doi: 10.1111/j.1467-789X.2010.00843.x

Comorbidities

Evidence for prospective associations among depression and obesity in population-based studies

M. S. Faith[1], M. Butryn[2], T. A. Wadden[1], A. Fabricatore[1], A. M. Nguyen[3] and S. B. Heymsfield[4]

[1]Center for Weight and Eating Disorders, Department of Psychiatry, University of Pennsylvania School of Medicine, Philadelphia, PA, USA; [2]Department of Psychology, Drexel University, Philadelphia, PA; [3]Merck & Co, Inc., North Whales, PA; [4]Pennington Biomedical Research Center, Baton Rouge, LA

Received 29 June 2010; revised 14 October 2010; accepted 11 November 2005

Address for correspondence: MS Faith, Center for Weight and Eating Disorders, University of Pennsylvania School of Medicine. 3535 Market Street – 3rd Floor, Philadelphia, PA 19104, USA.
E-mail: mfaith@mail.med.upenn.edu

Summary

Obesity may lead to depression or be one of its consequences. We reviewed population-based studies in order to, first, identify the most commonly used research methods, and, second, to evaluate the strength of evidence for prospective associations among obesity and depression. We examined 25 studies, of which 10 tested 'obesity-to-depression' pathways, and 15 tested 'depression-to-obesity' pathways. Descriptive statistics summarized the frequency with which various measurements, designs and data analytic strategies were used. We tallied the number of studies that reported any vs. no statistically significant associations, and report on effect sizes, identified moderating variables within reports, and sought common findings across studies. Results indicated considerable methodological heterogeneity in the literature. Depression was assessed by clinical interview in 44% of studies, weight and height were directly measured in 32%, and only 12% used both. In total, 80% of the studies reported significant obesity-to-depression associations, with odds ratios generally in the range of 1.0 to 2.0, while only 53% of the studies reported significant depression-to-obesity associations. Sex was a common moderating variable. Thus, there was good evidence that obesity is prospectively associated with increased depression, with less consistent evidence that depression leads to obesity. Recommendations for future research regarding study samples, measurement and data analysis are provided.

Keywords: Depression, obesity, risk factors.

obesity reviews (2011) **12**, e438–e453

Introduction

Obesity and depression are two prevalent disorders that are medically (1,2) and economically (3,4) costly to individuals and society. The prevalence of obesity among adults in the USA is 32% (5), while that of major depressive disorder is 6.7% (6). Paediatric obesity and depression also represent significant public health concerns, as their rates have increased in recent decades (7–9). Given these statistics, investigators need to better understand the causes of obesity and depression, in order to enhance strategies for their clinical care and prevention.

Prior reviews have cited evidence for the co-occurrence of obesity and depression (10), suggesting that these disorders may be causally linked. However, this literature consists primarily of cross-sectional investigations that could not test the direction of causal pathways. These studies also yielded inconsistent findings, including evidence for no associations (11), positive associations (12–14), negative associations (15), and sex- (16–18) or ethnicity-moderated (19) associations. Clear conclusions cannot be established from these findings.

Supported by a grant from Merck & Co., Inc.

Prospective studies have the advantage of being able to evaluate the temporal pathways from 'obesity-to-depression', and 'depression-to-obesity'. Longitudinal studies have increased over the years, including investigations (from diverse countries) that have included both children and adults. The aim of the present paper was twofold. Our first objective was to summarize the most commonly used research methods in prospective studies that tested obesity–depression associations, characterizing countries of origin, participant ages, measurement of obesity status and depression, and the use of covariates. Our second aim was to evaluate the strength of evidence for prospective obesity-to-depression and depression-to-obesity associations.

Methods

Pertinent articles were identified using a comprehensive literature search strategy. This included examining reference lists from review articles (10,20–22) and an electronic database (Medline). The electronic database search involved an electronic search that crossed the terms *obesity* and *depression*. The literature search did not include doctoral dissertations or unpublished works.

Studies were included if they were written in English, had a period of data collection of at least 1 year, and examined the relationship between depression and obesity in a prospective manner. Studies were included only if the sample was community- or population-based. Studies were required to have a minimum sample size of 100 participants. Studies were excluded if the sample was comprised only of treatment-seeking participants. For example, studies were excluded if the aim was to examine change in weight (or depression) during or after treatment for depression (or weight loss). Qualitative reports were excluded.

A total of 22 studies were identified that met our inclusion criteria. These studies yielded 10 analyses of obesity-to-depression associations (Table 1) and 15 analyses of depression-to-obesity associations (Table 2). Nineteen studies presented findings only on one temporal association (i.e. obesity-to-depression, or depression-to-obesity), while three presented findings on both pathways (23–25). We coded each of the studies with respect to the following methodological attributes: year of publication, sample size; method of weight and height measurement (i.e. reported vs. directly measured); method of depression measurement (i.e. self-report questionnaire vs. structured diagnostic interview); covariates (i.e. number and type); whether the analysis used incidence methodology (i.e. excluded participants who exhibited the outcome variable of interest at baseline); analytic treatment of weight status (i.e. continuous BMI, weight or weight change vs. categorical obesity status); analytic treatment of depression (i.e. continuous score vs. diagnostic categorization of depression); the covariates-adjusted relationship between depression and obesity, including the effect size (e.g. odds ratio, regression coefficient); the unadjusted relationship between obesity and depression, if provided; and whether or not sex and race were examined as moderators.

