Weight and Depressive Symptoms in Older Adults: Direction of Influence?

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Objectives. The purpose of this study was to clarify the direction of the relationship between changes in depressive symptoms and changes in weight in older adults.

Methods. The sample included a prospective cohort of individuals aged 53–63 (n = 9,130) enrolled in the Health and Retirement Study. We used separate cross-lagged models for men and women in order to study the impact of weight change on subsequent increases in depressive symptoms 2 years later and vice versa.

Results. Weight gain did not lead to increased depressive symptoms, and weight loss preceded increased depressive symptoms only in unadjusted models among men (odds ratio [OR] = 1.26, 95% confidence interval [CI] = 1.04–1.53). Increased depressive symptoms were not predictive of subsequent weight loss, but they were predictive of subsequent weight gain in unadjusted models only (men: OR = 1.24, 95% CI = 1.00–1.54; women: OR = 1.12, 95% CI = 1.00–1.26). In adjusted models, baseline depressive symptoms predicted both weight loss and weight gain among both men and women. Increase in functional limitations and medical conditions were significant predictors of both weight loss and weight gain. Baseline functional limitations also predicted increased depressive symptoms.

Discussion. Based on our findings, it is apparent that researchers need to examine the pathways between changes in weight and increases in depressive symptoms in the context of functional limitations and medical comorbidity.

Depression and weight problems are two of the most common and important problems in primary care, particularly among older adults. Depression is the most prevalent psychiatric disorder among elderly adults (Regier et al., 1988), and more than 70% of Americans aged 65–74 are either overweight or obese (U.S. Department of Health and Human Services, 2003). Weight problems and depression may cause moderate to severe impairments, and both conditions can be difficult to treat. Depression and weight problems (both being overweight or obese and being underweight) are also related to a large number of other physical and mental comorbidities, which impacts quality of life and also the likelihood of early mortality (Visscher et al., 2001).

Although weight and depression are associated with each other, the mechanism underlying the relationship is unknown. For example, both weight loss and weight gain are hallmark signs of depression. Weight changes, however, are not required for a depression diagnosis, nor are the mechanisms by which weight change and depression interact evident. The link between depression and weight problems may be due to one condition “causing” the other via psychological or biological mechanisms, or due to a third condition or consequence related to both weight problems and depression. For example, being overweight in a society that values thinness may lead to feelings of self-loathing and depression. Conversely, people with depression may use food to self-medicate low mood and/or may have increased appetite, leading to weight gain. Others who experience reduced appetite during depressive episodes may lose weight. Furthermore, some depression medications cause weight gain (Devlin, Yanovski, & Wilson, 2000; Zimmermann, Kraus, Himmerich, Schuld, & Pollmacher, 2003), and some drugs used to treat general medical conditions associated with obesity may bring on depressive symptoms (Brown & Stoudemire, 1998).

Although research has established cross-sectional associations between weight and depression (Friedman & Brownell, 1995; Johnston, Johnson, McLeod, & Johnston, 2004), these studies cannot make inferences about the direction of the relationship. A recent review by Faith, Calamaro, Dolan, and Pietrobelli (2004) found that research on the relationship between weight and depression is moving beyond cross-sectional studies into longitudinal designs in an effort to discern causality. However, these studies have yielded varying results, with some concluding that obese individuals have increased risk of depression (Roberts, Deleger, Strawbridge, & Kaplan, 2003) and others concluding the converse to be true, with baseline depression being predictive of obesity at follow-up (Hallstrom & Noppa, 1981; Raikkonen, Matthews, & Kuller, 2002).

None of the aforementioned studies have clarified whether the hallmark signs of depression, weight loss or gain, precede or follow the onset of depressive symptoms. Determining the temporal sequencing of weight changes and depression is critical to understanding the causal pathway and associated mechanisms. Furthermore, in older adults, the relationship between depression and weight may be different than the relationship found among younger adults, because functional
limitations and medical comorbidity associated with aging may cause weight change and may also be associated with changes in mood. The purpose of this analysis was to determine the independent effects of (a) changes in depressive symptoms on changes in weight and (b) changes in weight on changes in depressive symptoms in a cohort of older adults, after adjusting for changes in functional limitations and comorbid conditions, as well as baseline functional limitations, comorbid conditions, and demographic and behavioral factors.

**METHODS**

**Design of the Health and Retirement Study (HRS)**

The HRS is a nationally representative longitudinal study sponsored by the National Institute on Aging and conducted by the Institute for Social Research at the University of Michigan. Study organizers selected the HRS sample by using a multistage, area probability design of respondents in U.S. households. The study oversampled African Americans, Hispanics, and Florida residents. Researchers contacted each selected household by telephone and screened it for an age-eligible respondent who had been born between 1931 and 1941. Although the HRS includes data on the spouse or partner of age-eligible persons, the present analyses include data only for respondents born between 1931 and 1941. The HRS conducted in-home interviews in 7,702 households, representing an 82.0% response rate. This yielded 9,824 participants aged 51–61 years for initial face-to-face interviews in 1992. Researchers conducted follow-up telephone interviews every 2 years; interviews are available for 1994, 1996, 1998, 2000, and 2002.

