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Depressive symptoms are associated with obesity in adults with heart failure: An analysis of gender differences

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Abstract

Background—Depression is a predictor and consequence of obesity in the general population. Up to 50% of patients with heart failure exhibit elevated depressive symptoms or depressive disorders; however, research on the depression–obesity relationship in heart failure populations is limited, especially in regard to gender differences.

Aims—To conduct total-sample and gender-stratified analyses to determine whether depressive symptoms are associated with body mass index (BMI) in a sample of patients with heart failure.

Method—Participants were 348 (39% female, 26% non-White) patients with heart failure (aged 68.7 ± 9.7 years) recruited from urban medical centers. Depressive symptoms were measured using the Patient Health Questionnaire-9 (PHQ-9). Height and weight were used to compute BMI (kg/m^2). Regressions were performed for total sample and both genders. Regressions for BMI were run with demographic, medical, and psychological covariates in Step 1 and the PHQ-9 in Step 2.

Results—Regression results (total sample) revealed that the PHQ-9 was associated with BMI after adjusting for covariates ($\beta = .22$, $p = .004$). For males, the relationship between PHQ-9 and BMI remained ($\beta = .23$, $p = .024$) and was driven by those with severe obesity ($\text{BMI} \geq 40 \text{ kg}/\text{m}^2$). A trend between PHQ-9 and BMI was detected among females ($\beta = .19$, $p = .091$).

Conclusion—BMI is related to depressive symptoms in adults with heart failure even after adjusting for demographic and medical covariates. Depressive symptoms were associated with BMI in males, whereas a trend was detected among females. These findings could ultimately be used to improve heart failure outcomes for depressed, obese individuals with heart failure.

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Conflict of interest

None declared.

Keywords

Body mass index; depressive symptoms; obesity; heart failure; gender

Introduction

Obesity rates across the globe have more than doubled over the past three decades,^{1–3} and obesity has been named a global epidemic by the World Health Organization (WHO).⁴ Obesity rates reported by the WHO European Region are 20% for men and 23% for women.⁵ In the United States, prevalence rates are higher – not only in the general adult population (36%) but also in patient populations, particularly those with heart failure (HF). Indeed, an estimated 40% patients with HF are defined as obese (body mass index (BMI) ≥ 30 kg/m²),⁶ and obese individuals are two times as likely as their normal weight peers to have HF.⁷

Importantly, prospective studies indicate that depression is both a risk factor for and a potential consequence of obesity in the general US population as well as European samples.^{8,9} Depression–obesity associations are likely observed internationally given that body dissatisfaction and desire for thinness also have been reported across the globe.¹⁰ For example, despite relatively low obesity rates (10%) in Sweden,¹¹ depression was observed more often in Swedish obese individuals than in their non-obese peers.¹² These findings might be highly relevant to the HF population, given that 20–50% of individuals with HF^{13,14} present with elevated depressive symptoms or major depressive disorder.^{15–18} In addition, depression is associated with adverse HF outcomes, including increased hospital readmission rates and decreased survival.^{15,18,19} Obesity, on the other hand, has been associated with lower risk for adverse HF outcomes in HF, a phenomenon labeled the “obesity paradox.”²⁰ However, some authors question the validity of this paradox²¹ and speculate that it is an analytic artifact resulting from the failure to include severely obese individuals in statistical analyses and the inadequacy of BMI as a measure of adiposity in older adults and/or patients with HF.^{22,23}

Taken together, the aforementioned evidence suggests that the depression–obesity relationship may have important implications on HF outcomes; however, research on this relationship in HF populations is limited. One study suggests that obese patients with HF reported higher depressive symptoms than non-obese patients;²⁴ however, this study did not examine gender differences in the association. Such information is important, given that the depression–obesity relationship may be moderated by gender in non-HF samples. For instance, some studies support stronger relationships between depression and obesity among White women compared with non-Whites,^{25–28} whereas others have found stronger associations for men.²⁹ Another group of studies has found no gender differences.^{9,30} These conflicting findings in non-HF samples combined with the documented gender differences in prognosis among patients with HF (i.e. males having poorer prognosis than females^{20,31}) suggest that an analysis of gender differences in the depression–obesity relationship in HF is warranted. Such information will not only elucidate potential gender differences in the relationship between depressive symptoms and obesity in a HF population but may also

provide a reason for prognostic differences between the genders and/or help to identify subgroups of patients with HF that are at risk for poorer outcomes.

Thus, to better understand the complex relationships between gender, depression, and obesity in HF, we conducted total-sample and gender-stratified analyses to determine whether depressive symptoms were associated with BMI in a sample of patients with HF. Based on previous findings, we hypothesized that females would have greater depressive symptoms than males;^{32,33} however, due to the lack of previous evidence, we made no a priori hypothesis regarding gender differences in the relationship between depressive symptoms and BMI.

