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# Body weight status and onset of cognitive impairment among U.S. middle-aged and older adults

Xiaoling Xiang [a,*], Ruopeng An [b,1]

[a] School of Social Work, University of Illinois at Urbana-Champaign, 1010 West Nevada Street, Urbana, IL 61801, United States
[b] Department of Kinesiology and Community Health, College of Applied Health Sciences, University of Illinois at Urbana-Champaign, 2013 Huff Hall, 1206 South 4th Street, Champaign, IL 61820, United States

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ABSTRACT

*Objective:* To examine the relationship between body weight status and onset of cognitive impairment among U.S. middle-aged and older adults.
*Methods:* Study sample came from 1996 to 2010 waves of the Health and Retirement Study, consisting of 6739 community-dwelling adults born between 1931 and 1941 who were free from cognitive impairment in 1996. Body mass index (BMI) was calculated from self-reported height/weight. Cognitive impairment was defined by a composite score of 11 or lower on the immediate and delayed word recall, serial 7's, and backwards counting tests. Kaplan–Meier estimator and Cox proportional hazards model were performed to examine the association between base-year body weight status and future onset of cognitive impairment.
*Results:* Compared with their normal weight counterparts, the unadjusted hazard ratio (HR) for cognitive impairment incidence was 2.03 (95% confidence interval: 1.38–3.00), 1.15 (1.02–1.29), 1.28 (1.14–1.43), and 1.59 (1.33–1.92) among underweight (BMI $< 18.5$), overweight ($25 \leq$ BMI $< 30$), class I obese ($30 \leq$ BMI $< 35$), and class II obese or above (BMI $\geq 35$) participants, respectively. The unadjusted relationship between obesity and cognitive impairment onset appeared stronger among females than among males. After adjusting for base-year individual sociodemographics, functional limitations and chronic conditions, the estimated associations between body weight status and cognitive impairment were attenuated but remained statistically significant for underweight participants.
*Conclusion:* Underweight is a robust risk factor for onset of cognitive impairment in later life. Weight management programs targeting middle-aged and older adults should focus on achieving and maintaining optimal body weight.



## 1. Introduction

Dementia is a major cause of disability in old age and associated with substantial societal costs. The estimated total costs of dementia worldwide were $604 billion in 2010, including $251 billion on informal care (Wimo et al., 2013). Even milder forms of cognitive impairment that do not meet the threshold of dementia can noticeably decrease quality of life (Nys et al., 2006), increase the incidence of functional limitations (Tabert et al., 2002), and drive up demands and costs for health care (Albert, Glied, Andrews, Stern, & Mayeux, 2002; Comas-Herrera, Wittenberg, Pickard, & Knapp, 2007). The adjusted annual conversion rate from mild cognitive impairment to dementia is 5–10%; and up to 40% of mild cognitive impairment cases eventually progress to dementia (Mitchell & Shiri-Feshki, 2009). The number of people with dementia is expected to increase as the demographic landscape of the nation rapidly transitions into an aging population (Ortman, Velkoff, & Hogan, 2014).

Recent observations suggest obesity to be a risk factor for dementia (Anstey, von Sanden, Sargent-Cox, & Luszcz, 2007; Beydoun, Beydoun, & Wang, 2008). Several hypotheses on the mechanisms linking obesity with cognitive impairment have been proposed, including shared genetic vulnerability, low-grade systemic inflammation, elevated lipids, and insulin resistance (Smith, Hay, Campbell, & Trollor, 2011). One study showed that a fat mass and obesity associated gene was related to impaired cognition (Keller et al., 2011), indicating possible shared genetic

* Corresponding author. Tel.: +1 217 419 6693; fax: +1 217 244 5220.
*E-mail address:* xxiang3@illinois.edu (X. Xiang).
[1] Tel.: +1 217 244 0966; fax: +1 217 333 2766.

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vulnerability between these conditions. Obesity can increase systemic inflammation, which has been linked to cognitive impairment (Trollor et al., 2010). Excessive body weight is associated with insulin resistance, which has been observed among people with dementia (Biessels, Kappelle, & Utrecht Diabetic Encephalopathy Study Group, 2005; Vanhanen et al., 1998). Both animal studies (Greenwood & Winocur, 1996; Molteni, Barnard, Ying, Roberts, & Gomez-Pinilla, 2002) and studies on humans (Edwards et al., 2011; Greenwood & Winocur, 2005) have demonstrated the adverse impact of diets high in saturated fat on cognitive function. The prevalence of adult obesity especially severe or morbid obesity has increased substantially over the past three decades (An, 2014). If obesity is a risk factor for cognitive impairment, the sweeping obesity epidemic could further fuel the dementia epidemic. Therefore, examining the relationship between obesity and cognitive impairment may carry important implications for determining the future burden of cognitive impairment and developing effective prevention programs.