**Measures and Variables Examined**

We extracted and recoded the following survey items:

**Depressive symptoms.**—Between 1994 and 2002, study organizers measured depressive symptomatology by using eight items from the Center for Epidemiologic Studies–Depression scale (CES-D). Rather than determining the presence or absence of recognized psychiatric disorders, this instrument measures a continuum of psychological distress (symptoms of depression and anxiety). Higher scores are indicative of more psychological distress. Readers may find a complete description of this scale elsewhere (Radloff, 1977). Cronbach’s alphas for the CES-D were greater than .80 in all years.

The response format to the 8-item CES-D was worded as “Would you say yes or no?”. We calculated the depressive symptom score (0–8) as the number of items endorsed at each time point (1994, 1996, 1998, 2000, 2002). We then calculated a change score between each two adjacent time points. We then categorized the resulting change score into being a 2-point or greater increase from the previous time point (representing an approximately 1 standard deviation increase in depressive symptoms) versus being less than a 2-point increase in depressive symptoms. The no increase in depressive symptoms group was the reference group for the analyses.

**Weight.**—The HRS collected self-reported data on weight and height at baseline and at each follow-up. We calculated body mass index (BMI) for each wave by dividing weight (kg) by height (m²) in order to standardize weights by height. We calculated changes in BMI for each wave between 1994 and 2002. We expressed weight changes as percentage change in BMI, which research has shown to be the most clinically relevant variable (Roubenoff & Kehayias, 1991). Because participants’ height did not change significantly during the study period, we use the term weight instead of BMI to ease interpretation, as changes in BMI over time could only be attributed to weight change and not change in height. We trichotomized the percent change in weight (BMI) between adjacent time points into having experienced at least a 5% decline in weight from the previous time point (weight loss), having experienced at least a 5% increase in weight from the previous time point (weight gain), and not having experienced a significant change in weight from the previous time point (no change; −5% < weight change < 5%). The no change group was the reference group in the analyses. We trichotomized weight change because both weight loss and weight gain are symptoms of clinical depression; therefore, it was important to test models where changes in depressive symptoms may affect weight loss and weight gain (and vice versa). In order to adjust for weight at the beginning of the time period, we categorized BMI into obese (BMI ≥ 30), overweight (25 ≤ BMI < 30), normal weight (19 < BMI < 25), and underweight (BMI ≤ 19). The normal weight group was the reference group in the analyses.

**Demographics.**—The HRS collected self-reported information on age, gender, race/ethnicity, marital status, and education level. Study organizers assessed age as a continuous variable. Researchers categorized race/ethnicity into White/Caucasian, Black/African American, Hispanic (non-Black), and Other. Marital status was married, divorced, widowed, or never married. The study grouped educational levels as 0–11 years, high school graduate, some college, college graduate, and post college.

**Health and behaviors.**—The HRS included questions about the number of self-reported chronic health problems as diagnosed by a doctor: hypertension, cancer, diabetes, pulmonary disease, heart disease, stroke, psychological problems, and arthritis. We used an additive scale of medical conditions (range 0–8), because other research supports it use (Ferraro & Wilmuth, 2000). Based on the distribution, we trichotomized this variable into 0 versus 1 versus 2 or more medical conditions. Other behavioral variables of interest that were evaluated included smoking history and alcohol use. We dichotomized cigarette smoking as current smoker versus current nonsmoker. We dichotomized alcohol use as heavy drinking (3 or more alcoholic drinks per day) versus no heavy drinking.

**Functional status.**—The HRS also included several yes/no measures of functional status, reflecting respondents’ functional limitations. We calculated a summed functional limitations score (range 0–8) by adding the responses to each of the eight yes/no items: difficulty sitting for 2 hr or more, stooping/kneeling/crouching, lifting or carrying an object weighing 10 pounds, picking up a dime from a table, lifting arms above shoulder level, walking several blocks, walking one block, and
climbing one flight of stairs. Based on the distribution, we dichotomized this variable into 0 versus 1 or more functional limitations.

**Health status changes.**—We calculated change in medical conditions and change in functional limitations scores between each adjacent time point. We included these variables in our models because we wanted to assess how the change in these constructs affected the two outcome variables of interest (change in weight and increase in depressive symptoms) given a recent study that found a dynamic interplay between disease, disability, and depression (Kelley-Moore & Ferraro, 2005). We dichotomized change in medical conditions into having experienced an increase of one or more medical conditions from the previous time point (vs no increase), and we dichotomized change in functional limitations into having experienced an increase of two or more functional limitations from the previous time point (versus less than a 2-point increase), based on the distributions of these variables.

