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# Challenging the “jolly fat” hypothesis among older adults: High body mass index predicts increases in depressive symptoms over a 5-year period

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**Challenging the “jolly fat” hypothesis among older adults: High body mass index predicts increases in depressive symptoms over a 5-year period**

**Abstract**

Several investigators have observed lowered risk for depression among obese older adults, coining the “jolly fat” hypothesis. We examined this hypothesis using baseline and 5-year follow-up BMI, depressive symptoms, and covariates from 638 community-based older adults. High objectively measured BMI and functional limitations predicted increased future depressive symptoms. However, symptoms did not predict future BMI. Self-reported BMI showed similar associations despite underestimating obesity prevalence. Results did not differ on the basis of gender. Results for this study, the first longitudinal reciprocal risk analysis between objectively measured BMI and depressive symptoms among older adults, do not support the “jolly fat” hypothesis.

**Keywords:** obesity, depression, geriatrics, epidemiology, physical function

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Recent meta-analytic evidence suggests the existence of a relationship between obesity and depression (de Wit et al. 2010). However, the directional nature of this relationship remains less clear among differently aged and gendered samples. Among longitudinal studies, there is some evidence that depression status predicts later obesity. In a meta-analysis of 17 samples, Blaine (2008) estimated a large effect size of baseline depression status on subsequent obesity when controlling for baseline obesity and other covariates. However, the only study composed entirely of older adults (age > 50; DiPietro et al. 1992) found that baseline depression was associated with marginally lower risk for future obesity, results consistent with the “jolly fat” hypothesis which posits that there are protective effects of obesity on risk for depressive symptoms (Crisp & McGuinness 1976; Crisp et al. 1980; Palinkas et al. 1996).

Results supportive of the "jolly fat" hypothesis have primarily been observed for older cohorts, have sometimes been stronger for males than for females, and have been obtained mostly from cross-sectional studies. Indeed, a major conclusion from a systematic review of epidemiological research is that there is a dearth of high quality longitudinal studies which measure the effect of obesity on future depression status (Atlantis & Baker 2008). Of the four studies which met their quality inclusion standards, three were composed of older adults, all from the same sample in Alameda County, California (Roberts et al. 2003; Roberts et al. 2000; Roberts et al. 2002). Contrary to the “jolly fat” hypothesis, both one and five-year longitudinal analysis revealed that self-reported obesity predicted significantly higher risk for depressive symptoms even with control for an array of covariates. These results have been supported by more recent longitudinal analysis of older adults, though findings vary by obesity measurement used. While studies using self-reported BMI have found obesity increasing risk for depressive

1 symptoms among women (Pan et al. 2012), but not in men (Xiang & An 2015), a study using  
2 objectively measured BMI found this association only among men (Vogelzangs et al. 2010).

3         To our knowledge, only two longitudinal studies have systematically assessed the  
4 reciprocal effects of depression and obesity among older adults (Roberts et al. 2003; Pan et al.  
5 2012). Both studies found that obese individuals were at a higher risk for reporting clinical  
6 levels of depressive symptoms, anti-depressant use, or receiving a diagnosis of depression 5-10  
7 years later. This was true even with control for baseline depression status and a range of  
8 covariates. Conversely, although Pan et al. (2012) found evidence of a bidirectional relationship,  
9 Roberts et al. (2003) did not find that depressed individuals were at a higher risk for obesity five  
10 years later. Compared with prior longitudinal studies on depression and obesity, the covariate  
11 models employed by Roberts et al. and Pan et al. were considerably more extensive.

12         Although these studies have methodological strengths, both were also limited to using  
13 self-reported obesity. Prior studies have found systematic differences between objectively  
14 measured ( $BMI_O$ ) and self-reported BMI ( $BMI_{SR}$ ). Specifically, individuals tend to overestimate  
15 height and underestimate weight, which results in a likely underestimation of obesity prevalence,  
16 particularly among older adults (Kuczmarski et al. 2001; Keith et al. 2011). Additionally, Keith  
17 et al. (2011) noted that  $BMI_{SR}$  failed to consistently predict mortality outcomes which were  
18 associated with  $BMI_O$ . As such, it is possible that  $BMI_{SR}$  and  $BMI_O$  may associate differently  
19 with other psychobiological outcomes.