Although there appears to be some correlation between obesity and risk for dementia in middle and older adulthood (Anstey et al., 2007; Beydoun et al., 2008), it remains unclear whether obesity in later life is associated with the onset of cognitive impairment, the pre-dementia stage. Existing work pertaining to the relationship between obesity and cognitive impairment primarily comprises cross-sectional studies (Dore, Elias, Robbins, Budge, & Elias, 2008; Kuo et al., 2006; Nilsson & Nilsson, 2009; Ward, Carlsson, Trivedi, Sager, & Johnson, 2005). Only a handful of studies examined the impact of obesity on cognition among older adults adopting a prospective, longitudinal design (Elias, Elias, Sullivan, Wolf, & D'Agostino, 2005; Han et al., 2009; Sturman et al., 2008). Findings from both types of studies are largely mixed and inconclusive (Smith et al., 2011). For example, one study found that waist circumference and waist–hip ratio (WHR) were inversely related to multiple cognitive domains among a sample of stroke and dementia-free community-dwelling adults (Dore et al., 2008), whereas another cross-sectional study reported overweight and obese adults aged 65–94 to have better cognitive performance compared with their normal weight counterparts (Kuo et al., 2006). One prospective study showed adverse effects of obesity on cognitive performance among participants of the Framingham Heart Study (Elias et al., 2005). In contrast, another prospective study found obesity to be associated with a lower level of decline in cognitive function in a biracial sample of adults aged 65 and older from south Chicago (Sturman et al., 2008). Population-based prospective studies are warranted to elucidate the relationship between body weight and cognitive function in old age.

It is not clear to what extent the mixed findings from previous studies have resulted from heterogeneities in the relationship between obesity and cognition across population subgroups (Elias et al., 2005; Han et al., 2009). Han et al. (2009) reported that an increase in adiposity as assessed by BMI, WHR, and waist circumference was associated with improved cognitive functioning in a two-year period among baseline obese men 60–85 years of age. This relationship was reversed among female participants, where an increase in obesity was associated with significant cognitive decline. The reasons are unknown but one plausible hypothesis is that obese men retain more testosterone in their body fat which in turn helps prevent cognitive impairment possibly via conversion to estrogen (Smith et al., 2011). Additional research is needed to identify population subgroups that are most vulnerable to the cognitive consequences of obesity.

The primary objective of this study was to examine the relationship of base-year body weight status (i.e., underweight, normal weight, and obesity) in relation to future onset of cognitive impairment in a nationally representative sample of U.S. middle-aged and older adults. We hypothesized that body weight status were positively associated with the risk of cognitive impairment. The uniquely large and diverse study sample with extended follow-up periods (14 years from 1996 to 2010) permitted assessment of the long-term impact of body weight on cognitive decline in the aging process. The secondary objective was to investigate whether the association of body weight status and cognitive function differed by sex and race/ethnicity.

## 2. Subjects and methods

### 2.1. Participants

Individual-level data came from the Health and Retirement Study (HRS), a nationally representative longitudinal survey of health conditions, health behaviors, employment, and retirement 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. HRS uses a complex, multistage, probability sampling design, with supplemental oversamples of African Americans and Hispanics. The methodology of the HRS involves using "proxy" respondents when the original respondent could not complete the interview, often due to illness, cognitive impairment, or other reasons. A total of 9762 age-eligible participants (51 years or older in 1992) and their spouses were interviewed during the first wave of the HRS in 1992. Follow-up interviews have been conducted every other year since, with overall response rates over 80% across waves. Detailed information on the HRS survey design, questionnaires, and relevant data is available on its website (http://hrsonline.isr.umich.edu).

This study used data from 8 waves of the HRS during 1996–2010. Data from 1992–1994 interviews were not used because the cognition measures used in these 2 waves were different from later waves. Among the 8327 age-eligible participants in the initial HRS cohort who were interviewed in 1996, the following participants were excluded from the analyses: proxy respondents, 473; currently with cognitive impairment (cognition score of 11 or lower), 1028; and missing height/weight, 87. The remaining 6739 participants born between 1931 and 1941 free of cognitive impairment in 1996 were included in the analytical sample and followed for an average of 13.2 years (6.6 waves).

### 2.2. Measure of cognition

The HRS cognitive measures were developed based on the Telephone Interview for Cognitive Status (TICS) (Brandt, Spencer, & Folstein, 1988), a validated cognitive screening instrument, and its modifications (Breitner et al., 1995). Following Langa, Kabeto, and Weir (2009), this study created a composite score of cognitive functioning using items of immediate and delayed word recall, a serial 7's, and backwards counting. During the immediate recall task, the interviewer read a list of 10 nouns (e.g., lake, car, army, etc.) to the respondent, and asked the respondent to recall as many words as possible from the list in any order. In the delayed recall task, the respondent was asked to recall the nouns previously presented as part of the immediate recall task after answering other survey questions. To reduce learning effects (i.e., improvement in cognitive tests over time due to experience with the tasks), several lists that contained different (but equivalent in difficulty level) nouns were constructed and administered across study waves. A total word recall score, ranging from 0 to 20, was calculated by summing up the number of words the respondent recalled correctly. In the serial 7's test, the respondent was asked to subtract 7 from 100, and continue subtracting 7 from each subsequent number for a total of 5 trials. The respondent was responsible for remembering the value from the prior subtraction.