**Data Analysis**

We conducted all analyses using the publicly available data files from the HRS (Institute for Social Research, 1995). We used a lagged covariate model to describe the relationship between change in weight and increase in depressive symptoms (Figure 1). We fit our models separately for men and women because previous research dating back to the Midtown Manhattan Study of the 1950s and 1960s (Moore, Stunkard, & Srole, 1962) suggested that the relationship between weight and depressive symptoms differs by gender. We confirmed that the relationship differed by gender in our data set, as well. We fit two models:

- **Change in weight model:**
  \[
  \log \frac{Pr(\Delta Wt_j = \text{increase})}{Pr(\Delta Wt_j = \text{no change})} = \mu_1 + \beta_1(\Delta Dep)_{ij-1} + \gamma_1'(\text{covariates})_{ij}
  \]
  and
  \[
  \log \frac{Pr(\Delta Wt_j = \text{decrease})}{Pr(\Delta Wt_j = \text{no change})} = \mu_2 + \beta_2(\Delta Dep)_{ij-1} + \gamma_2'(\text{covariates})_{ij},
  \]

- **Increase in depressive symptoms model:**
  \[
  \log \frac{Pr(\Delta Dep_j = \text{increase})}{Pr(\Delta Dep_j \neq \text{increase})} = \mu + \beta_1(\Delta Wt = \text{increase})_{ij-1} + \beta_2(\Delta Wt = \text{decrease})_{ij-1} + \gamma'(\text{covariates})_{ij},
  \]

where \(\Delta Wt\) and \(\Delta Dep\) denote the change in weight and depressive symptom outcomes, subscript \(j\) denotes person and subscript \(j\) denotes time period. The change in weight model was a multinomial logit model, and the increase in depression model was a logistic regression model.


We calculated change in weight for Time Periods 2, 3, and 4 and trichotomized these into gained weight, no change in weight, and lost weight as described previously in the section “Weight.” We calculated increase in depressive symptoms for Time Periods 1, 2, and 3 and dichotomized these into increased depressive symptoms versus no increase in depressive symptoms as described previously in the section “Depressive Symptoms.” We used a multinomial logit model because we had three categories of weight change. We used no weight change as the reference category and defined logits for weight gain and weight loss accordingly. We allowed the lagged effect of change in depressive symptoms to vary between the weight gain and weight loss logits. For all other predictors, we tested parameter estimates for equality across the two logits and made subsequent simplifications to the model where possible. We estimated standard errors by using the robust “sandwich” estimator (Huber, 1967) to account for correlation between multiple observations per participant over time.

The symbols \(\gamma_1\) and \(\gamma_2\) represented the vectors of regression coefficients associated with additional independent variables at the baseline (beginning of the time period). We added independent variables to each model, including demographic variables (age, education, race, marital status), baseline functional limitations and medical conditions, increase in functional limitations and number of medical conditions, smoking and drinking behaviors, and depressive symptoms and weight category at the beginning of the time period. We added a change point variable (1996–1998 = 1, 1998–2000 = 2, 2000–2002 = 3) to the final models in order to test whether the aging of this cohort affected changes in the outcome variable.

Our main interest was in estimating \(\beta_1\) and \(\beta_2\), because these coefficients estimated the lagged effect of increased depressive symptoms on change in weight. Exponentiating \(\beta_1\) gave the odds ratio that compared the odds of weight gain (vs no change) for participants showing increased depressive symptoms in the previous time period to the odds of weight gain for participants showing no increase in depressive symptoms in the previous time period; thus, we refer to this as the weight gain odds ratio. Similarly, exponentiating \(\beta_2\) gave the weight loss odds ratio of weight loss (vs no change) for participants previously showing increased depressive symptoms versus those previously showing no increase in depressive symptoms.
The increase in depressive symptoms model was identical to the change in weight model, with the roles of these variables switched from predictor to outcome variable and vice versa. Because the outcome increase in depressive symptoms was dichotomous, we used a logistic regression model. For this model, exponentiating $\beta_1$ gave the odds ratio that compared the odds of an increase in depressive symptoms (vs no increase) for participants showing a weight gain in the previous period versus participants showing no weight change in the previous period. Similarly, exponentiating $\beta_2$ gave the odds ratio that compared the odds of an increase in depressive symptoms for participants showing a weight loss in the previous period versus participants showing no weight change in the previous period.

We used the method of generalized estimating equations (Liang & Zeger, 1986; Zeger & Liang, 1986) in order to estimate regression coefficients. This method accounts for the correlation due to multiple observations available for each survey respondent over time. Generalized estimating equations modeling uses an independence working correlation matrix and a logit link function. We conducted analyses using SAS Version 9.0 (SAS Institute, Cary, NC) and Stata Version 8.2 (StataCorp LP, College Station, TX).