20         The present study uses objective measurements of BMI from the Maine-Syracuse  
21 Longitudinal Study (MSLS) to investigate the reciprocal effects of depressive symptoms and  
22 BMI and to further test the jolly-fat hypothesis among older adults. We hypothesize that BMI

1 will be positively associated with concurrent and future depressive symptoms; i.e., will be seen  
2 with cross-sectional and longitudinal analyses.

### 3 **Methods**

#### 4 **Participant Selection**

5 The MSLS includes 7 waves of demographic, cardiovascular risk factor and cognitive  
6 performance data gathered over a period of 35 years employing a sample of community-dwelling  
7 adults residing in Central New York. Since the original MSLS baseline [1976], individuals have  
8 been excluded based on diagnosed alcoholism, diagnosed psychiatric disorder, or inability to  
9 speak English. In this and other subsequent MSLS studies, additional exclusionary criteria have  
10 been included on a study-by-study basis. The University of Maine Institutional Review Board  
11 approved this study (reference number: 2005-07-04) and informed consent was obtained from all  
12 participants.

#### 13 **Attrition**

14 At baseline, older adults were selected for this analysis ( $n = 893, \geq 50y$ ). Participants no  
15 longer residing in New York State were lost to the study due to changes in research funding  
16 limiting travel of the investigative team out of state. Of the baseline participants, 71.4%  
17 responded again and were eligible at wave 7 follow-up ( $n = 638$ ). Additionally, participants  
18 were excluded from regression analysis for missing depressive symptom data ( $n = 27$ ), for  
19 missing objectively measured (BMI<sub>O</sub>;  $n = 42$ ) or self-reported BMI (BMI<sub>SR</sub>;  $n = 117$ ), or baseline  
20 covariate data ( $n = 25$ ).

21 Compared with individuals who were lost to follow-up or excluded, individuals retained  
22 at follow-up were 2.52 years younger (CI: 1.04 - 4.00,  $p = .002$ ), 2.5-points lower on the Zung  
23 Depression Inventory (ZDI; CI: 1.06 - 3.95,  $p = .001$ ), and had 0.48 fewer physical deficits (CI:

1 0.30 - 0.66,  $p < .001$ ) at baseline. However, neither group differed in baseline educational level,  
2 marital status, number of recent life changes, social activity/isolation level, number of chronic  
3 illnesses, or BMI<sub>SR/O</sub> ( $p > .05$ ). Table 1 presents baseline descriptive statistics for participants  
4 retained at follow-up.

5 INSERT TABLE 1 HERE

## 6 **Study Design**

7 The present study uses BMI, depressive symptoms, and covariate information from wave  
8 6 of the MSLS [2001-2005] as baseline data. Approximately 5-years later [2006-2010] at wave  
9 7 follow up, BMI and depressive symptoms were obtained. Objective measurement of height,  
10 weight, and physical performance obtained at wave 6 and wave 7 allowed for more  
11 comprehensive prospective models than were available in previous waves.

## 12 **Measures of Obesity and Depressive Symptoms**

13 Objectively measured height and weight at baseline and follow-up was assessed in the lab  
14 without shoes or heavy clothing to calculate BMI ( $\text{kg}/\text{m}^2$ ). In addition, self-reported height and  
15 weight were used to calculate an alternative index of BMI. BMI was analyzed as a continuous  
16 outcome and baseline adjustment variable and obesity was defined based on the World Health  
17 Organization obesity standard ( $\text{BMI} \geq 30$ ; WHO, 2010).

18 Baseline and outcome depressive symptoms were measured with the ZDI (Zung, 1965).  
19 The ZDI is composed of 20 self-reported, Likert scale items ranging in frequency from 1 “a little  
20 of the time” to 4 “most of the time” and summed to create a total raw score. Raw scores were  
21 then divided by 0.8, resulting in an index score range from 25 to 100 (Hunter & Murphy 2011).  
22 At baseline, ZDI items showed good internal consistency ( $\alpha = .80$ ). The ZDI has been reported  
23 to lack sufficient sensitivity to act as a categorical diagnostic measure (Thurber et al. 2002). As

1 such, index scores were used to represent the continuum of depressive symptom severity rather  
2 than major depressive disorder incidence. Individuals missing more than 2 items were excluded  
3 from analysis, scores for 2 or fewer missing items were imputed based on the mean of available  
4 ZDI data.

