



## Obesity and onset of depression among U.S. middle-aged and older adults



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### ABSTRACT

**Objectives:** This paper aims to examine the relationship between obesity and onset of depression among U.S. middle-aged and older adults.

**Methods:** Data came from 1994 to 2010 waves of the Health and Retirement Study. Study sample consisted of 6514 community-dwelling adults born between 1931 and 1941 who were free of clinically relevant depressive symptoms in 1994. Body mass index (BMI) was calculated from self-reported height/weight. Body weight status was classified into normal weight ( $18.5 \text{ kg/m}^2 \leq \text{BMI} < 25 \text{ kg/m}^2$ ), overweight ( $25 \text{ kg/m}^2 \leq \text{BMI} < 30 \text{ kg/m}^2$ ), and obesity ( $\text{BMI} \geq 30 \text{ kg/m}^2$ ). A score of  $\geq 3$  on the 8-item Center for Epidemiologic Studies Depression Scale was used to define clinically relevant depressive symptoms. Kaplan–Meier estimator and time-dependent Cox proportional hazards model were performed to examine the association between body weight status and onset of clinically relevant depressive symptoms.

**Results:** Unhealthy body weight was associated future onset of depression. Compared with their normal weight counterparts, overweight and obese participants were 13% (hazard ratio [HR] = 1.13, 95% confidence interval [CI] = 1.04–1.23) and 9% (HR = 1.09, 95% CI = 1.01–1.18) more likely to have onset of clinically relevant depressive symptoms during the 16 years of follow-up, respectively. The relationship between obesity and depression onset appeared stronger among females and non-Hispanic whites than their male and racial/ethnic minority counterparts.

**Conclusions:** Health care providers should be aware of the potential risk for depression among obese older adults.

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### Introduction

Obesity is a leading public health concern in the U.S. and worldwide [1]. Over one-third of American adults are obese and another one-third overweight [2]. The growth rate of adult obesity, especially severe or morbid obesity, does not appear to begin leveling off [3]. While the consequences of obesity on physical health have been well documented [1,4], its link to psychiatric disorders remains unclear. Depression is one of the most prevalent psychiatric disorders and a major contributor to the U.S. burden of disease [5]. Systematic reviews and meta-analyses of cross-sectional studies have suggested an association between obesity and depression [6–8]. One meta-analysis of prospective studies reported obesity at baseline to predict depression onset during follow-up [9], whereas another meta-analysis found no robust evidence [10].

It is not clear to what extent these inconsistencies can be explained by possible heterogeneity in the relationship between obesity and depression across population subgroups [10]. Most prospective, longitudinal studies examining the impact of obesity on depression have focused

on children and youth [11–14]. Few studies have examined the prospective impact of obesity on depression in the aging process [15–18]. Mid-life and older adulthood may be a critical period for the emergence and progression of the psychological risks of obesity. Across the life span, the prevalence of overweight and obesity peaks in midlife (roughly between 40 and 60 years of age) [2]. Midlife is also a period when host immunity begins to decline and the effects of unhealthy body weight and other poor health habits accumulate [19]. The declined immunity may make middle-aged and older adults particularly vulnerable to the psychological consequences of obesity. However, previous studies specifically focusing on middle-aged and older adults used regional samples and had relatively short follow-up period [15–18]. Their assessments of body weight and depression were undertaken at only one point in time [15–17]. In addition, most of these studies estimated odds ratio [15,17], which can dramatically overstate the relative risk for common outcomes [20].

The relationships between obesity and depression may also differ by sex and race/ethnicity. Previous studies have documented a stronger relationship between obesity and depression in women [7,11,21–23]. However, these studies have primarily focused on youth. It is less clear whether similar moderating effects of sex exist in older adulthood [23]. Only a few studies have examined the differential impacts of

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obesity on depression by race/ethnicity with mixed findings reported [16,21,24,25]. Population-based prospective studies are warranted to identify population subgroups that are most vulnerable to the psychological consequences of obesity.