Methods

Participants

The convenience sample for this cross-sectional study was 353 adults with HF enrolled in the larger, ongoing Heart Failure Adherence, Behavior, and Cognition Study (Heart ABC).^{34,35} Participants who had complete baseline data on the depressive symptoms measure and BMI were selected. Study eligibility requirements were as follows: (1) aged 50–85 years at enrollment, (2) physician-documented systolic HF diagnosis (ejection fraction < 40%) within 36 months of study enrollment, (3) New York Heart Association (NYHA) class II or III < 3 months' duration at the time of study enrollment, (4) no cardiac surgery within last three months, (5) no history of neurological disorder or injury (e.g. Alzheimer's disease, dementia, stroke, seizures), (6) no history of moderate or severe head injury, (7) no past or current history of psychotic disorders, bipolar disorder, learning disorder, developmental disability, renal failure requiring dialysis, or untreated sleep apnea, (8) no current substance abuse or within the past five years, and (9) no current use of home telehealth monitoring program for HF. For the current study, five underweight participants (BMI < 18.5 kg/m²) were excluded from the analyses given the documented u-shaped relationship between depressive symptoms and BMI.³⁶ Thus, the final sample used for analyses was $N = 348$.

Measures

Depressive symptom severity—Depressive symptom severity was assessed using the Patient Health Questionnaire-9 (PHQ-9).³⁷ The PHQ-9 consists of nine items assessing the following symptoms of depression: (1) anhedonia, (2) depressed mood, (3) sleep difficulties, (4) fatigue, (5) appetite changes, (6) poor self-esteem, (7) concentration problems, (8) psychomotor retardation/agitation, and (9) suicidal ideation. Items are rated 0=*not at all*, 1=*several days*, 2=*more than half the days*, and 3=*nearly every day*. The total sum of the items was used as a continuous score of depressive symptom severity, with higher scores signifying more severe symptom levels. A score of 5, 10, 15, or 20 represents mild, moderate, moderately severe, and severe depression levels respectively.³⁷ The PHQ-9 has demonstrated good reliability and validity.³⁷

Body mass index—Participants' BMI was calculated as kg/m² using weight and height. For the Heart ABC study, all patients were given an electronic scale to use for home weighing. For the purpose of the current study, baseline weights were obtained within four

weeks of enrollment from the electronic scale provided by the investigators. Patients' most recent heights were self-reported after enrollment or obtained from the medical record. We examined continuous BMI as well as the following weight categories: Underweight (excluded from regression analyses), Normal Weight, Overweight, and Obese Classes I, II, and III.

Covariates—The following variables were included as covariates: age (measured continuously), race (0 = White, 1 = non-White), education (1 = no schooling, 2 = eighth grade or less, 3 = ninth to 11th grade, 4 = high school, 5 = technical or trade school, 6 = some college, 7 = Bachelor's degree, 8 = Master's degree), socioeconomic status (SES), Charlson Comorbidity Index score,³⁸ baseline self-reported HF severity (as estimated by the four levels of the NYHA³⁹), and anxiety (PROMIS-Anxiety subscale⁴⁰). SES was estimated using subjects' zip code via a method similar to the one described by Roux et al.⁴¹ Z-scores were calculated for the SES score using indicators of income and education for each zip code. Higher scores indicate higher SES. The Charlson Comorbidity Index is a summary score of several medical conditions including diabetes, peripheral vascular disease, and myocardial infarction.³⁸ Medical diagnoses are assigned points, with more severe conditions receiving higher points; higher Charlson scores indicate a higher number and greater severity of medical comorbidities. Baseline self-reported heart failure severity was assessed by asking participants about their current symptoms/limitations (e.g. *Do you markedly reduce physical activity due to tiredness, heart fluttering, shortness of breath, anginal pain?*) Based on their self-reported symptoms, we assigned them to the corresponding NYHA class, ranging from Class I (Mild) and Class II – (Mild) to Class III (Moderate) and Class IV (Severe).³⁹ Thus, some patients were categorized as Class I or IV despite the initial inclusion criteria of physician-documented NYHA C lass II or III.

Procedure

The present analyses are from the parent Heart ABC Study, a large observational investigation of a representative sample of adults with HF.^{34,35} Participants were recruited from two separate health systems in northeast Ohio (Summa Health System, Inc. and University Hospitals), which allowed us to recruit patients from diverse geographic, socioeconomic, and race–ethnic backgrounds. Patients with documented diagnoses of systolic HF were recruited from inpatient cardiac units and outpatient practices. All patients gave their written, informed consent to participate in the study. The Institutional Review Boards of Kent State University, Summa Health Systems, Inc., and Case Western Research University approved all study procedures, and this investigation conforms to the principles outlined in the Declaration of Helsinki. Within four weeks of enrollment and written consent, a research assistant conducted the series of baseline self-report questionnaires including the PHQ-9 and provided the electronic scale to participants in order to obtain baseline BMI.