### 5 **Covariates**

6 Covariates were selected based primarily on the extensive models used by Roberts et al.  
7 (2003), their theoretical relevance as posited by Atlantis and Baker (2008), and their associations  
8 with BMI and ZDI within our own sample. This included adjustments for age, gender, education  
9 in years, marital status (married/living w/ partner vs. other), social isolation (no close friendships  
10 and/or co-residents vs. other), social activity (see: Robbins et al. 1994), recent life events  
11 (yes/no), physical functioning, and chronic medical conditions.

12 For the chronic illness composite, participants completed questionnaire items asking  
13 whether their doctor had told them if they had a range of medical issues during the past 6 years.  
14 This included summing occurrence data on heart trouble, high blood pressure, asthma,  
15 bronchitis/emphysema, rheumatoid arthritis, diabetes, stroke, cancer, cataracts, and circulatory  
16 issues.

17 The physical functioning deficit composite was measured with 3 timed performance  
18 tasks. Participants were asked to 1) stand up 3 times from a hard-backed chair with their arms  
19 folded, 2) walk forward ten feet, turn 180°, and walk back to their starting position, 3) from a  
20 standing position, complete a full 360° turn. Participants unable to complete a task or whose task  
21 completion time was among the slowest quartile in the baseline sample were considered to have  
22 a deficit in that area of performance. Total deficit scores ranged from “0” (no deficit) to “3”

1 (high deficit). Prior research has shown that a performance deficit in any of these tasks is  
2 predictive of future functional dependence in activities of daily living (Gill et al. 1995).

3 Self-reported family income, smoking status, and ethnicity were all collected and  
4 considered for analysis, but ultimately dropped from our final models. Family income was  
5 considered because it has been found to be related to depressive symptoms (Mendes De Leon et  
6 al. 1994). However, it was dropped from analysis because 1) a large number ( $n = 106$ ) of  
7 participants indicated that they “would rather not answer” this item and 2) when tested as a  
8 model covariate, family income did not change the results reported below. Smoking status was  
9 considered based on the quality assessment criteria by Atlantis and Baker (2008), but was  
10 dropped due to a lack of observed association with BMI<sub>SR/O</sub> or ZDI scores. Although ethnicity  
11 may moderate the relationship between obesity and depressive symptoms (Xiang & An 2015),  
12 detailed group analysis was not conducted due to a lack of sufficient sample size for non-white  
13 participant groups (Black/Hispanic/Asian/American Indian combined;  $n = 42$ ).

#### 14 **Statistical Analysis**

15 Hierarchical multivariate regression models were used to explore the cross-sectional and  
16 longitudinal association between BMI and ZDI with adjustment for covariates. Baseline (wave  
17 6) values for ZDI and BMI, respectively, were used as covariates for models in which wave 7  
18 ZDI or wave 7 BMI was the outcome of interest. Model 1 of the cross-sectional and longitudinal  
19 analyses included baseline age, sex, education, and marital status as covariates. Model 2  
20 contained all covariates from Model 1 in addition to life events, social isolation, and social  
21 activity. Model 3 included all covariates from Model 2 in addition to chronic health conditions  
22 and physical performance deficits.



1 Model variables met assumptions for parametric multivariate regression (Durbin-Watson;  
2  $1.94 \geq d \leq 2.04$ ) and minimal collinearity was observed (Max. variance inflation factor  $\leq 1.97$ ).  
3 Tests for non-linear associations between BMI and ZDI were conducted with the addition of  
4 quadratic predictor terms with adjustment for linear terms. Neither inclusion of quadratic terms  
5 for wave 6 BMI<sub>SR/O</sub> ( $p > .25$ ) nor wave 6 ZDI ( $p \geq .09$ ) improved fit across all models.