The present study examined the association of obesity and onset of clinically relevant depressive symptoms using data from a nationally representative longitudinal survey of U.S. middle-aged and older adults. The uniquely large and diverse study sample with extended follow-up periods (up to 16 years from 1994 to 2010) allowed us to examine the long-term psychological risks of obesity in the aging process and the differential impacts of obesity across sex and racial/ethnic subgroups.

## Methods

### Participants

Individual-level data came from the Health and Retirement Study (HRS), a nationally representative longitudinal survey of health conditions and health behaviors among middle-aged and older adults in the U.S. The HRS is sponsored by the National Institute on Aging (grant number NIA U01AG009740) and conducted by the University of Michigan. The HRS uses a complex probability sampling design, with supplemental oversamples of African Americans and Hispanics. A total of 12,652 participants (including 9762 age-eligible participants 51 to 61 years of age in 1992 and their spouses) were interviewed at baseline in 1992. Follow-up interviews have been conducted every other year, with over 80% response rates across waves. Detailed information on the survey design, questionnaires, and relevant data are available at the HRS website (<http://hrsonline.isr.umich.edu>).

The present study used 9 waves (1994–2010) of the HRS longitudinal dataset constructed by the RAND Corporation (RAND HRS Version M) [26]. At each wave, survey participants were screened for depression by the 8-item Center for Epidemiologic Studies Depression Scale (CES-D). Data from the 1992 interview were not used because the measure of depression was different from later waves. Among the 8837 age-eligible participants in the initial HRS cohort who were interviewed in 1994, the following participants were excluded from the analyses: CES-D not administered (proxy respondents), 539; missing CES-D score, 3; with clinically relevant depressive symptoms (CES-D score  $\geq 3$ ), 1657; missing body mass index (BMI), 61; and underweight adults (BMI  $< 18.5$ ), 63. The remaining 6514 participants without clinically relevant depressive symptoms in 1994 were included in the analytic sample and followed for an average of 12.4 years (7.2 waves).

### Measure of depression

Depressive symptoms were measured by the 8-item CES-D, a shortened version of the 20-item CES-D [27]. Participants were asked whether the following feelings were present (“yes” or “no”) much of the time during the past week – felt depressed, everything was an effort, sleep was restless, could not get going, felt lonely, felt sad, enjoyed life, and were happy. The CES-D score, ranging from 0 to 8, is the sum of 6 “negative” feelings and absence of 2 “positive” feelings. Melchior et al. [28] reported that the 8-item and 20-item CES-D scales were highly correlated ( $r = 0.93$ ) and had comparable discriminant validity. A cut-off score of 3 has been suggested by previous validation studies to indicate clinically relevant depressive symptoms. This cut-off score has a sensitivity of 0.71 and a specificity of 0.79 to predict major depressive episode(s) [29]. Participants were classified as having onset of clinically relevant depressive symptoms if they had a score of 3 and higher on the 8-item CES-D at any of the follow-up waves.

### Measure of obesity

Body weight status was measured by BMI calculated from self-reported height and weight (weight in kilograms divided by height

in meters squared) at each wave. Body weight status was classified into 3 categories: normal weight ( $18.5 \text{ kg/m}^2 \leq \text{BMI} < 25 \text{ kg/m}^2$ ), overweight ( $25 \text{ kg/m}^2 \leq \text{BMI} < 30 \text{ kg/m}^2$ ), and obesity ( $\text{BMI} \geq 30 \text{ kg/m}^2$ ) [30]. Underweight participants at baseline ( $\text{BMI} < 18.5 \text{ kg/m}^2$ ,  $n = 63$ ) were excluded from the analyses. Sensitivity analyses showed no substantial differences in the results when underweight participants were included.