Data analyses

Independent *t*-tests for continuous variables and chi-square analyses for categorical variables were used to assess differences between male and female patients on the study variables. To examine the associations between depressive symptoms and BMI, sets of multiple linear

regression analyses were performed for total sample as well as males and females separately. Each primary analysis was conducted with continuous BMI as the dependent variable. The association between depressive symptoms and BMI was examined by entering age, race–ethnicity, education level, SES, medical comorbidities, HF severity level, and anxiety in Step 1 and PHQ-9 in Step 2. Gender was also included as a covariate in Step 1 for analyses using the total sample. If depressive symptoms were related to continuous BMI in a gender-stratified regression model, an analysis of covariance (ANCOVA) was run to compare depressive symptoms levels across the BMI categories for that gender, adjusting for the same covariates as the regression models. All analyses were conducted using IBM® SPSS® version 20.0 statistical software (IBM Corporation). p -values $< .05$ were considered statistically significant, whereas p -values between .05 and .10 were considered trends.

Results

Gender differences in demographic and medical factors

As is seen in Table 1, approximately 75% of the sample was either overweight (28.2% of the total sample) or obese (47.7% of the total sample). No gender differences were observed in the percentage of individuals in each BMI category in chi-square (χ^2) analyses, $\chi^2(4, N = 348) = 8.24, p = .083$. Thus, males and females were equally likely to be categorized as normal weight, overweight, or obese. Similarly, no gender differences were observed for continuous BMI ($p = .210$). With regard to depressive symptom severity, the sample on average endorsed depressive symptom levels in the subclinical range (PHQ-9 score < 10).³⁷ Compared with males, who endorsed minimal depression (PHQ-9 score < 5), females had higher PHQ-9 scores ($t(346) = 2.74, p = .007$) that were indicative of mild depressive symptoms (PHQ-9 score 5–9) in independent t -tests.

With regard to covariates, females also had significantly lower SES ($t(339) = 4.22, p < .001$) and education ($\chi^2(6, N = 348) = 28.60, p < .001$) and were more likely to be non-White ($\chi^2(1, N = 348) = 21.84, p < .001$) compared with males. Females were also less likely than males to be in NYHA Class I ($\chi^2(3, N = 348) = 8.23, p = .041$) but were equally likely to be in Classes II, III, and IV. Females had higher PROMIS-Anxiety scores ($t(345) = 2.47, p = .014$). No gender differences were observed for age ($p = .079$) or the Charlson Comorbidity Index ($p = .703$).

Association between depressive symptoms and BMI

Total sample—Regression results for the total sample revealed that higher PHQ-9 was associated with greater BMI after controlling for the covariates, $\beta = .22, p = .004$ (see Table 2). The PHQ-9 accounted for 2% of the variance in BMI above and beyond gender, age, race–ethnicity, education level, SES, medical comorbidities, HF severity level, and anxiety scores, with the total model accounting for 13% of the variance in BMI. Of the covariates, BMI was associated with age ($\beta = -.27, p < .001$) and anxiety ($\beta = -.20, p = .006$) but not with gender, race–ethnicity, education level, SES, Charlson scores, or HF severity levels on Step 2 (all $ps > .18$).

Males—For males, regressions indicated that the relationship between PHQ-9 and BMI remained, $\beta = .23$, $p = .024$ (see Table 2), and the PHQ-9 accounted for 2% of the variance in BMI. With PHQ-9 in the model, age ($\beta = -.18$, $p = .016$) and anxiety ($\beta = -.24$, $p = .013$) were also associated with BMI for males, and no other covariates were related to BMI (all p s $> .203$). Together, the model accounted for 9% of the variance in BMI.

Given that the PHQ-9 was associated with BMI in males, an ANCOVA was conducted to determine whether average depressive symptom levels differed across BMI categories (Figure 1). The ANCOVA omnibus test adjusted for the same covariates as the regression models and was significant, $F(4, 192) = 2.55$, $p = .041$, $\eta^2 = .05$. Pairwise comparisons indicated that for males with BMI ≥ 40 kg/m² (Obese Class III) average depressive symptom scores ($M = 6.9$, $SD = 5.6$) were significantly higher than all other BMI group except BMI Obese Class II (M s = 3.6–4.8, SD s = 3.8–5.1) (p s $> .088$). On average, these patients in Obese Class III had depressive symptom scores that were more than 50% greater than their normal weight peers' scores. The depressive symptom scores of the other BMI categories did not differ from one another (p s $> .142$).