6 Statistical analyses were conducted with SPSS software (version 23.0).

## 7 Results

### 8 Objective vs. Self-Reported BMI

9 Bivariate and paired-samples t-test analysis were used to examine the convergent validity  
10 between objective and self-reported height and weight at baseline for participants who completed  
11 both wave 6 and wave 7. Participants both significantly underestimated their weight [ $t(601) = -$   
12  $10.71, p < .001$ ] by an average of 2.10 kg (95% CI = 1.72kg, 2.48kg) and overestimated their  
13 height [ $t(574) = 6.02, p < .001$ ] by an average of .94cm (95% CI = .63cm, 1.25cm). However,  
14 self-reported and objective measurement of height ( $r = .93, p < .001$ ) and weight ( $r = .97, p <$   
15  $.001$ ) were also highly associated. BMI<sub>SR</sub> obesity prevalence (29.2%) was significantly lower  
16 than what would be expected [ $\chi^2(1, 572) = 348.57, p < .001$ ] based on the BMI<sub>O</sub> obesity  
17 prevalence in our sample (38.8%).

### 18 Model Analyses

19 Results of analyses for cross-sectional and longitudinal multivariate regression models  
20 are presented in Table 2. With regard to the cross-sectional association between baseline BMI  
21 and ZDI, across all models the adjusted  $R^2$  values ranged from .07 to .20. BMI<sub>O</sub> predicted higher  
22 ZDI in Model 1 and Model 2 ( $p$ 's  $< .01$ ), but not in Model 3 ( $p = .16$ ). BMI<sub>SR</sub> predicted higher  
23 ZDI in Model 1 ( $p < .01$ ), but not in Model 2 or Model 3 ( $p$ 's  $> .10$ ).

1           Regression models examining the longitudinal association between baseline BMI and 5-  
2 year follow-up ZDI included adjustment for baseline ZDI as well as the other covariates. Across  
3 all models, adjusted  $R^2$  values ranged from .44 to .46. Baseline ZDI was strongly correlated with  
4 follow-up ZDI ( $r = .65, p < .001$ ), accounting for 26.6% to 35.6% of the total variance across all  
5 models. Both higher  $BMI_O$  and  $BMI_{SR}$  at baseline predicted higher ZDI at follow-up across all  
6 three regression models ( $p$ 's  $< .001$ ).

7           In addition to BMI, the covariates age and physical functioning were significant  
8 predictors of follow-up ZDI. For instance, in the  $BMI_O$  models: 1) older age was associated with  
9 higher follow-up ZDI in Model 1 ( $b = .07, SE = .03, p = .029$ ) and in Model 2 ( $b = .07, SE = .03,$   
10  $p = .023$ ), but not when adjusting for physical ailments and physical functioning in Model 3 ( $p >$   
11  $.25$ ); and 2) baseline physical functioning deficits predicted higher follow-up ZDI ( $b = .95, SE =$   
12  $.29, p = .001$ ) in the full regression model (Model 3).

13           Regression models examining the longitudinal association between baseline ZDI and  
14 follow-up BMI included adjustment for baseline BMI and the other covariates. Across all  
15 models, adjusted  $R^2$  values ranged from .68 to .78. Baseline BMI was highly correlated with  
16 follow-up BMI ( $r_{objective} = .89, r_{SR} = .83$ ), accounting for 56.0% to 67.7% of the total variance  
17 across all models. For both BMI variables across all three models baseline ZDI failed to predict  
18 follow-up BMI ( $b \leq .02, p \geq .20$ ).

19           INSERT TABLE 2 HERE

20           Supplementary analyses were conducted to examine gender differences, given that  
21 evidence supporting the "jolly fat" hypothesis has been found predominantly for males, that is,  
22 others have found that obese men and women have differential levels of risk for depressive  
23 symptoms (Atlantis & Baker 2008; Xiang & An 2015). Adjusted for all other cross-sectional

1 (wave 6) model 3 covariates, females reported higher ZDI scores than males (in BMI<sub>O</sub> model,  $b$   
2 = 2.18,  $SE = .66$ ; in BMI<sub>SR</sub> model,  $b = 2.16$ ,  $SE = .68$ ,  $p$ 's < .01). However, results for  
3 BMI\*gender interaction terms included in the BMI<sub>SR/O</sub> cross-sectional and longitudinal models  
4 were non-significant ( $p$ 's > .25) when predicting either baseline ZDI or longitudinal change in  
5 ZDI.