### Individual characteristics

The following individual characteristics were controlled for in multivariate survival analyses: sex, age in years, race/ethnicity (non-Hispanic white, non-Hispanic African American, non-Hispanic other race/multi-race, and Hispanic), education (education less than high school, high school graduate, some college, and college graduate), marital status (married or partnered, separated or divorced or widowed, and never married), household net wealth (divided into 4 quartiles), smoking status (non-smoker, past smoker, and current smoker), heavy drinking (5 or more drinks per day), physical activity (engagement in vigorous physical activity or sports, such as heavy housework, aerobics, running, swimming, or bicycling 3 or more times per week), index of difficulty in performing activities of daily living (ADLs) constructed by the RAND Corporation [26] (tasks including bathing, eating, dressing, walking across a room, and getting in or out of bed), and self-reported diagnoses of chronic conditions (hypertension, diabetes, heart disease, stroke, cancer, lung disease, and arthritis). These characteristics have been shown to co-vary with body weight status and depression [14,17,24]. All the covariates were measured in 1994. Time-varying covariates were not used in the analyses to avoid over-adjustment bias – controlling for an intermediate variable on a causal path from exposure to outcome could bias estimates for the total exposure effect [31].

### Statistical analyses

Descriptive statistics on baseline individual characteristics were stratified by baseline body weight status. Survival analysis was conducted to examine the relationship between body weight status and onset of clinically relevant depressive symptoms. In survival analysis, survival is defined as absence from clinically relevant depressive symptoms by the end of the study. A participant is considered a survivor till an episode of clinically relevant depressive symptoms (failure) was first reported, if ever. Participants who died, lost during follow-up, or had proxy interviews without ever reporting an episode of clinically relevant depressive symptoms were censored at the last wave when they had a non-proxy interview ( $N = 1722$ ). Participants who were alive and remained free from an episode of clinically relevant depressive symptoms by 2010 were censored at the last wave of the study ( $N = 2334$ ). Kaplan–Meier estimator [32] was used to estimate the unadjusted survival function across survey waves. Time-dependent Cox proportional hazards models [33] were used to estimate the unadjusted and adjusted hazard ratios (HRs) for onset of clinically relevant depressive symptoms at the current wave in relation to time-varying body weight status at the prior wave. Cox models were performed on the overall study sample and subsamples stratified by sex and race/ethnicity (non-Hispanic white, non-Hispanic African American, non-Hispanic other race/multi-race, and Hispanic, whereas non-Hispanic other race/multi-race was excluded due to small sample size). In addition to the subgroup analyses, Cox models with relevant interaction terms (i.e., body weight status \* sex, body weight status \* race/ethnicity) were tested to supplement the results from subgroup analyses.

The HRS sampling frame and individual-level weights at 1994 interview were incorporated in all estimates. Statistical analyses were conducted using Stata 11.1 SE version (StataCorp, TX).

**Results**

Table 1 presents the descriptive statistics of the study sample stratified by baseline body weight status. Participants were on average 57.8 years of age in 1994 and predominantly non-Hispanic white (84.5%). Individual sociodemographics, health behavior, and chronic conditions differed substantially by body weight status. During the 16 years of follow-up, 37% (95% confidence interval [CI] = 35.5%–38.5%, N = 2458) of the initially depression-free sample experienced the onset of clinically relevant depressive symptoms. The rates of depression onset increased monotonically with body weight status, from 34.3% (95% CI = 31.8%–36.8%) among normal weight participants to 37.2% (95% CI = 35.2%–39.2%) among overweight participants and 40.9% (95% CI = 38.6%–43.3%) among obese participants.

Fig. 1 shows the estimated Kaplan–Meier survival curves. Compared with normal weight participants, those who were overweight or obese appeared to have higher risks for developing clinically relevant depressive symptoms in subsequent waves. The unadjusted hazard ratio (HR) for depression incidence was 1.08 (95% CI = 0.99–1.18) among overweight participants and 1.17 (95% CI = 1.07–1.28) among obese participants (see Table 2).