Females—A trend between higher PHQ-9 and greater BMI was detected among females, $\beta = .19$, $p = .091$, and accounted for 2% of the variance (see Table 2). Age was also associated with BMI ($\beta = -.39$, $p < .001$), but no other covariates were related to BMI (p s $> .24$). Taken together, all the covariates and PHQ-9 accounted for 19% of the variance in BMI among females.

Given the trend between PHQ-9 and BMI, an ANCOVA was conducted across the BMI categories for females (Figure 1). The omnibus test was not significant, $F(4, 124) = 1.16$, $p = .333$, $\eta^2 = .04$; thus, we do not report the pairwise comparisons. However, we still present the average depressive symptom levels across BMI categories for females in Figure 1 for descriptive purposes.

Multicollinearity

Importantly, given the strong correlation between the PROMIS-Anxiety and the PHQ-9 scores ($r(347) = .67$ for total sample; $r(210) = .67$ for males; and $r(137) = .66$), we checked the collinearity statistics for each regression model. The tolerance inflation factor (TIF) and variance inflation factor (VIF) scores were all $< .47$ and < 2.11 , respectively, indicating that multicollinearity was not a problematic issue, as defined by a TIF $> .10$ and a VIF > 10 .⁴²

Discussion

The objective of the current study was to examine whether depressive symptoms were associated with obesity in a sample of patients with HF. We found that depression symptom scores were related to BMI in the total sample – after adjusting for demographic and medical factors as well as for anxiety. Our hypothesis that females would have greater depressive symptom severity than males was supported, as females scored 32% higher on the PHQ-9 than males. When we conducted gender-stratified analyses, we found that higher PHQ-9 scores were related to higher BMI for males, and that this effect was driven by those with severe obesity (BMIs ≥ 40 kg/m²). Males with severe obesity (Class III) had significantly

higher average depressive symptom scores. In contrast, only a statistical trend was detected between greater depression symptoms and higher BMI for females ($p = .091$). No significant differences in average depressive symptom scores by BMI class existed for females.

Our finding that obese patients with HF experience greater depressive symptom severity than their lower weight peers is supported by previous literature examining the depression–obesity relationship in persons with HF.²⁴ Furthermore, our results also support the finding that females endorse greater self-reported depressive symptom severity than males,^{32,33} although it should be noted that the overall mean depressive symptom level for the total sample was in the subclinical range (< 10). Our sample's rates of obesity were also similar to previous estimates of obesity within HF samples.⁶ In the present sample, 28% of participants were overweight and 48% were obese, with males and females equally likely to be in these categories. However, numerous covariates (i.e. SES, education, race, HF severity, anxiety, depression) differed among males and females in the current sample. For instance, females had significantly lower SES and education levels and were more likely to be non-White. Females were less likely to be in NYHA Class I and endorsed greater anxiety and depression severity levels.

The current study extends the previous literature by examining gender differences in the relationship between depression and obesity in HF, an analysis which has not been done previously. Specifically, we found that depressive symptoms are associated with BMI for males, an effect driven by those with severe obesity. Thus, obesity severity level may moderate the depression–obesity relationship among males, with higher classes endorsing the most severe depression levels. In contrast, the depression–obesity relationship failed to reach significance among females, and several factors may explain these findings. First, the relationship between depression and obesity has been shown to be stronger among women with higher SES than among women with lower SES.^{43,44} In the present sample, women's SES was approximately two standard deviations lower than men's and one standard deviation below the sample mean. Similarly, women in our sample were more likely to be non-White. One study of racial–ethnic differences in weight gain in a community sample over 34 years found that the differences in weight gain between African American and Caucasian women can largely be attributed to differences in socioeconomic conditions and not depression.⁴⁵ Thus, the lower SES and greater percentage of non-Whites among the females in our sample may have contributed to the weaker relationship between BMI and depression observed for women. Second, women's depressive symptoms may have reflected disease status rather than genuine depression, as participants with the worst HF disease severity also had the worst depression severity. Specifically, individuals in NYHA Class IV had significantly higher depression levels than the other classes, and adjusting for disease severity and SES weakened the relationship between depression and BMI in our sample.