There were a total of 4,253 men and 4,877 women respondents in the HRS data set who were interviewed between 1994 and 2002. In order to account for attrition and missing data when fitting regression models, we employed multiple imputation (Rubin, 1987) using the SAS-based software IVEware (available at http://www.isr.umich.edu/src/smp.ive), which creates imputed data sets using the sequential regression approach (Raghunathan, Lepkowski, van Hoewyk, & Solenberger, 2001). We imputed five different data sets, treating each respondent’s data as an independent data vector that included all variables, baseline and time-period specific. We imputed both covariates and outcomes, if missing. We analyzed each data set using generalized estimating equations as described in the preceding paragraph, and we combined the results using the SAS procedure MIANALYZE. We also created a new variable, “alive,” to categorize eligible participants by whether they lived through the entire study period; we included this in our multivariable models. We presumed that participants with missing data at one point who were never interviewed again had either died or dropped out of the study (alive = 0).

We performed a sensitivity analysis to address the potential ceiling effect of the change in depressive symptoms variable. For example, respondents who reported a CES-D score of 7 or higher were unable to have an increased depressive symptom score of at least two CES-D points, given the maximum score of 8 on any one assessment. We ran the analyses on all participants and then again after removing the cases where a participant had a baseline CES-D score of 7 or greater. Because both sets of analyses yielded nearly identical results, we did not exclude cases where baseline CES-D scores were 7 or greater at the beginning of the time period.

**RESULTS**

Table 1 shows demographic characteristics of male and female participants. For men, the mean BMI increased each year from 1994 to 2002 (range of means = 27.4–28.0), as did the number of functional limitations ($M_s = 0.9$ and 1.3 in 1994 and 2002, respectively). Mean number of depressive symptoms increased from 1994–1998 ($M_s = 2.7$ and 3.0 in 1994 and 1998, respectively) and then slightly decreased in 2000 and held steady in 2002 ($M_s = 2.9$ in 2000 and 2002). Smoking rates decreased over the years (from 25% in 1994 to 18% in 2002), whereas the number of men who had at least one comorbid medical condition increased over the years (from 68% in 1994 to 85% in 2002).

For women, the mean BMI increased each year from 1994 to 2002 ($M_s = 27.2$ and 27.9 in 1994 and 2002, respectively), as did the number of functional limitations ($M_s = 1.5$ and 1.9 in 1994 and 2002, respectively). The mean number of depressive symptoms did not exhibit any specific patterns from 1994–2002 (range of means = 3.0 to 3.3). Smoking rates decreased (from 22% in 1994 to 15% in 2002), and the percentage of female respondents with at least one comorbid medical condition increased from 74% in 1994 to 87% in 2002.

Between 7.8%–12.5% of men and 10.9%–15.5% of women had increases of two or more CES-D points across adjacent study waves. A greater proportion of women than men (19.8%–22.1% vs 16.5%–18.0%) gained 5% or more weight between adjacent study waves. Women also were more likely than men to lose 5% or more weight between adjacent study waves (14.2%–16.5% vs 12.0%–14.8%).

**Increased Depressive Symptoms Model: Men**

For men who did not have a weight change during the previous time period, 9.4% had subsequent depressive symptom increases, compared to 11.6% of men who had gained...
Increased Depressive Symptoms Model: Men

For men who did not have a weight change during the previous time period, 13.0% had subsequent depressive symptom increases, compared to 13.4% of women who had gained weight previously and 14.4% of women who had lost weight previously. In both univariable and multivariable models, weight change was not a significant predictor of subsequent depressive symptom increases in women (Table 4).

Baseline weight category was not a significant predictor of increased depressive symptoms, either in univariable or multivariable models. Although increases in functional limitations, medical conditions, and baseline medical conditions were not significantly predictive, baseline functional limitations were predictive of increased depressive symptoms (adjusted OR = 1.19, 95% CI = 1.04–1.36). Another significant predictor of increased depressive symptoms in women was smoking (adjusted OR = 1.15, 95% CI = 1.03–1.29). The risk of increased depressive symptoms was lower for women with higher education levels.

Change in Weight Models: Men

For men who did not have depressive symptom increases during the previous time period, 15.4% had subsequent weight gain compared to 17.4% of men with previous depressive symptom increases, and 11.8% lost weight during follow-up compared to 14.7% of men with previous depressive symptom increases. In univariable models (Table 3), previous depressive symptom increase was significantly predictive of weight gain (adjusted OR = 1.24, 95% CI = 1.00–1.54) but not weight loss. In adjusted models, however, previous depressive symptom increase was predictive of neither weight loss nor weight gain.