Table 2 presents the adjusted HRs for the onset of clinically relevant depressive symptoms in relation to prior-wave body weight status. Compared with their normal weight counterparts, overweight and obese participants were 13% (HR = 1.13, 95% CI = 1.04–1.23) and 9% (HR = 1.09, 95% CI = 1.01–1.18) more likely to develop clinically relevant depressive symptoms during the 16 years of follow-up, respectively.

Table 3 presents the adjusted HRs from the subgroup analyses by sex and race/ethnicity. Being overweight (HR = 1.20, 95% CI = 1.10–1.31) or obese (HR = 1.18, 95% CI = 1.08–1.30) was significantly associated with future onset of clinically relevant depressive symptoms among females. No significant association between body weight status and depression onset was found among males. The interaction term of obesity and male (HR = 0.81, 95% CI = 0.67–0.97, p = 0.026) was statistically significant. Being overweight (HR = 1.17, 95% CI = 1.07–1.29) or obese (HR = 1.11, 95% CI = 1.02–1.21) was significantly associated with future onset of clinically relevant depressive symptoms among non-Hispanic whites.

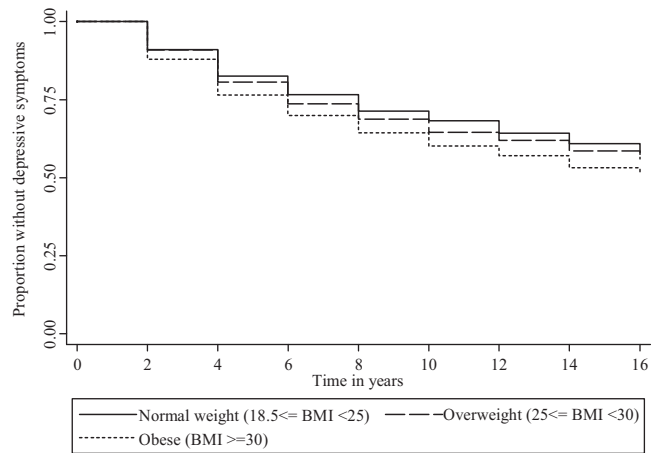


Fig. 1. Unadjusted Kaplan–Meier survival curves of time to onset of clinically relevant depressive symptoms by body weight status. Each data point denotes the percentage of participants who had not developed clinically relevant depressive symptoms at a given time.

No significant association between body weight status and depression onset was found among non-Hispanic African Americans or Hispanics. The interaction term of being overweight and African American was statistically significant (HR = 0.72, 95% CI = 0.57–0.92, p = 0.008).