For the first study aim, we calculated descriptive statistics (percentages, means and standard deviations) for each of the above methodological attributes. We also identified the most commonly used assessment tools for measuring depression, and the frequency with which various covariates were used. For the second aim, we tallied the number of studies that reported significant obesity-to-depression (Table 1) and depression-to-obesity pathways (Table 2), respectively. As noted below, the number of covariates differed greatly across studies. Thus, when reviewing each study and determining whether there was a significant result, we also looked to the most highly covariates-adjusted results. For studies that provided data both for covariates-adjusted and unadjusted results, we reviewed the extent to which findings changed when covariates were not included in the statistical model. Finally, we determined the number of studies that found evidence for sex- or ethnicity-moderation and described the nature of the interaction. Given the significant methodological heterogeneity of the studies reviewed and the inconsistent treatment of covariates (as described in our Results), we decided that a formal meta-analysis would not be appropriate or meaningful.

Results

Obesity as a predictor of depression

Study characteristics

There was striking variability in the methodological attributes of the 10 studies that tested obesity as a predictor of depression (Table 3). Most studies, 70%, were conducted in the USA. Sample sizes ranged from 446 to 74 332 persons and follow-up periods from 1 to 31 years. Four studies examined paediatric samples, one of which began in childhood (24) and three in adolescence (23,26,27). Among the six non-paediatric studies, two began in earlier adulthood (i.e. ages 20 and older) (28,29) and four in middle adulthood (ages 50 and older) (25,30–32).

Five studies (50%) used reported weights and heights along with questionnaire assessment of depression (23,24,27,28). Four studies (40%) used reported weights and heights and interview assessment of depression (25,26,30,31). One study (10%) used measured weights and heights and questionnaire assessment of depression (29), and no study used directly measured weights and heights in conjunction with interview assessment of depression.

Table 1 Studies in which obesity is examined as a predictor of depression

Fist author (reference number)	Year	Design	*n*	Obesity measure*	Depression measure	Covariates (number)	Incidence analysis?†	Observed relationship (with covariates adjusted)	Odds ratio (95% confidence interval) or other statistics reported	Unadjusted result	Sex as moderator	Race as moderator
Anderson (26)	2007	Prospective community-based study of an adolescent cohort in upstate New York, followed for 20 years	661	Parent and SR of weight and height (categorical)	Diagnostic Interview Schedule for Children (categorical: diagnosis of MDD)	SES, race, smoking, parent psychopathology (4)	Yes	Females who were obese as adolescents had increased risk of depression at follow-up, compared with non-obese adolescents; NS for males.	Hazard ratios: males: not provided; females: 3.2 (1.1, 9.5).	N/A	Sig.	N/A
Ball (28)	2009	Prospective community-based study of females ages 22–27 in Australia, followed for 3 years	6 677	SR weight and height (categorical)	CES-D; (categorical: scores 10 or higher = depression)	Education, marital status, occupation, smoking, parity, serious health problems, physical activity, baseline depression level (8)	No	Obese participants, compared with normal weight, had higher risk for depression at follow-up.	1.33 (1.08,1.65)	1.81 (1.50, 2.18)	N/A	N/A
Bjerkeset (29)	2008	Prospective community-based study of adults age 20 and older in Norway, followed for ~10 years	7 4332	Measured weight and height (categorical)	Hospital Anxiety and Depression Rating Scale – Depression items (categorical: scores 8 or higher = depression)	Age, sex, education, marital status, smoking, alcohol use, physical activity, vigour, nervousness, calmness, cheerfulness, tranquilizer use (12)	No	Obese participants, compared with normal weight, had higher risk for depression at follow-up.	All participants: 1.29 (1.14, 1.45). Males: 1.41 (1.17, 1.70). Females: 1.21 (1.03, 1.41).	N/A	N/A	N/A
Goodman (23)	2002	Prospective, community-based study of adolescents, followed for 1 year	9 374	SR weight and height (categorical)	CES-D (categorical: scores of 24 or higher for females and 22 or higher for males = depression)	Age, sex, race, parental obesity, parent education, number parents at home, baseline CES-D score (7)	No	Those who were obese at baseline did not have increased risk of depression at follow-up.	1.16 (0.81–1.65)	NS ($P = 0.43$)	NS	NS