## 6 **Discussion**

7 In this longitudinal study, we tested the “jolly fat” hypothesis in the context of assessing  
8 the relationship between depressive symptoms and BMI among a community sample of older  
9 adult men and women. We found that older adults with higher baseline BMI reported increases  
10 in depressive symptoms over a five-year period. This was true even when adjusting for baseline  
11 depressive symptoms, age, sex, education, marital status, social isolation, social activity, chronic  
12 illness, and physical functioning. These results were unchanged whether self-reported or  
13 objectively measured BMI was used, thus validating the self-report findings of Roberts et al.  
14 (2003).

## 15 **Strengths**

16 This study adds methodological strength to the currently limited longitudinal research on  
17 obesity and depression among older adults, much of which is lacking objective measurement of  
18 BMI (Atlantis & Baker 2008). Although we found in our sample that objective measurement  
19 and self-reported height and weight were highly correlated and similar in predicting baseline  
20 level and change in depressive symptoms, there was also a small, but consistent tendency for  
21 participants to underestimate weight while overestimating height, resulting in a considerable  
22 underestimation of the true obesity prevalence within our sample. While using self-reported  
23 BMI as a continuous variable is likely justifiable for associative analysis, logit models, in which

1 dependent variables are categorical (i.e., self-reported obesity; Cox 1958) may experience a  
2 considerable loss of predictive power due to misclassification. Additionally, researchers  
3 studying obesity prevalence are encouraged to either use objective BMI or to consider statistical  
4 adjustments for reporting bias among older adults.

5         Regression models also incorporated a composite measure of physical functioning which  
6 consisted of objectively timed performance tasks predictive of difficulty in activities of daily  
7 living (Gill et al. 1995), which up until this point has been missing from associative research  
8 examining depression and obesity. Additionally, although Roberts et al. (2003) adjusted for self-  
9 reported functional limitations (e.g., bathing, dressing), they did not include objective  
10 measurement of physical functioning. Prior studies have reported moderate overlap, but distinct  
11 differences between self-reported and objectively measured physical functioning. Until now,  
12 studies have been unable to account for the potentially confounding associations between self-  
13 reported physical functioning and well-known dimensions of depression such as self-efficacy  
14 and mastery rather than actual performance (Becofsky et al. 2013; Kempen et al. 1996).

15         Lastly, in this study we observed changes in BMI and depressive symptoms over time by  
16 including both baseline and follow-up levels in our models. This allowed us to simultaneously  
17 test the bidirectional associations between BMI and depressive symptoms among older adults,  
18 adding to the scant research with such a design (Pan et al. 2012; Roberts et al. 2003). Although  
19 there is evidence for symptoms of depression being both an outcome and predictor of incident  
20 obesity (Blaine 2008; Atlantis & Baker 2008), to our knowledge no meta-analysis has  
21 statistically accounted for potential publication bias. As such, the strength of pooled evidence  
22 for bidirectionality as assessed by non-reciprocal models may be overestimated. In the context of

1 a reciprocal analysis and a sufficiently powered sample, we observed significant directional  
2 associations with BMI as predictor, but not as an outcome of baseline depressive symptoms.

### 3 **Limitations**

4 It should be noted that our sample was both highly educated and predominately white.  
5 This limited our ability to conduct a more thorough analysis of socioeconomic and ethnic group  
6 differences which may moderate the association between obesity and depressive symptoms  
7 (Xiang & An 2015). However, with regard to our main predictor/outcome variables, obesity and  
8 depressive symptoms, our sample did not vary considerably from national estimates among older  
9 adults (Center for Behavioral Health Statistics and Quality 2015; Ogden et al. 2014). For  
10 additional health demographic information of the MSLS sample see Crichton et al. (2014).

11 Regarding attrition, participants who were available at follow-up tended to be younger,  
12 less likely to report depressive symptoms, and less functionally impaired compared with those  
13 who were unavailable. Although functional impairment and to a lesser extent advanced age were  
14 associated with increased depressive symptoms in our models, it is possible that the magnitude  
15 of their effects were partially attenuated. Similarly, the effect of depressive symptoms on BMI  
16 may have been partially attenuated; however, there was little evidence for a bidirectional trend.