Baseline depressive symptoms were predictive of weight loss (adjusted OR = 1.06, 95% CI = 1.00–1.11) but not weight gain. Increases in functional limitations, medical conditions, and baseline functional limitations were each significant predictors of both weight loss and gain. Current smokers were more likely than non-smokers to lose weight (adjusted OR = 1.29, 95% CI = 1.12–1.48), and both Black and Hispanic men were more likely than White men to lose and gain weight (Black men adjusted OR = 1.15, 95% CI = 1.01–1.31; Hispanic men adjusted OR = 1.30, 95% CI = 1.10–1.53).

Table 3. Predictors of Subsequent Weight Changes in Older Men in the Health and Retirement Study (1994–2002)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Unadjusted OR for Weight Gain (95% CI)</th>
<th>Adjusted OR for Weight Gain (95% CI)</th>
<th>Unadjusted OR for Weight Loss (95% CI)</th>
<th>Adjusted OR for Weight Loss (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depressive symptom increase</td>
<td>1.29 (0.96–1.73)</td>
<td>1.21 (0.92–1.58)</td>
<td>1.24 (1.00–1.54)</td>
<td>1.16 (0.91–1.47)</td>
</tr>
<tr>
<td>Baseline depressive symptoms</td>
<td>1.17 (1.10–1.25)</td>
<td>1.06 (1.00–1.11)</td>
<td>1.14 (1.09–1.19)</td>
<td>1.04 (0.99–1.09)</td>
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<tr>
<td>Baseline weight</td>
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<tr>
<td>Obese (vs normal weight)</td>
<td>1.16 (1.00–1.33)</td>
<td>1.18 (1.01–1.38)</td>
<td>0.94 (0.81–1.10)</td>
<td>0.93 (0.80–1.08)</td>
</tr>
<tr>
<td>Overweight (vs normal weight)</td>
<td>0.90 (0.79–1.01)</td>
<td>0.97 (0.86–1.10)</td>
<td>0.91 (0.81–1.03)</td>
<td>0.96 (0.86–1.08)</td>
</tr>
<tr>
<td>Underweight (vs normal weight)</td>
<td>2.18 (1.30–3.66)</td>
<td>1.33 (0.76–2.34)</td>
<td>2.08 (1.44–3.00)</td>
<td>1.40 (0.94–2.06)</td>
</tr>
<tr>
<td>Baseline functional limitation</td>
<td>1.76 (1.59–1.96)</td>
<td>1.25 (1.14–1.38)</td>
<td>1.48 (1.33–1.64)</td>
<td>1.25 (1.14–1.38)</td>
</tr>
<tr>
<td>Increase in functional limitation</td>
<td>1.55 (1.27–1.88)</td>
<td>1.28 (1.02–1.62)</td>
<td>1.48 (1.28–1.72)</td>
<td>1.25 (1.08–1.46)</td>
</tr>
<tr>
<td>Baseline medical comorbidity</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>1 (vs none)</td>
<td>1.15 (1.00–1.34)</td>
<td>1.00 (0.86–1.17)</td>
<td>1.13 (0.95–1.34)</td>
<td>1.03 (0.87–1.22)</td>
</tr>
<tr>
<td>2+ (vs none)</td>
<td>1.56 (1.36–1.78)</td>
<td>1.09 (0.94–1.27)</td>
<td>1.39 (1.21–1.59)</td>
<td>1.09 (0.95–1.25)</td>
</tr>
<tr>
<td>Increase in medical comorbidity</td>
<td>1.34 (1.20–1.49)</td>
<td>1.17 (1.05–1.32)</td>
<td>1.39 (1.23–1.57)</td>
<td>1.27 (1.13–1.43)</td>
</tr>
</tbody>
</table>

Note: OR = odds ratio; CI = confidence interval.

Increased Depressive Symptoms Model: Women

For women who did not have a weight change during the previous time period, 13.0% had subsequent depressive symptom increases, compared to 13.4% of women who had gained weight previously and 14.4% of women who had lost weight previously. In both univariable and multivariable models, weight change was not a significant predictor of subsequent depressive symptom increases in women (Table 4).

Baseline weight category was not a significant predictor of increased depressive symptoms, either in univariable or multivariable models. Although increases in functional limitations, medical conditions, and baseline medical conditions were not significantly predictive, baseline functional limitations were predictive of increased depressive symptoms (adjusted OR = 1.19, 95% CI = 1.04–1.36). Another significant predictor of increased depressive symptoms in women was smoking (adjusted OR = 1.15, 95% CI = 1.03–1.29). The risk of increased depressive symptoms was lower for women with higher education levels.