Table 1 *Continued*

Fist author (reference number)	Year	Design	*n*	Obesity measure*	Depression measure	Covariates (number)	Incidence analysis?†	Observed relationship (with covariates adjusted)	Odds ratio (95% confidence interval) or other statistics reported	Unadjusted result	Sex as moderator	Race as moderator
Herva (27)	2006	Prospective, community-based study of adolescents in Finland, followed for 31 years	8 451	SR weight and height (categorical)	HSCL (categorical: three different score cut-offs used = 1.55, 1.75, 2.01)	Father's social class, family type, chronic diseases, smoking, alcohol use (5)	No	When using cut-off of 1.75, female participants with paediatric obesity at age 14, compared with normal weight participants, had higher risk of depression at age 31. When using cut-off of 2.01, male participants with paediatric obesity at age 14, compared with normal weight participants, had higher risk of depression at age 31.	HSCL cut-off of 1.55: males: 1.08 (0.72, 1.61); females: 1.33 (0.97, 1.83). HSCL cut-off of 1.75: males: 1.18 (0.73, 1.89); females: 1.64 (1.16, 2.32). HSCL cut-off of 2.01: males: 1.97 (1.06–3.68); females: 1.55 (0.93, 2.59).	Same pattern of results. HSCL cut-off of 1.55: males: 1.15 (0.79, 1.69); females: 1.34 (0.98, 1.82). HSCL cut-off of 1.75: males: 1.26 (0.80, 1.96); females: 1.63 (1.16, 2.29). HSCL cut-off of 2.01: males: 1.94 (1.07, 3.52); females: 1.49 (0.89, 2.47).	Sig.	N/A
Rhew (24)	2008	Prospective study of sixth grade students in Seattle, followed for 1 year	446	SR weight and height (categorical)	Mood and Feelings Questionnaire (continuous)	Sex, household income, education of primary caregiver, physical development, race, baseline depression score (6)	No	Overweight participants, compared with normal weight participants, did not have significantly different depression scores at follow-up.	Beta = –0.08, $P = 0.52$	N/A	NS	N/A
Roberts (30)	2000	Prospective, community-based Alameda County study of adults age 50 and up, followed for 1 year	2 098	SR weight and height; obesity defined with standard BMI categories or as being at 85th percentile or above (categorical)	Items from PRIME-MD (categorical: MDD diagnosis)	Age, sex, marital status, education, daily activities, chronic conditions, financial stress, life events, social isolation and support (10)	Yes	Obese participants, defined with 85% cut-off, had higher rates of depression at follow-up than normal weight subjects. However, when BMI > 30 cut-off is used, result was NS.	85th percentile cut-off: 1.73 (1.04, 2.87). BMI > 30 cut-off: 1.43 (0.85, 2.43)	Both significant unadjusted: 85th percentile cut-off: 1.91 (1.18, 3.08). BMI > 30 cut-off: 1.67 (1.02, 2.74).	N/A	N/A
Roberts (31)	2002	Prospective, community-based Alameda County study of adults age 50 and up, followed for 5 years	1 739	SR weight and height (categorical)	Items from PRIME-MD (categorical: MDD diagnosis)	Age, sex, education, marital status, chronic medical conditions, activities of daily living, physical activity, financial strain, recent life events, social isolation, social support (11)	Yes	Obese participants had higher rates of depression at follow-up than normal weight participants. (Note: when participants who were depressed at baseline were retained in analysis, result was NS.)	1.77 (1.03, 3.05)	N/A	NS	N/A

Table 1 *Continued*

Fist author (reference number)	Year	Design	*n*	Obesity measure*	Depression measure	Covariates (number)	Incidence analysis?†	Observed relationship (with covariates adjusted)	Odds ratio (95% confidence interval) or other statistics reported	Unadjusted result	Sex as moderator	Race as moderator
Roberts (25)	2003	Prospective, community-based Alameda County study of adults age 50 and up, followed for 5 years	1 748	SR weight and height (categorical)	Items from PRIME-MD (categorical: MDD diagnosis)	Age, sex, education, marital status, financial strain, recent life events, social isolation, social support, chronic conditions, difficulty with usual daily activities (10)	Yes	Obese participants had higher rates of depression at follow-up than non-obese. (Note: when participants who were depressed at baseline were retained in analysis, result was NS.)	1.79 (1.06, 3.02)	2.09 (1.44, 3.03)	NS	N/A
Sachs-Ericsson (32)	2007	Prospective, community-based study of adults over 65 in North Carolina, followed for 3 years	2 406	SR weight and height (continuous)	CES-D (continuous)	Depression, age, sex, race, income, education, smoking, physical functioning at time 1 and 2, chronic health conditions at time 1 and 2, self-rated health at time 1 and 2 (13)	No	BMI predicted depression at follow-up. Race was a significant moderator: Influence of BMI on depression was greater for African–American than Caucasian participants. Race x BMI x Education interaction also was significant: influence of BMI on depression was greatest for African–Americans with less education; depression scores among less educated Caucasian subjects were high regardless of BMI.	BMI as a predictor of depression: beta = 0.05, standard error = 0.02.	N/A	NS	Sig.

*Standard adult or paediatric categories (as appropriate for age of sample) were used to classify obesity unless otherwise noted.

†Studies were coded as having conducted an incidence analysis if participants who were obese at baseline were excluded from analyses.

BMI, body mass index; CES-D, Center for Epidemiological Studies Depression Scale; HSCL, Hopkins Symptom Checklist; MDD, major depressive disorder; N/A, not analyzed or applicable; NS, not significant; PRIME-MD, Primary Care Evaluation for Mental Disorders; SES, socioeconomic status; Sig., significant; SR, self-report.

Table 2 Studies in which depression is examined as a predictor of obesity

First author (reference number)	Year	Design	*n*	Obesity measure*	Depression measure	Covariates (number)	Incidence analysis?†	Observed relationship (with covariates adjusted)	Odds ratio (95% confidence interval) or other statistics reported	Unadjusted result	Sex as moderator	Race as moderator
Barefoot (43)	1998	Prospective study of North Carolina college students, followed for ~20 years	4726	Baseline = measured, in college records; follow-up = SR of weight and height (change in BMI = DV)	MMPI Obvious Depression Scale (categorical: 1 standard deviation above norms = depression)	Sex, baseline BMI, exercise, smoking (4)	N/A	Participants with depression gained less weight than non-depressed if initially lean, but gained more weight if initially heavy.	b (unstandardized) = –19.68, $P < 0.01$.	N/A	NS	N/A
Bardone (44)	1998	Prospective cohort study of adolescent females followed from age 15 to age 21 in New Zealand	459	Measured weight and height (BMI = DV)	DISC (categorical; diagnosis of MDD or dysthymia = depression)	SES, parent smoking, presence of father figure, childhood health, maternal health, maternal BMI, age at menarche (7)	N/A	Depression at age 15 did not predict BMI at age 21.	Beta = 0.05	Beta = 0.06 (NS)	N/A	N/A
Chiriboga (49)	2008	Prospective community-based study of adults in Central Massachusetts, followed for 1 year	572	Measured weight (weight = DV)	Beck Depression Inventory (continuous)	Age, education, marital status, height, caloric intake, % calories from fat, % calories from carbohydrates, % calories from protein, fibre intake, alcohol, physical activity, smoking, season (13)	N/A	Depression did not predict weight change.	Regression coefficient: males: 0.00 (NS), females: –0.02 (NS).	Regression coefficient: males: 0.026 (NS), females: –0.014 (NS).	NS	N/A
DiPietro (48)	1992	Prospective community-based national sample (NHANES I) of adults 25–74 years old in USA, followed for ~8 years	1794	Measured height and weight (weight change = DV)	CES-D (categorical: 16 or higher = depressed)	Education, smoking, alcohol use, physical activity, parity, age, BMI, follow-up time (7)	N/A	Young males depressed at baseline gained more weight over follow-up than non-depressed; education moderated effect (those with less education gained more). Young females depressed at baseline gained less weight over follow-up than non-depressed (those with less education gained less weight). Older males and females depressed at baseline lost marginally more weight than non-depressed ($P = 0.06$).	Betas: young males: 6.2 (2.9, 9.4); young females: –3.2 (–5.7, –0.8) older adults: –2.2 (–4.4, 0.1).	N/A	Sig.	N/A