**Table 1**  
Sample characteristics by baseline body weight status.

Baseline characteristics	Normal weight	Overweight	Obesity
Sample size	2214	2716	1584
Proportion in population (%)	35.3 (33.6, 37.1)	41.7 (40.5, 43.0)	23.0 (21.7, 24.3)
Age in years	57.8 (57.7, 58.0)	57.8 (57.6, 57.9)	57.8 (57.7, 57.9)
<b>Sex (%)</b>			
Female	62.7 (60.5, 64.8)	42.2 (40.5, 43.9)	52.3 (49.3, 55.4)
Male	37.3 (35.2, 39.5)	57.8 (56.1, 59.5)	47.7 (44.6, 50.7)
<b>Race/ethnicity (%)</b>			
White, Non-Hispanic	88.5 (84.1, 91.8)	84.3 (78.7, 88.6)	78.6 (70.8, 84.9)
African American, Non-Hispanic	5.5 (3.3, 9.0)	8.2 (5.1, 12.9)	13.7 (8.8, 20.7)
Other race/multi-race, Non-Hispanic	2.2 (1.4, 3.5)	1.7 (1.1, 2.5)	1.0 (0.6, 1.5)
Hispanic	3.8 (2.3, 6.1)	5.9 (4.0, 8.5)	6.8 (4.4, 10.2)
<b>Education attainment (%)</b>			
Less than high school	15.3 (13.0, 17.9)	17.4 (15.3, 19.8)	22.5 (19.4, 25.9)
High school	36.2 (33.1, 39.4)	39.8 (27.1, 42.7)	41.7 (39.0, 44.5)
Some college	23.6 (21.4, 26.0)	20.9 (19.2, 22.7)	19.9 (17.8, 22.2)
College graduate and higher	24.9 (21.4, 28.6)	21.9 (19.4, 24.6)	15.9 (13.6, 18.7)
<b>Marital status (%)</b>			
Married/partnered	75.8 (73.6, 77.9)	82.0 (80.0, 83.8)	77.8 (75.1, 79.9)
Divorced/separated/widowed	20.6 (18.5, 22.8)	14.7 (13.1, 16.4)	18.9 (17.0, 21.0)
Never married	3.6 (2.7, 4.8)	3.4 (2.7, 4.2)	3.5 (2.6, 4.7)
Household net worth (\$)	334,041 (296,789, 371,293)	286,811 (249,022, 324,600)	244,456 (208,880, 280,032)
<b>Smoking status (%)</b>			
Non-smoker	37.2 (35.3, 39.1)	36.5 (34.5, 38.5)	39.1 (36.6, 41.2)
Past smoker	34.6 (32.5, 36.8)	43.9 (41.4, 46.5)	46.1 (43.4, 48.8)
Current smoker	28.2 (26.8, 30.1)	19.6 (18.2, 21.0)	14.8 (13.4, 16.3)
5 or more drinks/day (%)	0.7 (0.5, 1.1)	1.00 (0.6, 1.5)	0.9 (0.5, 1.3)
Exercise 3 or more times/week (%)	28.7 (26.2, 31.3)	23.3 (21.4, 25.2)	16.3 (14.5, 18.3)
Difficulty in activities of daily living (%)	2.4 (1.6, 3.0)	2.9 (2.3, 3.6)	4.5 (3.4, 5.9)
<b>Chronic Disease (%)</b>			
Hypertension	21.4 (19.5, 23.4)	34.4 (32.3, 36.3)	48.5 (45.2, 51.8)
Diabetes	3.8 (2.9, 5.0)	7.5 (6.3, 8.8)	15.8 (14.1, 17.7)
Stroke	2.1 (1.5, 2.8)	2.1 (1.6, 2.7)	2.1 (1.4, 3.1)
Heart disease	8.3 (7.3, 9.5)	9.5 (8.5, 10.5)	13.2 (11.3, 15.3)
Cancer	6.4 (5.6, 7.4)	5.1 (4.3, 6.1)	5.0 (3.9, 6.2)
Lung disease	5.1 (4.3, 6.1)	3.9 (3.3, 4.6)	4.7 (3.7, 5.9)
Arthritis	29.8 (28.0, 31.6)	34.3 (32.2, 36.4)	45.4 (43.1, 47.8)

Note. 95% confidence intervals in parentheses.

**Table 2**  
Onset of clinically relevant depressive symptoms in relation to body weight status in middle-aged and older adults.