Change in Weight Models: Women

For women who did not have depressive symptom increases during the previous time period, 19.8% had subsequent weight changes, and 11.8% lost weight during follow-up compared to 14.7% of men with previous depressive symptom increases. In univariable models (Table 3), previous depressive symptom increase was significantly predictive of weight gain (adjusted OR = 1.24, 95% CI = 1.00–1.54) but not weight loss. In adjusted models, however, previous depressive symptom increase was predictive of neither weight loss nor weight gain.
gain, as compared to 20.9% of women with previous depressive symptom increases. Similarly, 15.2% of women with no depressive symptom increases previously lost weight during follow-up, compared to 16.2% of women who did have prior depressive symptom increases. In univariable models (Table 5), previous increases in depressive symptoms was significantly predictive of weight gain (unadjusted OR = 1.12, 95% CI = 1.00–1.26) but not weight loss. In adjusted models, however, previous depressive symptom increases was not predictive of either weight loss or gain.

Depressive symptoms at the beginning of the time period were, however, predictive of both weight gain (adjusted OR = 1.04, 95% CI = 1.00–1.07) and weight loss (adjusted OR = 1.03, 95% CI = 1.00–1.06) in women. Previous increases in functional limitations and medical conditions as well as baseline functional limitations and medical conditions were all significantly predictive of subsequent weight loss and weight gain. Current smokers were more likely than non-smokers to both gain and lose weight (adjusted OR = 1.15, 95% CI = 1.05–1.25 in both models). The risk of losing weight was lower for women with higher education levels, as well.

**DISCUSSION**

Our study revealed a number of important findings, some new and some that replicate those of previous studies. In summary (Tables 6–8), we found that previous weight loss predicts subsequent increases in depressive symptoms only in men and only in unadjusted models. We found no other evidence that weight change predicts future increases in depressive symptoms in either men or women. Furthermore, baseline weight category was not associated with increased depressive symptoms. After examining the relationship in the opposite direction, we found that an increase in depressive symptoms leads to weight gain (but not weight loss) in both men and women, but only in unadjusted models. In adjusted models, however, baseline depressive symptoms did predict weight loss for men and both weight loss and weight gain for women. This finding confirms that weight changes in both directions are sequela of depressive symptoms for women. Simply put, whereas some individuals gain weight when they get depressed, others lose weight. This could be due to appetite changes causing increased intake of food or, conversely, decreased intake. Our results are congruent with those of previous research studies, although no previous studies have examined the impact of crude changes in weight and depressive symptom scores over time as predictors of the other.

We found no evidence in support of the “jolly fat” hypothesis (Crisp & McGuinness, 1976; Crisp, Queenan, Sittampalam, & Harris, 1980; Palinkas, Wingard, & Barrett-Connor, 1996), because men (and women) in our sample who were overweight or obese at baseline were no less likely than normal weight men (or women) to have subsequent increases in depressive symptoms; this supports the work of Roberts, Strawbridge, Deleger, & Kaplan (2002). Additionally, we did not find evidence to support the work of Roberts and colleagues (2003), who found that obesity predicts the onset of depression in women, because baseline weight did not predict future increases in depressive symptoms in our sample.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Unadjusted OR for Weight Loss (95% CI)</th>
<th>Adjusted OR for Weight Loss (95% CI)</th>
<th>Unadjusted OR for Weight Gain (95% CI)</th>
<th>Adjusted OR for Weight Gain (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depressive symptom increase</td>
<td>1.11 (0.96–1.29)</td>
<td>1.03 (0.98–1.09)</td>
<td>1.12 (1.00–1.26)</td>
<td>1.05 (0.93–1.18)</td>
</tr>
<tr>
<td>Baseline depressive symptoms</td>
<td>1.11 (1.08–1.14)</td>
<td>1.03 (1.00–1.06)</td>
<td>1.09 (1.06–1.13)</td>
<td>1.04 (1.00–1.10)</td>
</tr>
<tr>
<td>Baseline weight</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obese (vs normal weight)</td>
<td>1.64 (1.44–1.87)</td>
<td>1.44 (1.24–1.66)</td>
<td>1.34 (1.20–1.50)</td>
<td>1.19 (1.06–1.35)</td>
</tr>
<tr>
<td>Overweight (vs normal weight)</td>
<td>1.32 (1.17–1.48)</td>
<td>1.26 (1.11–1.43)</td>
<td>1.26 (1.13–1.41)</td>
<td>1.21 (1.08–1.37)</td>
</tr>
<tr>
<td>Underweight (vs normal weight)</td>
<td>1.93 (1.46–2.55)</td>
<td>1.57 (1.18–2.09)</td>
<td>2.21 (1.78–2.76)</td>
<td>1.88 (1.50–2.37)</td>
</tr>
<tr>
<td>Baseline functional limitation</td>
<td>1.60 (1.44–1.78)</td>
<td>1.13 (1.04–1.22)</td>
<td>1.52 (1.17–1.99)</td>
<td>1.13 (1.04–1.22)</td>
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<tr>
<td>Increase in functional limitation</td>
<td>1.36 (1.19–1.55)</td>
<td>1.17 (1.02–1.35)</td>
<td>1.48 (1.25–1.75)</td>
<td>1.32 (1.10–1.59)</td>
</tr>
<tr>
<td>Baseline medical comorbidity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 (vs none)</td>
<td>1.36 (1.15–1.62)</td>
<td>1.22 (1.03–1.45)</td>
<td>1.19 (1.06–1.34)</td>
<td>1.12 (0.98–1.26)</td>
</tr>
<tr>
<td>2+ (vs none)</td>
<td>1.76 (1.51–2.04)</td>
<td>1.30 (1.10–1.54)</td>
<td>1.45 (1.31–1.62)</td>
<td>1.20 (1.07–1.35)</td>
</tr>
<tr>
<td>Increase in medical comorbidity</td>
<td>1.33 (1.17–1.50)</td>
<td>1.20 (1.05–1.36)</td>
<td>1.37 (1.21–1.55)</td>
<td>1.26 (1.12–1.43)</td>
</tr>
</tbody>
</table>