Table 2 *Continued*

First author (reference number)	Year	Design	*n*	Obesity measure*	Depression measure	Covariates (number)	Incidence analysis?†	Observed relationship (with covariates adjusted)	Odds ratio (95% confidence interval) or other statistics reported	Unadjusted result	Sex as moderator	Race as moderator
Franko (45)	2005	Prospective, community-based study of females in USA (NGHS) followed from age 16 to 21	1554	SR weight, measured height (categorical and continuous)	CES-D (continuous)	Race, site, parent education, prior BMI (4)	No	Higher depression score at age 16 or 18 predicted increased risk of obesity at age 21. Same pattern true when BMI was examined as a continuous DV.	Depression at age 16: 3.11 (1.13, 5.12). Depression at age 18: 3.50 (1.26, 5.80).	N/A	N/A	NS
Goodman (23)	2002	Prospective, community-based study of USA adolescents, followed for 1 year (Add Health)	9374	SR weight and height (categorical)	CES-D (categorical: scores of 24 or higher for females and 22 or higher for males = depression)	Age, sex, race, parental obesity, parent education, number parents at home, self-esteem, smoking, conduct disorder, physical activity, baseline BMI (11)	Yes	Participants with depression at had higher risk of obesity at follow-up.	2.39 (1.05, 5.45)	$P = 0.048$	NS	NS
Goodwin (40)	2009	Prospective, community-based study of males in Finland from age 8 to 18–23	2712	Diagnosis of obesity by physician using ICD-10 criteria during military examination (categorical)	Children's Depression Inventory (categorical: below 50% = no depression, 50–90% = moderate depression, 90% or up = severe depression)	Mother's education, somatic health problems at age 8 (2)	No	Depression in childhood did not predict increased risk of obesity in young adulthood.	Moderate depression: 1.4 (0.9, 2.3); severe depression: 0.5 (0.2, 1.4).	Moderate depression: 1.4 (0.9, 2.3); severe depression: 0.7 (0.3, 1.8).	N/A	N/A
Hasler (46)	2005	Prospective, community-based study of young adults in Zurich followed from age 19 to 40	591	SR weight and height (categorical)	Retrospective interview at age 19 about presence of depressive symptoms during childhood	BMI at age 20, physical activity, SES, family history of weight problems, stratified sampling (5)	No	Females who reported childhood depression were at higher risk for adult obesity. NS for males.	Hazard ratio: females: 11.52, standard error: 1.24; males: 1.10, standard error: 0.66.	Females with childhood depression had higher adult BMI than those without history ($P < 0.01$). NS for males.	Sig.	N/A
Pine (41)	1997	Prospective, community-based study of adolescents in upstate New York, followed for 9 years, from age 9–18 to age 17–28	644	SR weight and height. BMI continuous and categorical (obesity = BMI above 80th percentile; 25.86 for females and 27.31 for males)	DISC (continuous: number of MDD symptoms)	Physical health, smoking, social class, IQ, parent sociopathy, conduct disorder (6)	No	Adolescent depression did not predict BMI in adulthood (however, unadjusted results were significant for females).	Regression coefficient: 0.15 (0.20).	Prediction of obesity status: males: 1.46 (0.78, 2.74); females: 3.06 (1.91, 4.91).	NS	N/A

Table 2 *Continued*

First author (reference number)	Year	Design	*n*	Obesity measure*	Depression measure	Covariates (number)	Incidence analysis?†	Observed relationship (with covariates adjusted)	Odds ratio (95% confidence interval) or other statistics reported	Unadjusted result	Sex as moderator	Race as moderator
Pine (42)	2001	Prospective study of New York city children age 6–17 with and without MDD, followed for 10–15 years	177	SR weight and height; BMI continuous and categorical	SADS (categorical: MDD diagnosis)	Age, sex, cigarette use, alcohol use, social class, pregnancy, medication history (7)	Yes	For most covariate models, but not all, participants who had childhood depression had higher BMI as adults than participants without childhood depression.	OR for overweight adjusted for childhood poverty = 1.7 (0.9, 3.2). OR for other models not provided.	Children with MDD had higher BMI as adults than children without MDD ($t(175) = 2.7$, $P < 0.01$) and had higher risk of being overweight as adults (1.9 (1.02, 3.4))	NS	N/A
Richardson (39)	2003	Prospective, community-based study of children in New Zealand, followed from birth to age 26	1037	Measured weight and height (categorical)	DISC (categorical: MDD diagnosis)	Childhood BMI, parental obesity, SES, maternal depression (4)	Yes, but stats not reported – same results	Depression in early adolescence (age 11–15) was not a significant predictor of obesity at age 26 (females and males not analysed separately). Females with depression in late adolescence (age 18–21) had increased risk of obesity at age 26. NS for males.	Relative risk ratios: Early adolescence: 0.50 (0.19–1.28). Late adolescence: females: 2.32 (1.29–3.83); males: 0.90 (0.37–2.02).	N/A	Sig.	N/A
Rhew (24)	2008	Prospective study of sixth grade students in Seattle, followed for 1 year	446	Measured or SR weight and height (continuous)	Mood and Feelings Questionnaire (categorical: 15 or higher = depression)	Household income, education of primary caregiver, pubertal stage, race, baseline BMI (5)	N/A	Using SR weight and height: Depressed males experienced smaller increases in BMI over 1 year than non-depressed males. Depressed females experienced larger increases in BMI over 1 year than non-depressed females. Using measured weight and height: NS effects.	Using SR weight and height: males: –1.18 (–1.99, –0.37); females: 2.49 (1.44, 3.54). Using measured weight and height: males: 0.12 (–0.49, 0.73); females: 0.54 (–0.42, 1.51).	SR: males: –0.48 (–1.64, 0.67); females: 3.16 (1.91, 4.40).	Sig.	N/A