The present findings have numerous clinical and research implications. First, these findings are especially important because depression is associated with negative outcomes in patients with HF, and our results suggest that obesity is associated with more severe depressive symptoms. Accordingly, obese persons with HF may be a subgroup at greater risk for poor outcomes. Second, healthcare providers should provide screening for and monitoring of depressive symptoms in HF outpatients and inpatients, especially if their patients present

with obesity. In our sample, individuals in higher BMI classes (e.g. Class III) may experience the greatest emotional distress and may especially benefit from early depression screening and intervention. As recommended in previous studies, even the presence of mild depressive symptoms (e.g. PHQ score of 5–9) without a full diagnosis of major depression warrants continued monitoring and possibly intervention.³⁷ Third, researchers should pursue investigations that clarify how obesity may influence depression treatment or vice versa in persons with HF as well as how these factors act together to impact HF self-management. Finally, the possibility of a third factor which exacerbates both obesity and depression in HF should be explored. For example, there may be a predisposing genotype or gene by environment interaction that contributes to the relationship of depression and obesity. Hypothalamic–pituitary–adrenal axis dysregulation resulting in excessive cortisol secretion could also contribute to the relationship.⁴⁶ It is unlikely that one factor singlehandedly causes this observed relationship, but physiological, genomic, or behavioral processes may represent strong pathways that meaningfully interact to link obesity and depression.

The current study has several limitations that should be noted. First, the current cross-sectional data does not allow us to determine the directionality of the relationship between depression and obesity in patients with HF. Given that depression and obesity have shown bidirectional relationships,⁹ future studies should aim to determine whether excess adiposity is a predictor or consequence of depression in HF in males and females. Additionally, prospective research is also needed in order to clarify the validity of the “obesity paradox”²⁰ and how obesity and depression act together to influence HF outcomes such as hospitalizations, healthcare use, and mortality. Considering that males tend to have poorer prognoses than females,^{20,31} these longitudinal investigations should certainly consider the role of gender in the depression–obesity relationship. Next, we examined depression and anxiety but did not assess other forms of psychopathology; thus, future research should further examine whether BMI is associated with other disorders, such as binge eating disorder, which has been linked with both depression and obesity.⁴⁶ Additionally, certain characteristics of the present sample may limit generalizability. For instance, the present sample comprised US patients with reduced ejection fraction who were predominantly White with Class III HF severity levels and at least a high school education level. Future studies in more diverse samples are needed to confirm whether our results replicate in patients who live outside the USA, have preserved ejection fraction, are non-White, have more or less severe HF severity levels, and/or are have lower education levels. Lastly, our measure of obesity (i.e. BMI) does not account for other common causes of excess weight in patients with HF, including water retention.⁴⁷ Unfortunately, our study did not have physiological assessment of patients’ total body water so we could not precisely control for water retention. Future studies should utilize BMI alongside other physiological measures of adiposity and total body water (e.g. body composition analyses) in order to support more sophisticated conclusions about the relationship of true adiposity and depression in adults with HF.

In brief conclusion, BMI is related to depressive symptoms in adults with HF even after controlling for numerous demographic and medical covariates. The current paper extends the literature by also examining gender differences in the depression–obesity relationship. Depression was associated with BMI in males and the effect was driven by persons with

severe obesity. In contrast, a trend between depressive symptoms and BMI was detected among females and may be explained by notable gender differences across a variety of covariates, such as SES and race–ethnicity. Considering the documented deleterious effects of depression on HF outcomes,^{15,18,19} the ability to identify factors associated with depressive symptoms has implications for clinical practice (e.g. early intervention) and research (e.g. implications for health outcomes). Longitudinal examination is needed to determine the association of depressive symptoms and obesity with HF outcomes, such as rehospitalization and mortality. Such findings could ultimately be used to improve quality of life, decrease hospitalization, and prolong lifespans for depressed and/or obese individuals living with HF.