Note: OR = odds ratio; CI = confidence interval.
Table 6. Summary of Significant Predictors of Increased Depressive Symptoms in Men and Women Surveyed in the Health and Retirement Study (1994–2002)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Previous weight change</td>
<td>Yes +, weight loss vs no change&lt;sup&gt;a&lt;/sup&gt;</td>
<td>No</td>
</tr>
<tr>
<td>Baseline weight category</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Increase in functional limitations</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Increase in medical conditions</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Baseline functional limitations</td>
<td>Yes +</td>
<td>Yes +</td>
</tr>
<tr>
<td>Baseline medical conditions</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Current smoking</td>
<td>No</td>
<td>Yes +</td>
</tr>
<tr>
<td>Education level</td>
<td>No</td>
<td>Yes –</td>
</tr>
</tbody>
</table>

Note: In adjusted models, p < .05 unless otherwise noted.
<sup>a</sup>Significance found only in univariate models.

Table 7. Summary of Significant Predictors of Weight Gain in Men and Women Surveyed in the Health and Retirement Study (1994–2002)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Previous increased depressive symptoms</td>
<td>Yes +&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Yes +&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Baseline depressive symptoms</td>
<td>Yes +</td>
<td>Yes +</td>
</tr>
<tr>
<td>Increase in functional limitations</td>
<td>Yes +</td>
<td>Yes +</td>
</tr>
<tr>
<td>Increase in medical conditions</td>
<td>Yes +</td>
<td>Yes +</td>
</tr>
<tr>
<td>Baseline functional limitations</td>
<td>Yes +</td>
<td>Yes +</td>
</tr>
<tr>
<td>Baseline medical conditions</td>
<td>No</td>
<td>Yes +</td>
</tr>
<tr>
<td>Smoking</td>
<td>No</td>
<td>Yes +</td>
</tr>
<tr>
<td>Race/ethnicity</td>
<td>Yes, Blacks and Hispanics &gt;</td>
<td>No, Whites</td>
</tr>
<tr>
<td>Education level</td>
<td>No</td>
<td>Yes –</td>
</tr>
</tbody>
</table>

Note: In adjusted models, p < .05 unless otherwise noted.
<sup>a</sup>Significance found only in univariate models.

We did find that increases in depressive symptoms were predictive of weight gain in men and women (albeit only in unadjusted models) and that baseline number of depressive symptoms predicted weight loss in both men and women and weight gain in women only. This latter finding partly supports the findings of Raikkonen and colleagues (2002) and Hallstrom and Noppa (1981), who found that baseline depressive symptoms are predictive of obesity at follow-up.

Functional limitations and medical conditions were highly predictive variables in many of the models we tested. Increases in functional limitations and number of medical conditions were significantly predictive of both weight loss and weight gain for men and women. These findings are congruent with previous research that concludes that disability or functional limitation is related to both relative weight and weight change in elders (Coakley et al., 1998; Friedmann, Elasy, & Jensen, 2001; Jenkins, 2004; Jensen & Friedmann, 2002; Launer, Harris, Rumpel, & Madans, 1994; Zamboni et al., 1999; Zoico et al., 2004). Although we did not find significant associations between increases in functional limitations or medical conditions and subsequent increases in depressive symptoms, we did find that having functional limitations at baseline heightened the risk of having increased depressive symptoms subsequently in both men and women. This finding is congruent with several previous studies that have shown that functional status and disability are related to depression (Cronin-Stubbbs et al., 2000; Penninx, Leveille, Ferrucci, van Eijk, & Guralnik, 1999; Roberts, Kaplan, Shema, & Strawbridge, 1997; Travis, Lyness, Shields, King, & Cox, 2004).