Table 2 *Continued*

First author (reference number)	Year	Design	*n*	Obesity measure*	Depression measure	Covariates (number)	Incidence analysis?†	Observed relationship (with covariates adjusted)	Odds ratio (95% confidence interval) or other statistics reported	Unadjusted result	Sex as moderator	Race as moderator
Roberts (25)	2003	Prospective, community-based Alameda County study of adults age 50 and older, followed for 5 years	1748	SR weight and height (categorical)	PRIME-MD (categorical: MDD diagnosis or not)	Age, sex, education, marital status, financial strain, recent life events, social isolation, social support, chronic conditions, activities of daily living (10)	Yes	Participants with depression did not have greater risk of obesity at follow-up. However, when analyses were unadjusted and outcome was prevalence, not incidence, depression did raise risk for obesity.	1.01 (0.46, 2.22)	1.32 (0.65, 2.70)	NS	N/A
Stice (47)	2005	Prospective, community-based study of females age 11–15 at baseline followed for 4 years in South-western US city	496	Measured weight and height (categorical)	SADS (continuous: symptom severity index)	Dietary restraint, compensatory behaviours, perceived parental obesity (3)	Yes	Depressive symptoms at baseline did not predict obesity onset during 4-year follow-up (however, was significant unadjusted).	2.32 (0.62–8.65)	4.62 (1.67, 12.74)	N/A	N/A
Vogelzangs (50)	2008	Prospective community-based study of adults age 70–79 in Pennsylvania and Tennessee, followed for 5 years	2088	Measured weight and height (continuous)	CES-D (continuous or categorical: scores 16 or higher = depression)	Sex, age, race, site, education, alcohol, physical activity, diabetes, CVD, other chronic diseases, prescription medications (11)	No	Baseline depression (measured categorically or continuously) did not predict subsequent BMI.	Betas: depression as continuous variable = 0.03, $P = 0.11$; as categorical = 0.03, $P = 0.18$.	N/A	NS	NS

*Standard adult or paediatric categories (as appropriate for age of sample) were used to classify obesity unless otherwise noted.

†Studies were coded as having conducted an incidence analysis if participants who were obese at baseline were excluded from analyses.

BMI, body mass index; CES-D, Center for Epidemiological Studies Depression Scale; CVD, cardiovascular disease; DISC, Diagnostic Interview Schedule for Children; DV, dependent variable; NHANES, National Health And Nutrition Examination Survey; ICD-10, International Classification of Diseases-10; MDD, major depressive disorder; MMPI, Minnesota Multiphasic Personality Inventory; N/A, not analyzed or applicable; NGHS, National Heart, Lung, and Blood Institute Growth and Health Study; NS, not significant; PRIME-MD, Primary Care Evaluation for Mental Disorders; SADS, Schedule for Affective Disorders and Schiozphrenia; SES, socioeconomic status; Sig. significant; SR, self-report.

Table 3 Characteristics of all included studies

	Studies ($n = 10$) in which obesity was examined as a predictor of depression	Studies ($n = 15$) in which depression was examined as a predictor of obesity	All studies ($n = 25$)
Location (*n*, %)			
USA	7 (70.0)	11 (73.3)	18 (72.0)
Europe (non-Scandinavia)	0 (0.0)	1 (6.6)	1 (4.0)
Scandinavia	2 (20.0)	1 (6.6)	3 (12.0)
Australia/New Zealand	1 (10.0)	2 (13.3)	3 (12.0)
Sample at baseline (*n*, %)			
Children	1 (10.0)	4 (26.6)	5 (20.0)
Adolescents	3 (30.0)	7 (46.6)	10 (40.0)
Adults	2 (20.0)	2 (13.3)	4 (16.0)
Older adults	4 (40.0)	2 (13.3)	6 (24.0)
Anthropometric assessment (*n*, %)			
Self-report	9 (90.0)	5 (33.3)	14 (56.0)
Measured	1 (10.0)	7 (46.6)	8 (32.0)
Both	0 (0.0)	3 (20.0)	3 (12.0)
Depression assessment (*n*, %)			
Questionnaire	6 (60.0)	8 (53.3)	14 (56.0)
Interview	4 (40.0)	7 (46.6)	11 (44.0)
Incidence analysis conducted (*n*, %)			
Yes	4 (40.0)	5 (33.3)	9 (36.0)
No	6 (60.0)	10 (66.6)	16 (64.0)
Covariates modelled (*n*, %)			
Yes	10 (100.0)	15 (100.0)	25 (100.0)
No	0 (0.0)	0 (0.0)	0 (0.0)
Effect size estimate (*n*, %)			
Odds ratio, hazard ratio, or relative risk ratio	8 (80.0)	9 (60.0)	17 (68.0)
Regression coefficient or beta weight	2 (20.0)	6 (40.0)	8 (32.0)
Relationship found between depression and obesity (*n*, %)			
Significant	8 (80.0)	8 (53.3)	16 (64.0)
Non-significant	2 (20.0)	7 (46.6)	9 (36.0)
Sex examined as a moderator (*n*, %)			
Yes	7 (70.0)	11 (73.3)	18 (72.0)
No	3 (30.0)	4 (26.6)	7 (28.0)
Sex significant as a moderator	2 (20.0)	4 (26.6)	6 (24.0)
Race examined as a moderator (*n*, %)			
Yes	2 (20.0)	3 (20.0)	5 (20.0)
No	8 (80.0)	12 (80.0)	20 (80.0)
Race significant as a moderator	1 (10.0)	0 (0.0)	1 (4.0)