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References

1. Flegal KM, Carroll MD, Kuczmarski RJ, et al. Overweight and obesity in the United States: Prevalence and trends, 1960–1994. *Int J Obesity Relat Metab Disord*. 1998; 22:39–47.
2. Flegal KM, Carroll MD, Kit BK, et al. Prevalence of obesity and trends in the distribution of body mass index among US adults, 1999–2010. *JAMA*. 2012; 307:491–497. [PubMed: 22253363]
3. World Health Organization. [accessed 17 September 2013] Obesity. Situation and trends. Global Health Observatory (GHO). 2013. http://www.who.int/gho/ncd/risk_factors/obesity_text/en/index.html
4. World Health Organization. World Health Organization Technical Report Series. Vol. 894. Geneva: World Health Organization; 2000. Obesity: Preventing and managing the global epidemic. Report of a WHO consultation; p. 1-253.
5. World Health Organization. [accessed 9 May 2014] Obesity. Data and statistics. Health Topics. 2014. <http://www.euro.who.int/en/health-topics/noncommunicable-diseases/obesity/data-and-statistics>
6. Kapoor JR, Heidenreich PA. Obesity and survival in patients with heart failure and preserved systolic function: A U-shaped relationship. *Am Heart J*. 2010; 159:75–80. [PubMed: 20102870]
7. Kenchiah S, Evans JC, Levy D, et al. Obesity and the risk of heart failure. *N Engl J Med*. 2002; 347:305–313. [PubMed: 12151467]
8. Blaine B. Does depression cause obesity? *J Health Psychol*. 2008; 13:1190–1197. [PubMed: 18987092]
9. Luppino F, de Wit L, Bouvy P, et al. Overweight, obesity, and depression: A systematic review and meta-analysis of longitudinal studies. *Arch Gen Psychiatry*. 2010; 67:220–229. [PubMed: 20194822]
10. Swami V, Frederick DA, Aavik T, et al. The attractive female body weight and female body dissatisfaction in 26 countries across 10 world regions: Results of the International Body Project I. *Pers Soc Psychol Bull*. 2010; 36:309–325. [PubMed: 20179313]
11. Organisation for Economic Co-operation and Development. [accessed 9 May 2014] Obesity and the economics of prevention – fit not fat Sweden key facts. Health Policies and Data. 2008. <http://www.oecd.org/health/health-systems/obesityandtheeconomicsofprevention-fitnotfat-swedenkeyfacts.htm>

12. Sullivan M, Karlsson J, Sjöström L, et al. Swedish obese subjects (SOS) – an intervention study of obesity. Baseline evaluation of health and psychosocial functioning in the first 1743 subjects examined. *Int J Obes Relat Metab Disord.* 1993; 17:503–512. [PubMed: 8220652]
13. Trojano L, Incalzi RA, Acanfora D, et al. Cognitive impairment: A key feature of congestive heart failure in the elderly. *J Neurol.* 2003; 250:1456–1463. [PubMed: 14673579]
14. Haworth J, Moniz-Cook E, Clark A, et al. Prevalence and predictors of anxiety and depression in a sample of chronic heart failure patients with left ventricular systolic dysfunction. *Eur J Heart Fail.* 2005; 7:803–808. [PubMed: 16054436]
15. Jiang W, Alexander J, Christopher E, et al. Relationship of depression to increased risk of mortality and rehospitalization in patients with congestive heart failure. *Arch Intern Med.* 2001; 161:1849–1856. [PubMed: 11493126]
16. Koenig HG. Depression in hospitalized older patients with congestive heart failure. *Gen Hosp Psychiatry.* 1998; 20:29–43. [PubMed: 9506252]
17. Freedland KE, Rich MW, Skala JA, et al. Prevalence of depression in hospitalized patients with congestive heart failure. *Psychosom Med.* 2003; 65:119–128. [PubMed: 12554823]
18. Rutledge T, Reis VA, Linke SE, et al. Depression in heart failure: A meta-analytic review of prevalence, intervention effects, and associations with clinical outcomes. *J Am Coll Cardiol.* 2006; 48:1527–1537. [PubMed: 17045884]
19. Adams J, Kuchibhatla M, Christopher EJ, et al. Association of depression and survival in patients with chronic heart failure over 12 years. *Psychosomatics.* 2012; 53:339–346. [PubMed: 22281436]
20. Curtis JP, Selter JG, Wang Y, et al. The obesity paradox: Body mass index and outcomes in patients with heart failure. *Arch Intern Med.* 2005; 165:55. [PubMed: 15642875]
21. Habbu A, Lakkis NM, Dokainish H. The obesity paradox: Fact or fiction? *Am J Cardiol.* 2006; 98:944–948. [PubMed: 16996880]
22. Stevens J, Cai J, Pamuk ER, et al. The effect of age on the association between body-mass index and mortality. *N Engl J Med.* 1998; 338:1–7. [PubMed: 9414324]
23. Fitzpatrick AL, Kuller LH, Lopez OL, et al. Midlife and late-life obesity and the risk of dementia: Cardiovascular health study. *Arch Neurol.* 2009; 66:336. [PubMed: 19273752]
24. Evangelista LS, Moser DK, Westlake C, et al. Impact of obesity on quality of life and depression in patients with heart failure. *Eur J Heart Fail.* 2006; 8:750–755. [PubMed: 16530474]
25. Gavin A, Rue T, Takeuchi D. Racial/ethnic differences in the association between obesity and major depressive disorder: Findings from the Comprehensive Psychiatric Epidemiology Surveys. *Public Health Rep.* 2010; 125:698–708. [PubMed: 20873286]
26. Anderson S, Murray D, Johnson C, et al. Obesity and depressed mood associations differ by race/ethnicity in adolescent girls. *Int J Pediatr Obes.* 2011; 6:69–78. [PubMed: 20367561]
27. Vogelzangs N, Kritchevsky SB, Beekman AT, et al. Depressive symptoms and change in abdominal obesity in older persons. *Arch Gen Psychiatry.* 2008; 65:1386. [PubMed: 19047525]
28. Vogelzangs N, Kritchevsky SB, Beekman AT, et al. Obesity and onset of significant depressive symptoms: Results from a community-based cohort of older men and women. *J Clin Psychiatry.* 2010; 71:391. [PubMed: 20021992]
29. Mustillo S, Worthman C, Erkanli A, et al. Obesity and psychiatric disorder: Developmental trajectories. *Pediatrics.* 2003; 111:851–859. [PubMed: 12671123]
30. Roberts RE, Deleger S, Strawbridge WJ, et al. Prospective association between obesity and depression: Evidence from the Alameda County Study. *Int J Obes.* 2003; 27:514–521.
31. Simon T, Mary-Krause M, Funck-Brentano C, et al. Sex differences in the prognosis of congestive heart failure: Results from the Cardiac Insufficiency Bisoprolol Study (CIBIS II). *Circulation.* 2001; 103:375–380. [PubMed: 11157688]
32. Parissis JT, Mantziari L, Kaldoglou N, et al. Gender-related differences in patients with acute heart failure: Management and predictors of in-hospital mortality. *Int J Cardiol.* 2012; 20:185–189.
33. Kao C-W, Chen T-Y, Cheng S-M, et al. Gender differences in the predictors of depression among patients with heart failure. *Eur J Cardiovasc Nurs.* 2013; 13:320–328. [PubMed: 23814176]