Independent variable	# of persons	# of person-years observations	Unadjusted HR (95% CI)	Adjusted HR (95% CI)
<i>Body weight status</i>				
Normal weight	2214	9168	Reference	Reference
Overweight	2716	12,418	1.08 (0.99, 1.18)	1.13 (1.04, 1.23)**
Obesity	1584	7745	1.17 (1.07, 1.28)**	1.09 (1.01, 1.18)*
<i>Age in years</i>				
Male				0.99 (0.98, 1.00)*
				0.71 (0.65, 0.77)***
<i>Race/ethnicity</i>				
White, non-Hispanic				Reference
African American, non-Hispanic				1.03 (0.94, 1.12)
Other race/multi-race, non-Hispanic				1.33 (1.02, 1.73)*
Hispanic				1.20 (1.08, 1.32)**
<i>Education attainment</i>				
Less than high school				Reference
High school				0.89 (0.83, 0.96)**
Some college				0.85 (0.78, 0.93)**
College graduate and higher				0.73 (0.64, 0.84)***
<i>Marital status</i>				
Married/partnered				Reference
Divorced/separated/widowed				1.08 (1.00, 1.17)
Never married				0.94 (0.77, 1.15)
<i>Household net worth</i>				
1st quartile				Reference
2nd quartile				0.91 (0.83, 0.99)*
3rd quartile				0.82 (0.74, 0.90)***
4th quartile				0.87 (0.78, 0.97)*
<i>Smoking status</i>				
Non-smoker				Reference
Past smoker				1.08 (0.99, 1.18)
Current smoker				1.29 (1.18, 1.42)***
5 or more drinks/day				1.51 (1.19, 1.92)**
Exercise 3 or more times/week				0.90 (0.83, 0.98)*
Had difficulty in activities of daily living				1.18 (1.05, 1.32)**
<i>Chronic conditions</i>				
Hypertension				1.05 (0.99, 1.12)
Diabetes				1.08 (0.98, 1.19)
Stroke				0.98 (0.80, 1.19)
Heart disease				1.14 (1.04, 1.25)**
Cancer				0.93 (0.79, 1.10)
Lung disease				1.10 (0.97, 1.25)
Arthritis				1.28 (1.20, 1.36)***

Note. HR = hazard ratio; CI = confidence interval.

- \* p < .05.
- \*\* p < .01.
- \*\*\* p < .001.

**Discussion**

The present study examined the onset of clinically relevant depressive symptoms in relation to body weight status among a

nationally representative sample of middle-aged and older adults. Being overweight or obese significantly predicted future onset of depressive symptoms. The relationship between unhealthy weight status and depression appeared stronger among females

**Table 3**  
Onset of clinically relevant depressive symptoms in relation to body weight status among middle-aged and older adults, by sex and race/ethnicity.

	Body weight status									
	Normal weight		Overweight				Obesity			
	# of persons	# of person-years	# of persons	# of person-years	Unadjusted HR (95% CI)	Adjusted HR (95% CI)	# of persons	# of person-years	Unadjusted HR (95% CI)	Adjusted HR (95% CI)
<i>Sex</i>										
Female	1388	5805	1202	5518	1.27 (1.16, 1.40)*	1.20 (1.10, 1.31)*	861	3929	1.34 (1.22, 1.47)*	1.18 (1.08, 1.30)*
Male	826	3363	1514	6900	1.00 (0.84, 1.18)	1.01 (0.86, 1.20)	723	3816	1.00 (0.84, 1.18)	0.96 (0.82, 1.12)
<i>Race/ethnicity</i>										
White, non-Hispanic	1832	7929	2079	10,008	1.09 (0.99, 1.19)	1.17 (1.07, 1.29)*	1073	5653	1.16 (1.05, 1.27)*	1.11 (1.02, 1.21)*
African American, non-Hispanic	220	688	397	1521	0.82 (0.66, 1.03)	0.84 (0.68, 1.04)	360	1517	1.01 (0.82, 1.24)	0.93 (0.76, 1.14)
Hispanic	117	399	205	731	1.05 (0.77, 1.44)	1.06 (0.78, 1.41)	135	515	1.04 (0.71, 1.51)	1.07 (0.73, 1.56)

Note. Normal weight status is the reference group when estimating the hazard ratios (HR); CI = confidence interval.

- \* p < .05.

and non-Hispanic whites than their male and racial/ethnic minority counterparts.