Other important demographic and behavioral predictors of weight change and depressive symptom increases are worth noting. First, smoking was significantly predictive of weight gain, weight loss, and depressive symptom increases in women, but only weight loss in men. Second, both Black and Hispanic men were more likely than White men to have weight changes, possibly due to Black and Hispanic men having more functional limitations and medical comorbidity than White men (Kington & Smith, 1997; Moody-Ayers, Mehta, Lindquist, Sands, & Covinsky, 2005). Other studies, however, have failed to find significant differences by racial or ethnic status (Wu & Schimmele, 2005). Third, the risk of increased depressive symptoms and weight changes decreased with higher education levels in women only.

Although previous research has determined that depression and obesity are highly correlated, the direction of causality and underlying mechanisms have remained unclear. No previous study has simultaneously examined the impact of changes in weight and depressive symptoms over time as predictors of the other. In our study, we accomplished our primary goal of clarifying the relationship between changes in weight and depressive symptoms over time in the cohort of U.S. adults enrolled in the HRS, a nationally representative sample of nearly 9,000 adults born between 1931 and 1941. The 2-year cycle of repeated measures in the HRS provided a unique opportunity to unravel the complicated pathways and interactions between weight change and changes in depressive symptoms, as well as to test previously proposed conceptual frameworks of these relationships. We were able to examine these complex relationships using lagged covariate models stratified by gender because of the large sample size and resulting power of the study. Furthermore, each of our analyses controlled for baseline levels of the independent variable of interest (either BMI or number of depressive symptoms), enabling us to isolate the impact of changes of one variable on
subsequent changes in the other. Investigators can use these results to further the line of research examining the complex pathways between depressive symptoms and weight in older adults.

Limitations of our study are the following. First, our findings only generalize to older adults. The relationship we found between depressive symptoms and weight may be different than the relationship that might be found among younger adults, as medical comorbidity and functional limitations associated with aging may cause weight change and may also be associated with changes in mood. It will be important to address this issue in future analyses. Second, participants self-reported the height and weight data used to calculate BMI. We were, however, only assessing change in BMI (and not crude BMI). Furthermore, we categorized weight change into less than or greater than a 5% change from the previous time point, so the errors of self-reports are not of such concern. Third, the HRS did not include an assessment of depression from an operationalized instrument. This limitation, however, may be not be so great an issue in analyses of older adults, who tend to experience symptoms of depression differently than younger adults and who more often experience symptoms below the threshold necessary to meet diagnostic criteria (Blazer, Hughes, & George, 1987). Moreover, the CES-D is one of the most widely used and validated depressive symptom survey instruments, and numerous studies of depression in elderly adults have shown that the CES-D is a valid depression tool (Andresen, Malmgren, Carter, & Patrick, 1994; Irwin, Artin, & Oxman, 1999; Lewinsohn, Seeley, Roberts, & Allen, 1997). Finally, because study organizers did not systematically collect data on medication use from all participants in the HRS, we are unable to decipher the role of medications in influencing both weight and depressive symptoms over time. The results of this study, however, will provide us with preliminary findings on which to base future work that will examine the effect of medications and treatment more closely.

Depression and obesity affect a large proportion of older adults, and both conditions are important risk factors for morbidity and mortality. Our study has important implications for future research projects as well as clinical practice and prevention efforts. Based on our findings, it is apparent that researchers need to examine the causal pathways between weight and depressive symptoms in the context of functional limitations and, especially, medical comorbidity as mediators or moderators of the significant relationships. For example, do increases in depressive symptoms in women “cause” weight gain indirectly through new comorbid medical conditions or greater functional limitations? Similarly, do increases in depressive symptoms in men “cause” weight loss indirectly through functional limitations? Further research is needed to elucidate the mechanisms that underlie these relationships. It also will be important to look at what kinds of comorbid conditions have the most influence on changes in weight and depressive symptoms so that professionals can target them in preventative efforts. Other life events, such as retirement and spousal death, may influence these constructs and need to be investigated as well.

While investigators are studying these research questions, experts can improve preventative efforts by targeting the risk factors for increases in depressive symptoms, weight loss, and weight gain that we identified in this study. Efforts to reduce increased depressive symptoms in older adults should focus on reducing functional limitations. Physicians should monitor men who lose weight for increased depressive symptoms. Health professionals should monitor both men and women with increased depressive symptoms for weight gain. Providers should recognize that men with high levels of depressive symptoms are more likely to lose weight, and that women with high levels of depressive symptoms are more likely to either lose or gain weight, than are persons without many depressive symptoms. Increases in and high levels of both functional limitations and medical conditions are good reasons to monitor for weight changes in both directions among both men and women.


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