34. Clinicaltrials.gov. [accessed 22 August 2013] Self-management and Cognitive Function in Adults with Heart Failure (Heart ABC) – Identifier: NCT01461629. 2011. <http://clinicaltrials.gov/ct2/show/NCT01461629?term=heart+abc&rank=1>
35. Hawkins MA, Gunstad J, Dolansky M, et al. Greater body mass index is associated with poorer cognitive functioning in male heart failure patients. *J Card Fail.* 2014; 20:199–206. [PubMed: 24361776]
36. De Wit LM, van Straten A, van Herten M, et al. Depression and body mass index, a u-shaped association. *BMC Public Health.* 2009; 9:14. [PubMed: 19144098]
37. Kroenke K, Spitzer RL. The PHQ-9: A new depression diagnostic and severity measure. *Psychiatr Ann.* 2002; 32:1–7.
38. Charlson ME, Pompei P, Ales KL, et al. A new method of classifying prognostic comorbidity in longitudinal studies: Development and validation. *J Chronic Dis.* 1987; 40:373–383. [PubMed: 3558716]
39. Bennett JA, Riegel B, Bittner V, et al. Validity and reliability of the NYHA classes for measuring research outcomes in patients with cardiac disease. *Heart Lung.* 2002; 31:262–270. [PubMed: 12122390]
40. Pilkonis PA, Choi SW, Reise SP, et al. Item banks for measuring emotional distress from the Patient-Reported Outcomes Measurement Information System (PROMIS®): Depression, anxiety, and anger. *Assessment.* 2011; 18:263–283. [PubMed: 21697139]
41. Roux AVD, Merkin SS, Arnett D, et al. Neighborhood of residence and incidence of coronary heart disease. *N Engl J Med.* 2001; 345:99–106. [PubMed: 11450679]
42. Kline, RB. Principles and practice of structural equation modeling. New York, NY: Guilford Press; 2011.
43. Ross CE. Overweight and depression. *J Health Soc Behav.* 1994; 35:63–79. [PubMed: 8014430]
44. Stunkard AJ, Faith MS, Allison KC. Depression and obesity. *Biol Psychiatry.* 2003; 54:330–337. [PubMed: 12893108]
45. Baltrus PT, Lynch JW, Everson-Rose S, et al. Race/ethnicity, life-course socioeconomic position, and body weight trajectories over 34 years: The Alameda County Study. *J Inform.* 2005; 95:1595–1601.
46. Faith MS, Matz PE, Jorge MA. Obesity–depression associations in the population. *J Psychosom Res.* 2002; 53:935–942. [PubMed: 12377306]
47. Bell NH, Schedl HP, Bartter FC. An explanation for abnormal water retention and hypoosmolality in congestive heart failure. *Am J Med.* 1964; 36:351–360. [PubMed: 14131879]

Implications for practice

- Greater depression is linked to higher BMI in heart failure.
- The depression–BMI link is stronger in men than in women.
- Depression and obesity may impact heart failure self-management.
- Depressed, obese persons with heart failure may have poorer heart failure outcomes.