Nine studies treated obesity status as a categorical predictor, whereas the remaining study used BMI as continuous measure. Eight studies treated depression as a categorical/diagnostic outcome, and the remaining two studies examined depression as a continuous measure. The most commonly used measures were the Center for Epidemiological Studies Depression Scale (33), which was used in three investigations (23,28). The Primary Care Evaluation of Mental Disorders (PRIME-MD) (34) was also used in three reports (25,30,31). Other measures of depression included the Diagnostic Interview Schedule for Children (35) ($n = 1$), the Hospital Anxiety and Depression Rating Scale – Depression subscale (36) ($n = 1$), the Hopkins Symptom Checklist (37) ($n = 1$) and the Mood and Feelings Questionnaire (38) ($n = 1$). The number of covariates varied from 4 to 13 across studies with a mean ± standard deviation of 8.6 ± 3.1 covariates.

Evidence for prospective associations

Eight of the 10 studies (80%) found that obesity, or increased body mass index (BMI), was associated prospectively with depression onset or elevated depression levels (25–32), with covariates-adjusted odds ratios generally in the range of 1.0 to 2.0. Anderson *et al.* (26) found that obese, compared with non-obese, adolescent females, had a significantly higher risk of developing major depressive disorder over a 20-year follow-up period. Similarly, Ball *et al.* (28) found that among young women, those who were obese at baseline had a significantly higher risk of depression at 3-year follow-up than those who were normal

weight at baseline. In a study by Bjerkeset *et al.* (29), adults who were obese at baseline, compared with those of normal weight, had a significantly higher risk for depression at follow-up, which occurred approximately 10 years after baseline. Herva *et al.* (27) found that participants who were obese at age 14 had higher rates of depression at age 31 than those who were normal weight at age 14. Roberts *et al.* (25,30,31) conducted three studies with participants in the Alameda County study, who were all age 50 years or older at baseline. Across all studies, they found that participants who were obese at baseline had greater risk for depression than normal weight participant, over follow-up periods ranging from 1 to 5 years. Finally, a study of older adults conducted by Sachs-Ericsson found that BMI predicted depressive symptoms, such that participants with the highest BMI at baseline had a greater number of depressive symptoms at 3-year follow-up (32). It should be noted that, out of these eight studies reporting significant prospective associations, four conducted incidence analyses that tested for the onset of new cases of depression (25,26,30,31) (see Table 1).

Two of the 10 studies had null findings (23,24). Both of these investigations were conducted with paediatric participants who were followed only for 1 year.

No studies were found in which a null unadjusted obesity–depression association became significant when covariates were added to the model. However, Roberts *et al.* (30) reported unadjusted associations that were significant but which became non-significant when adjusting for covariates.

Seven studies examined sex as a moderator of the relationship between obesity and depression (23–27,31,32), of which two reported significant findings. One study found that obesity predicted depression in females but not males (26). Another investigation reported that obese females, but not males, had a higher risk of depression when a moderate-severity criterion of depression was used. However, obese males, but not females, had a higher risk of depression when a high-severity criterion of depression was used (27).

Only two studies tested race/ethnicity as a moderator (23,32), one of which found a significant effect (23). Sachs-Ericsson *et al.* (32) found that the influence of BMI on depression was greater among African–American than Caucasian participants. Race and education also interacted with depression, in that the influence of BMI on depression was greatest for African–American participants with less education.

Depression as a predictor of obesity

Study characteristics

Studies that examined depression as a predictor of obesity also were characterized by marked methodological variability (Table 3). Eleven studies (73%) were conducted in the USA. Sample size ranged from 177 to 9374 participants, and follow-up periods from 1 to 21 years. Eleven studies examined paediatric samples, four of which began in childhood (39–42) and seven in adolescence (23,24,43–47). Among the four non-paediatric studies, two began in earlier adulthood (ages 25 and older) (48,49) and two in middle adulthood (ages 50 years and older) (25,50). Three studies (20%) relied on reported weight and height and on questionnaire assessment of depression (23,43,45). Four studies (27%) used self-report of weight and height, with interview assessment of depression (25,41,42,46). Five studies (33%) measured weight and height and used questionnaire assessment of depression (24,40,48–50). Only three studies (20%) measured weight and height and used interview assessment of depression (39,44,47).