The study finding of the association between unhealthy weight and depression is consistent with previous longitudinal studies focusing on middle-aged and older adults [15–18]. Using data from the Nurse's Health Study, Pan et al. [17] reported baseline obesity to be associated with a 10% increase in the odds of physician-diagnosed depression and/or antidepressant use in a sample of middle-aged and elderly women. In a community sample of participants 50 years of age and over recruited from Alameda County, California, Roberts et al. [15] found baseline obesity to be associated with a two-fold increase in the odds of a major depressive episode 5 years later. Studies using self-reported depressive symptoms have documented similar findings [16, 34]. In addition, emerging evidence from intervention studies has suggested possible causal link between obesity and depression. Dixon et al. [22] found that weight loss after bariatric surgery was associated with a significant decline in depression scores at year 1 and again at year 4 after the surgery. Kloiber et al. [35] reported that overweight and obese patients with major depressive disorder showed slower clinical response to depression treatment compared with their normal weight counterparts.

Obesity may cause depressive symptoms through several biological, social, and cognitive pathways. Dysregulation of the HPA axis is found among obese and depressed subjects and both anti-obesity drugs and antidepressants regulate the HPA axis [36], suggesting potential pathway between obesity and depression through the stress-response system. Pervanidou et al. [37] provided preliminary evidence on the stress-response system pathway. They found increased cortisol concentrations among obese children with comorbid depression or anxiety compared with obese children without any affective disorders. Obesity also increases the risks for heart disease, stroke and functional impairment, and decreases health-related quality of life, which may trigger depressive symptoms [38]. In addition, obese individuals often encounter repeated discrimination, which can lead to negative affect [39]. In a national sample of English-speaking adults aged 25–74 years old, Carr and Friedman [40] found severely obese individuals were significantly more likely to report being treated rudely by strangers, acquaintances, and professionals compared with their normal weight counterparts. Obesity may also cause body image dissatisfaction, resulting in lower self-esteem and subsequently depressive symptoms [38]. Friedman et al. [41] found body-image satisfaction partially mediated the relationship between obesity severity and depression for both women and men at a residential weight control facility.

In the present study, being overweight had comparable impact on the onset of depressive symptoms relative to being obese. Previous studies have documented a strong dose–response relationship between BMI and adverse physical health outcomes [42,43]. It is possible that the psychological risks of obesity may not be linear. It is also possible that our finding is a result of a survival effect in an elderly sample. As individuals who are obese are more likely to die at younger ages, obese individuals who live through their middle age may have survival genes that protect them from depression.

This study found a stronger relationship between obesity and depressive symptoms in women. This finding is consistent with several cross-sectional [21,23,44] and prospective studies [11,22,45]. A systematic review of primarily cross-sectional studies supported the association between obesity and depression outcomes for women but not men [6]; however, these results were from synthesis of effect sizes and were not formally tested for statistical significance. In a meta-analysis of prospective studies, Rooke and Thorsteinsson [10] did not find any significant moderators in the relationship between baseline body weight status and depression at follow-up, possibly due to the small main effect. They did, however, found significantly stronger association between depression and weight gain among females. The reasons for the sex differences in the association between body weight status and depression remain unclear. One hypothesis is that women

have greater internalized stigmatization of obesity than men, which could lead to lower self-esteem and more severe depressive symptoms [46,47].

In the present study, unhealthy body weight status had greater association with the onset of clinically relevant depressive symptoms among non-Hispanic whites compared with their racial/ethnic minority counterparts. Only a few studies have examined the differential effects of obesity on depression by race/ethnicity with mixed findings reported [16,21,24,25]. Simon et al. [24] found a stronger association between obesity and depression among non-Hispanic whites in a nationally representative sample of U.S. adults. Faith et al. [25] analyzed data from 4 nationally representative surveys and did not find any significant relationship between body weight and self-esteem among African Americans. In a cross-sectional study of national data, no racial differences were found on the association between obesity and major depression, suicide attempts, and suicide ideation among adults [21]. In contrast, Sachs-Ericsson et al. [16] reported a stronger influence of BMI on depressive symptoms among African Americans in a longitudinal survey of older adults residing in North Carolina. The inconsistencies in study findings could be due to differences in study samples (e.g., different age groups), design and methods, follow-up durations, and measures of depression. The reasons for potential racial differences in the relationship of unhealthy weight and depression are unclear. Racial differences in body image and perceptions of healthy body weight have been reported [47,48]. Compared with whites, African Americans seem to have a more positive attitude toward obesity [47] and are more likely to accept their weight at higher BMIs [48]. The racial differences in body weight perceptions could help explain the differential effects of obesity on depression by race/ethnicity found in the present study. Additional research with diverse populations is warranted to better understand the sociodemographic ingredients in the relationship between obesity and depression.