17 Our results indicate that higher BMI may contribute to increases in depressive symptoms  
18 over time. Despite our findings, it is possible that chronic illness and low physical functioning,  
19 which tend to increase risk for depressive symptoms may contribute to weight loss among aging  
20 adults as well as weight gain due to lowered physical activity and loss of muscle tone (St-  
21 Arnaud-McKenzie et al. 2010). However, only one individual in our wave 6 sample was  
22 measured to be underweight by objective measurement ( $BMI < 18.5$ ). As such, our conclusions  
23 are limited to normal and overweight/obese older adults and our results may mask associations

1 between low-weight and depressive symptoms mediated by nutrient deficits as a result of  
2 appetite loss (i.e., B12, folate; Kim et al. 2008). However, our results are consistent with the  
3 homeostasis theory of obesity which posits that some individuals may over-consume energy-  
4 dense, low-nutrient foods (e.g, sugar-sweetened beverages) as a form of emotion regulation  
5 (Marks 2016). These homeostatic disturbances extended over the course of several years may  
6 contribute to continued weight gain and declines in physical health which in turn increases risk  
7 for experiencing depressive symptoms.

### 8 **Conclusions and Future Directions**

9       The results of this study clearly do not support the “jolly fat” hypothesis among older  
10 adults. This is an important finding because the notion that obesity is protective may lead to  
11 behaviors that are not consistent with good health practices. Higher BMI was related to greater  
12 risk, not less risk, for depressive symptoms. There was no indication that the BMI-associated  
13 risk for depressive symptoms interacted with gender. Studies that obtained results supportive of  
14 the “jolly fat” hypothesis among older adults, and predominantly for males, were conducted two  
15 or more decades ago (Crisp & McGuinness 1976; Crisp et al. 1980; Palinkas et al. 1996; DiPietro  
16 et al. 1992). It is possible that increased awareness of the diversity and severity of health risks  
17 associated with obesity has made positive associations with depressive symptoms more likely not  
18 only through resultant individual perceptions of threat to health but also through an increased  
19 prevalence of perceived weight-based discrimination overall among U.S. adults (Andreyeva et al.  
20 2008). Although women likely experience greater weight-related discrimination overall  
21 compared with men, more recent analysis suggests that these differences may not extend into  
22 older adulthood (Roehling et al. 2007; Eichner 2002; Puhl et al. 2008). Lastly, recent findings do  
23 suggest that older adults and especially the oldest-old ( $\geq 80y$ ) experience considerably lower

1 rates of weight-based discrimination compared with younger adults (Puhl et al. 2008; Jackson et  
2 al. 2015), which may explain previously inconsistent findings between younger and older  
3 samples.

4         Conversely, we did not find any association between baseline depressive symptoms and  
5 BMI change five years later, contrary to the 10-year bidirectional results reported by Pan et al.  
6 (2012). It is possible that behaviors associated with depressive symptoms (e.g., appetite  
7 disturbance, lowered activity) require a longer incubation period in order to cause significant  
8 weight gain among older adults. In our own 5-year models, BMI was measured to be highly  
9 stable, which may have masked the potential for more subtle weight gain over a longer duration.

10         Regardless of gender, higher baseline BMI was associated with a net increase of  
11 depressive symptoms. Although this latter finding is similar to the longitudinal results of  
12 Roberts et al. (2003), it differs from other cross-sectional and longitudinal results that have  
13 shown obese women to be at a higher risk for depressive symptoms in comparison to men  
14 (Atlantis & Baker 2008; Xiang & An 2015). Our results indicate that although older adult  
15 women may be at a higher risk for depressive symptoms, their risk for depressive symptoms as a  
16 function of BMI does not differ significantly from that of older adult men. It is possible that  
17 gender differences in weight-based discrimination diminish in older adulthood or that other  
18 forms, such as age-based discrimination become more salient (Puhl et al. 2008). Future research  
19 should investigate the potential mediating role of weight-based discrimination among obese  
20 older adults in the development of depressive symptoms. In a review recently published by  
21 Marks (2015), only one state (i.e., Michigan) was identified as having laws prohibiting weight  
22 discrimination. However, this also opens up the possibility of tracking the efficacy of such

1 legislation on reducing the prevalence of weight-based discrimination and incident depression  
2 among overweight/obese individuals.