Six studies treated obesity status as a categorical predictor, six used BMI as a continuous measure and three used both categorical and continuous measures. Ten studies treated depression as a categorical/diagnostic outcome, whereas four examined it as a continuous measure, and one report analysed both categorical and continuous measures of depression. The most common measures of depression were the Center for Epidemiological Studies Depression Scale (33) ($n = 4$) and the Diagnostic Interview Schedule for Children (35) ($n = 3$). Other measures included the Children's Depression Inventory (51,52) ($n = 2$), the Schedule for Affective Disorders and Schizophrenia ($n = 2$), the PRIME-MD (34) ($n = 1$), the Mood and Feelings Questionnaire (53) ($n = 1$), the Minnesota Multiphasic Personality Inventory Obvious Depression Scale (54) ($n = 1$), the Beck Depression Inventory (55) ($n = 1$) and an unstructured clinical interview ($n = 1$). The number of covariates ranged from 2 to 14 across studies, with a mean $\pm$ standard deviation of 6.6 $\pm$ 3.3 covariates.

Evidence for prospective associations

Eight of the 15 studies (53%) found that depression was a significant predictor of obesity, BMI or weight gain over time (23,24,39,42,43,45,46,48). In most of these studies, depression was found to increase risk for obesity (23,39,42,45,46,48), with odds ratios generally in the range of 2.0 to 3.0. We note that, out of these eight studies reporting significant prospective associations, four conducted incidence analyses that tested for the onset of new cases of obesity (23,25,39,42) (see Table 2). Franko *et al.* (45) found that participants with higher levels of depression at age 16 or 18 years had a greater risk of obesity at age 21. In a sample of adolescents followed for 1 year, Goodman *et al.* (23) found that those with depression at baseline, compared with non-depressed counterparts, had a greater risk of obesity at follow-up. Hasler *et al.* (46) found that females who experienced depression during childhood, compared with females who did not, had a

greater risk for obesity in adulthood. Pine *et al.* (42) found that children who had depression had a higher BMI as adults than children who had been free of depression. Richardson *et al.* (39) found that females who experienced depression in late adolescence had increased risk for obesity at age 26.

Barefoot *et al.* (43) found that the influence of depression depended on initial weight. In this study, which followed college students for approximately 20 years, participants who were depressed at baseline, compared with non-depressed participants, gained more weight over time if they were initially high in BMI, but gained less weight over the follow-up period if they were initially low in BMI. DiPietro *et al.* (48) found that young men who were depressed at baseline gained more weight over the follow-up period (approximately 8 years) than those who had not been depressed. However, as noted below, depression increased risk of weight gain for women. Rhew *et al.* (24) found the opposite: boys who were depressed at baseline had smaller increases in (self-reported) BMI over a 1-year follow-up period than non-depressed boys, and girls who were depressed at baseline experienced larger increases in BMI during the 1-year follow-up period than non-depressed girls. However, they found that when *measured* weight and height were used in a subsample of participants, these results became non-significant.

Interestingly, not all of the eight studies reporting significant depression-obesity associations reported positive relationships. There was evidence from three studies that some groups of depressed participants had a *lower* risk for obesity if they had a history of depression (24,43,48).

Eleven studies examined sex as a moderator of the relationship between obesity and depression (23–25,39,41–43,46,48–50,56). Of these, four studies found evidence for moderation, with three reporting that depression raised the risk for obesity or increased weight gain only among females (24,39,46). DiPietro *et al.* found that a history of depression was associated with *reduced* weight gain among females but *greater* weight gain among males (48). This study also found that sex interacted with age and education in moderating the depression-to-obesity association. Specifically, young depressed men with less education gained *more* weight compared with their more highly educated counterparts. In contrast, young depressed women with less education gained *less* weight compared with their more highly educated counterparts. There was no significant evidence that race/ethnicity moderates depression-to-obesity associations in three studies that tested this effect (23,45,50).

The modelling of covariates influenced results. Three studies reported instances in which results were significant when unadjusted but non-significant when particular covariates were entered into the model (41,42,47).

Discussion

This review finds good evidence for obesity-to-depression prospective associations, with 80% of the studies reporting significant covariates-adjusted associations among these disorders. In contrast, evidence for depression-to-obesity pathways was less consistent, with only 53% of the reviewed studies finding significant associations. Thus, in the general population, being obese increases the risk of developing depression, with odds ratios generally in the range of 1.0 to 2.0. However, being depressed does necessarily or consistently increase the risk of becoming obese. As we only reviewed population-based samples, the null findings likely were not due to low statistical power.

An important question for the field concerns the causal mechanisms that underlie obesity-to-depression pathways in the population. Stigma and social prejudices against obesity may mediate this link, as there is extensive evidence of antifat attitudes (57–60). Such biases and weight teasing have been documented among health professionals, peers and even family members (61,62). Weight teasing was associated prospectively with elevated depression in a population-based sample of adolescents (63), as well as with greater suicidal thoughts and attempts (64). Functional impairment, which is greater among obese individuals (65), may also mediate the effect of weight status on depression. Obese individuals have greater difficulties with mobility and poorer health-related quality of life (66). Physiological or endocrinological mechanisms, such as those related to stress response or inflammation (67,68), might also be pathways mediating the relationship between obesity and depression.

Evidence was mixed for depression-to-obesity pathways although, among those studies reporting significant associations, odds ratios generally in the range of 2.0 to 3.0. Thus, it is important to consider potential mechanisms by which depression may promote obesity in certain subgroups. First, several classes of antidepressant medications are associated with undesired excess weight gain as a side effect (69). Tricyclics are associated with the greatest weight gain, followed by certain types of selective serotonin re-uptake inhibitors – paroxetine (70) and mertazapine (71). Another potential pathway by which depression may promote obesity is through impaired sleep quality. Poorer sleep quality is common in depression (72,73), and inadequate sleep is a risk factor for obesity (74). Another potential mechanism for individuals with atypical depression is hyperphagia, as over-eating is a hallmark feature of this disorder (75–77). Finally, depression is associated with physical inactivity (78,79), which could promote excess weight gain.