Various factors such as functional limitations, compromised immune system, comorbidity, and social isolation make older adults particularly vulnerable to the health consequences of depression [49]. The present study suggests that having an unhealthy body weight may aggravate the burden of depression in older age. The burden of obesity on psychological wellbeing is likely to increase substantially over the next several decades as the older population rapidly grows [50]. Although heightened attention has been given to obesity reduction, few programs specifically target older adults. Developing effective obesity intervention strategies among middle-aged and older adults may have added benefits of reducing the incidence of depression. Increasing access to depression screening and treatment among older adults is equally important. Depression among older adults is frequently missed, improperly diagnosed, and inadequately treated [51], possibly as a result of stigma associated with depression [52] and perceptions of depression as normal part of aging [53]. Obesity has been associated with cognitive decline [54] and functional limitations [55], posing additional challenges to diagnosing depression among obese older adults. However, current guideline from the U.S. Preventive Services Task Force [56] on screening for depression does not consider obesity as a risk factor and/or comorbidity of depression. Given the barriers in depression screening in relation to obesity, health care providers should be aware of the potential risk for depression among obese older adults and monitor their mood status.

A few limitations of this study should be noted. Modeling results based on data from prospective observational studies should be interpreted as correlations rather than causations. CES-D measures the number of self-reported depressive symptoms in the past week, which can vary greatly from week to week. The 8-item CES-D cutoff score had a sensitivity of 0.71 and a specificity of 0.79 to predict a clinically diagnosable major depressive episode [29]. This means that about 30% of people with a clinically diagnosable major depressive episode will be missed whereas about 20% of people classified as having depression in the present study will not meet the criteria for major

depression. The relatively lower sensitivity and specificity introduce measurement errors and may result in underestimation of the association between obesity and depression. Body weight status was based on BMI calculated from self-reported weight and height, which is subject to reporting error and biases particularly among older adults [57]. BMI does not distinguish between body fat and lean body mass, and it is not as accurate a predictor of body fat in older adults as it is in their younger and middle-aged counterparts [58]. By excluding participants with current depression at the baseline, this study analyzed the relationship between obesity and depression among community-dwelling older adults who were relatively healthier and less fragile at baseline compared with the average of their age-cohorts in the population and their institutionalized counterparts. In addition, attrition is an important concern in longitudinal aging studies. Banks, Muriel, and Smith [59] found household net wealth to be negatively correlated with attrition rates in the HRS. Because wealthier elders are less likely to develop depression [60], attrition attributable to wealth could result in underestimation of the relationship between obesity and depression.

In conclusion, being overweight or obese predicted onset of clinically relevant depressive symptoms among middle-aged and older adults up to 16 years of follow-up. The impact of unhealthy body weight status appeared to be stronger among females and non-Hispanic whites. Effective obesity interventions targeting middle-aged and older adults may have added benefits of reducing the incidence of geriatric depression. Health care providers should closely monitor mood status in overweight and obese older adults. Future studies are warranted to confirm these findings using different measures of adiposity and explore potential mechanisms linking obesity with depression.

### Conflict of interest statement

We wish to confirm that there are no known conflicts of interest associated with this publication and there has been no significant financial support for this work that could have influenced its outcome.

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