3 Objective physical impairment also independently predicted changes in depressive  
4 symptoms and its combined association with chronic illness attenuated much of the association  
5 between depression and aging. Future analysis should explore the possible mediating effect of  
6 physical functioning interventions on the relationship between obesity and depressive symptoms.  
7 It is possible that older obese individuals, who are more likely to experience depressive  
8 symptoms may benefit from interventions targeting limitations in physical functioning.  
9 Increased physical activity, which has been shown to reduce the risk for depressive symptoms  
10 among obese individuals (King et al. 2013), may play a key role by slowing the rate of functional  
11 impairment independent of weight loss (Chalé-Rush et al. 2010).

12

### 13 **Supplementary Information Requests**

14 Additional information relating to this article may be obtained by request by emailing the  
15 primary author.

### 16 **Conflicts of Interest**

17 The authors declare that there are no competing financial interests in relation to the work  
18 described.

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**Table 1 Wave 6 Baseline Variables**

Variable	Value	% Female	% Male	<i>p</i> < .05
Age	50-59	32.0	29.6	
	60-69	25.7	35.6	
	70-79	32.8	26.4	
	80 or older	9.5	8.4	
Years of Education ≥ 12		94.6	95.6	
Married/Cohabiting		54.5	79.6	*
Recent Life Events		76.8	66.4	*
Social Activity	Mean(SD)	21.7(5.3)	20.3(4.8)	
Moderate/ High Isolation		30.4	18.3	*
Zung Depression Inventory	< 40	43.8	47.8	
	40-49	31.8	34.9	
	50-59	13.7	18.3	
	≥ 60	6.1	3.6	
Obesity Rate (objective)		38.3	39.6	
Obesity Status (self-report)		29.0	29.5	
Chronic Conditions	0	34.8	32.8	
	1	19.8	57.8	
	2	16.0	20.0	
	3 or more	29.4	22.4	
Physical Deficits	None	53.6	60.4	
	Low	15.7	16.8	
	Medium	14.7	6.8	*
	High	16.0	16.0	

*\*p* < .05 refers to differences in category prevalence by gender

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**Table 2. Regression coefficients showing relations between BMI and depressive Symptoms**

<u>Cross-sectional</u>						
	<u>Objective BMI<sup>A</sup></u>			<u>Self-Report BMI<sup>B</sup></u>		
	<i>b</i>	<i>SE</i>	<i>p-value</i>	<i>B</i>	<i>SE</i>	<i>p-value</i>
<b>Model 1</b>	0.16	0.06	= .008	0.17	0.06	= .006
<b>Model 2</b>	0.16	0.06	= .005	0.15	0.06	= .11
<b>Model 3</b>	0.08	0.06	= .16	0.06	0.06	> .25
<u>Longitudinal: BMI Predicting Depressive Symptoms</u>						
	<u>Objective BMI<sup>C</sup></u>			<u>Self-Report BMI<sup>D</sup></u>		
	<i>b</i>	<i>SE</i>	<i>p-value</i>	<i>B</i>	<i>SE</i>	<i>p-value</i>
<b>Model 1</b>	0.23	0.05	< .001	0.24	0.06	< .001
<b>Model 2</b>	0.23	0.05	< .001	0.24	0.06	< .001
<b>Model 3</b>	0.20	0.05	< .001	0.20	0.06	< .001
<u>Longitudinal: Depressive symptoms Predicting BMI</u>						
	<u>Objective BMI<sup>E</sup></u>			<u>Self-Report BMI<sup>F</sup></u>		
	<i>b</i>	<i>SE</i>	<i>p-value</i>	<i>B</i>	<i>SE</i>	<i>p-value</i>
<b>Model 1</b>	0.01	0.01	> .25	-0.01	0.02	> .25
<b>Model 2</b>	0.02	0.01	> .25	< 0.01	0.02	> .25
<b>Model 3</b>	0.02	0.01	= 0.20	< 0.01	0.02	> .25

Model 1 = age, sex, education, and marital status. Outcome variable adjusted at baseline if longitudinal model

Model 2 = Model 1 + recent life events, social isolation, and social activity

Model 3 = Model 2 + chronic illness and physical performance deficits

<sup>A</sup>*n* = 810    <sup>B</sup>*n* = 761    <sup>C</sup>*n* = 582    <sup>D</sup>*n* = 544    <sup>E</sup>*n* = 540    <sup>F</sup>*n* = 495