



## 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).

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