Figure 1.

Average depressive symptom severity levels across weight categories.

Covariates appearing in the model were: age, race–ethnicity, education level, socioeconomic status, Charlson score, New York Heart Association class, and PROMIS-Anxiety total score. Error bars represent standard errors.

Normal Weight = body mass index (BMI) 18.5–24.9 kg/m²; Overweight = BMI 25.0–29.9 kg/m²; Obese Class I = BMI 30.0–34.9 kg/m²; Obese Class II = BMI 35.0–39.9 kg/m²; Obese Class III = BMI ≥ 40 kg/m².

Table 1

Characteristics of participants.

	Total sample (max. N=378)	Males (max. N=211)	Females (max. N=137)
Demographic factors			
Age	68.7 ± 9.7	69.4 ± 9.3	67.5 ± 10.1
Female	137 (39.4)	–	–
Non-White	92 (26.4)	37 (17.5)	55 (40.1) ^a
Education level			
Eighth grade or less	10 (2.9)	6 (2.8)	4 (2.9)
9–11th grade	35 (10.1)	12 (5.7)	23 (16.8) ^a
High school	95 (27.0)	50 (23.7)	44 (32.1)
Technical or trade school	38 (10.6)	21 (10.0)	16 (11.7)
Some college	96 (27.0)	58 (27.5)	36 (26.3)
Bachelor's degree	44 (12.6)	34 (16.1)	10 (7.0) ^a
Master's degree	35 (9.8)	30 (14.2)	4 (2.9) ^a
SES Z-score	−0.05 (4.4)	0.74 (4.3)	−1.3 (4.2) ^a
Medical factors			
Charlson score	3.3 ± 1.8	3.3 ± 1.8	3.4 ± 1.6
Taking diuretic	245 (70.6)	140 (66.7)	105 (76.6) ^a
Self-reported HF severity			
NYHA Class I	35 (10.1)	28 (13.3)	7 (4.9) ^a
NYHA Class II	80 (22.4)	49 (23.2)	29 (21.2)
NYHA Class III	219 (62.4)	126 (59.7)	91 (66.4)
NYHA Class IV	19 (5.2)	8 (3.8)	10 (7.3)
BMI category			
Normal Weight (BMI 18.5–24.9 kg/m ²)	84 (24.1)	48 (22.7)	36 (26.3)
Overweight (BMI 25.0–29.9 kg/m ²)	98 (28.2)	67 (31.8)	31 (22.6)
Obese Class I (BMI 30–34.9 kg/m ²)	92 (26.4)	60 (28.4)	32 (23.4)
Obese Class II (BMI 35–39.9 kg/m ²)	46 (13.2)	23 (10.9)	23 (16.8)
Obese Class III (BMI 40.0 kg/m ²)	28 (8.0)	13 (6.2)	15 (10.9)
BMI (kg/m ²)	30.4 ± 6.7	30.0 ± 6.2	31.0 ± 7.4
Psychological factors			
PROMIS	13.0 ± 5.3	12.4 ± 5.0	13.8 ± 5.7 ^a
PHQ-9	4.7 ± 4.9	4.1 ± 4.8	5.6 ± 5.1 ^a

Continuous variables represented with mean ± SD. Categorical variables represented with N(%).

^a $p < .05$ for independent *t*-test or chi-square test, indicating differences between males and females.

SES: socioeconomic status; NYHA: New York Health Association; HF: heart failure; BMI: body mass index; PROMIS: Patient Reported Outcomes Measurement Information System; PHQ-9: Patient Health Questionnaire-9.

Table 2

Regressions of PHQ-9 depression scale predicting body mass index.

	Total sample (N=339) ^a				Males (N=204)				Females (N=135)			
	β	R ²	F		β	R ²	F		β	R ²	F	
Step 1	–	.10	–	4.75 [*]	–	.06	–	1.93	–	.17	–	3.73 [*]
Gender	.03				–				–			
Age	–.27 [*]				–.18 [*]				–.40 [*]			
Race	.02				.02				–.00			
Education	–.05				–.05				–.05			
SES	.01				–.03				.06			
Charlson	–.02				–.07				.05			
NYHA	.13 [*]				.15 [*]				.09			
PROMIS	–.06				–.10				–.01			
Step 2	–	.13	.02	8.38 [*]	–	.09	.02	5.19 [*]	–	.19	.02	2.90
PHQ-9	.22 [*]				.23 [*]				.19			

^aUnderweight participants ($n = 5$) were excluded from analyses.

^{*} $p < .05$.

SES; socioeconomic status; NYHA; New York Health Association; PROMIS; Patient Reported Outcomes Measurement Information System; PHQ-9; Patient Health Questionnaire-9.