited, that first-degree relatives of probands with morbid obesity are more likely to have mental disorders than relatives of controls, particularly depression, bipolar disorder, and antisocial personality disorders.[40] From these data, however, it is not possible to partition variance attributable to genetics *vs* environment. But the results provide additional evidence for a link between obesity and psychopathology.

Obesity is a complex phenomenon. There is now good evidence for important genetic and physiological components in the etiology of obesity, and further that obesity is quite heterogeneous with regard to etiology, effects of obesity on health, and response to treatment.[8,38,41]

Friedman and Brownell[12] argue that research should focus more on which subgroups of the obese have more psychological dysfunction, the nature of the dysfunction, and associated risk and protective factors. To this, we might add that studies are also needed that focus not just on whether there are mental health effects of obesity, but also whether these effects are specific to particular mental health outcomes or are more generic in nature. More data are also needed on the natural history of obesity and mental health to ascertain the nature and magnitude of reciprocal effects and the implications of such effects for the prevention and treatment of obesity.

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Appendix

Items contained in DSD-12 (response: yes/no, for nearly every day for past 2 weeks)

1. Feeling sad, blue, or depressed.
2. Loss of interest or pleasure in most things.
3. Feeling tired out or low on energy most of the time.
4. Loss of appetite or weight loss.
5. Overeating or weight gain.
6. Trouble falling asleep or staying asleep.
7. Sleeping too much.
8. More trouble than usual concentrating on things.
9. Feeling down on yourself, no good, or worthless.
10. Being so fidgety or restless that you moved around a lot more than usual.
11. Moved or spoke so slowly that other people could have noticed.
12. Thought about death more than usual, either your own, someone else's, or death in general.