Rather than there being a single obesity–depression association for *all* individuals in the population, our review suggests that there are *subgroups* of individuals among

whom these relationships exist but not others. Among those studies conducting formal moderation analyses, we found relatively consistent evidence for sex moderation, with prospective associations being found in females but not males. This was true in studies that tested obesity-to-depression pathways (26) and depression-to-obesity pathways (41,46). This is consistent with findings from cross-sectional studies (16,49). In contrast, only five studies in this review formally tested for ethnicity-moderation effects, of which only one found evidence to support this (32). Future studies need to evaluate the potential moderating role of ethnicity, as well as other demographic factors. For example, cross-sectional studies suggest that education level (potentially a proxy for socioeconomic status) and degree of obesity (80) may moderate the association among depression and obesity. Prospective studies need to evaluate these variables as potential moderators.

This review revealed a number of findings regarding methodology. First, we found that a wide range of covariates has been modelled in the literature, ranging from 2 to 13 across studies. There was no consistent rule for covariate selection across studies, and it was not always clear how findings within studies would have changed if controlling for different variables. This lack of consistency in the modelling of covariates is a major limitation in the literature that hinders its systematic review.

Second, we found that most studies used self-report measures of weight and height, rather than direct measures, and assessed depression using questionnaires rather than clinical interview. Moreover, only 12% of the studies reviewed used both directly measured weight and height and clinical assessment of depression. This is a limitation in the field, as under-reporting of weight and over-reporting of height are well established, (81,82) and interviews have greater diagnostic specificity. State-of-the-art assessment of both body composition and depression should be prioritized in future studies.

Third, 72% of the studies were conducted in North America. More research is needed on this topic globally, as well as the potential impact of cultural attitudes and beliefs about both mental health disorders and obesity.

There are several clinical implications of this review. Healthcare providers should be alert to the possibility of depression in patients with obesity and screen for depressive symptoms appropriately. Similarly, providers should recognize that some patients with depression might be at increased risk for obesity. Healthcare providers should monitor weight change in these patients and provide recommendations for weight control, as needed. To the extent that depression and obesity co-occur in some individuals, efforts to prevent obesity might have the added benefit of reducing prevalence of depression, and programmes that target depression prevention might have the added benefit of reducing prevalence of obesity. Treatment programmes that reverse obesity or depression over the long term might also reduce the risk for the co-occurring disorder.

This review has several caveats that should be considered. First, the heterogeneity of the studies with respect to uniform covariates, statistical analyses and reported effect size estimates did not allow for a formal meta-analysis to be conducted. Second, we included certain studies in which incidence analyses were not conducted, allowing participants with the target outcome at baseline to be included in analyses. Third, most studies did not formally test multiple moderators of the obesity–depression relationship, limiting the extent to which one can understand moderation of this relationship. Fourth, this review excluded cross-sectional studies and clinical samples, which limited the number of studies that could be included but strengthened the conclusions that could be drawn.

Based on the present findings, we propose a number of study sample, measurement and data analysis recommendations to guide future research. These suggestions should not be treated as exhaustive. Regarding samples, there is a need for more research with ethnically diverse populations given evidence that depression-obesity associations differ by race/ethnicity (19). Only 20% of the studies in our review formally tested for effect moderation by race/ethnicity. There also is need for a more globally representative data base, as 70% of the studies in our review were conducted in the USA. That obesity and depression are worldwide health problems underscores this recommendation. Additionally, prospective research beginning at childhood is warranted, as only 20% of the studies did this. Findings from paediatric studies might help to inform early prevention strategies before the onset of either disorder.

Regarding measurement, future studies are encouraged to use standardized and validated assessments of depression as well as direct rather than reported measures of weight and height, given the reporting inaccuracies associated with the latter methodology (83,84). Interestingly, only 12% of the studies in our review used *both* direct measures of weight and height along with interview assessment of depression, underscoring this need. New insights also might be attained through refined body composition assessments that can better differentiate lean and fat mass, as illustrated by Tanofsky-Kraf *et al.*'s use of dual energy X-ray absorptiometry (85). Finally, studies that measure proposed mediating variables would be informative. Mediators could be psychosocial (e.g. exposure to weight discrimination), physiological (e.g. adipokines) or pharmacological (e.g. medication use). These and other mechanisms that potentially link obesity and depression need to be directly assessed.

Regarding data analysis, future investigations should clearly state 'rules' for the manner in which covariates are modelled, given the diversity of approaches in the literature. Studies should present both unadjusted and

covariates-adjusted associations, to clarify how zero-order associations between obesity and depression change in the presence of other variables. The reasons for including specific covariates in competing statistical models should be stipulated a priori. Exploratory or post hoc analyses with covariates should be identified as such. Beyond the topic of covariates, additional data analysis recommendations for future studies include: controlling baseline levels of the follow-up dependent variable, which has not been done consistently; discussing effect size estimates in terms of clinical as well as statistical significance; and greater modelling of disorder severity. On this last point, there is evidence that obesity–depression associations may be non-linear and strongest among individuals with a BMI $\geq$ 40 (80).

In summary, this review found good evidence that obesity is prospectively associated with increased depression, or onset of the disorder. In total, 80% of studies found evidence for significant obesity-to-depression associations, whereas only 53% found evidence for depression-to-obesity pathways. At the same time, there was enormous methodological heterogeneity among studies, especially with respect to the selection and statistical modelling of covariates, which hindered quantitative synthesis of studies. Few studies used measured weights and heights jointly with interview assessments of depression. Higher quality research must be conducted to elucidate the causal relationships between depression and obesity, the subgroups among whom they exist, and mediating pathways.

Conflict of Interest Statement

No conflict of interest was declared.

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