THE GERONTOLOGICAL SOCIETY OF AMERICA®

Journals of Gerontology: Medical Sciences
cite as: *J Gerontol A Biol Sci Med Sci*, 2018, Vol. 73, No. 2, 198–203
doi:10.1093/gerona/glx152
Advance Access publication August 7, 2017

OXFORD

Research Article

Body Mass Index and Decline in Cognitive Function in Older Black and White Persons

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Received: March 6, 2017; Editorial Decision Date: July 28, 2017

Decision Editor: Anne Newman, MD, MPH

Abstract

Background: While body mass index (BMI) is higher in black compared to white persons, little is known about BMI and change in cognition in cohorts with a large proportion of blacks. We examine relations of BMI with decline in global cognition and five cognitive domains, in older blacks and whites, and determine whether relations differ by race.

Methods: Participants were 2,134 persons without baseline dementia (33% black; 75% women; mean age =77.9 [range 53–100] and education = 14.7 years, Mini-Mental State Examination = 28.0), enrolled in one of two longitudinal, community-based cohort studies of aging (Minority Aging Research Study; Rush Memory and Aging Project). Summary scores of global cognition and five domains were based on 19 neuropsychological tests administered annually. Mixed-effects models, controlling for age, sex, education, and race, were used to examine the relation of baseline BMI to change in cognition.

Results: Baseline BMI = 28.4 units (30.3 in blacks [95% confidence interval (CI): 27.2–27.7]; 27.4 in whites [95% CI: 29.8–30.7]). During a mean annual follow-up of 6 years (*SD* = 4), lower baseline BMI was related to faster decline in global cognition ($p = .002$), and semantic memory ($p < .001$) and episodic memory ($p = .004$), but not working memory, perceptual speed, or visuospatial ability (all $p > .08$). The relationship of BMI with change in cognition was not modified by race (all $p > .09$).

Conclusions: Late-life lower BMI relates to faster rates of decline in cognition, specifically semantic memory and episodic memory, in both blacks and whites. The effect of BMI on cognition appears to be similar in both racial groups.

Keywords: Body mass index, Cognitive decline, Cohort, Memory, Race

Identifying risk factors for cognitive decline in older adults is a high priority in medical research and practice, particularly for risk factors that are potentially modifiable. Body mass index (BMI) is a biometric measure largely determined by weight in three tissues, muscle, adipose, and bone, and when high or low, is associated with poor health outcomes, including death (1,2). BMI is modifiable by several means, including diet and exercise. Several studies have examined the relationship of late-life BMI to cognition in aging, with many but not all suggesting that low BMI is associated with cognitive impairment and decline, and is related to incident mild cognitive impairment and dementia (3–5). However, few studies have directly addressed race in the relation of late-life BMI to cognition, and results have been mixed (6,7). Given the substantial racial difference in BMI with blacks having a higher prevalence of overweight and obese individuals (8), and stronger effects of BMI on various conditions including diabetes (9,10), metabolic syndrome (11), and hypertension, it is surprising that the relation of BMI to cognitive decline in this population, and whether the relation differs from that in whites, has received little attention.

Here, we used data from two longitudinal, community-based cohort studies of aging in the Chicagoland area, the Minority Aging Research Study and the Rush Memory and Aging Project, with data in more than 2,100 older black or white persons, to examine whether late-life BMI is related to change in cognition, including in global cognition and in five different cognitive domains. We also examined whether this association differed between blacks and

whites, and considered potential effects of age and medical conditions on relations.

Methods

Cohort Studies and Participants

Participants for this study were enrolled in one of two longitudinal, community-based cohort studies of aging and cognition, the Rush Minority Aging Research Study (12) or the Rush Memory and Aging Project (13) (see Supplementary Material). The Rush Institutional Review Board of Rush University Medical Center approved both studies.

Because the Minority Aging Research Study was designed to have essentially identical recruitment techniques and a large overlap of identical data collection with the Memory and Aging Project (including cognitive testing), combining data from both cohorts allows the conduct of studies on racial differences in health and aging in a large group of older blacks and whites. The follow-up rate is 91% in the Minority Aging Research Study and 86% in Memory and Aging Project.

Clinical Evaluations

Participants underwent annual uniform, structured clinical evaluations, which included a detailed medical history, physical examination (including data from which to calculate BMI), and neuropsychological testing (see below). Each participant was asked to self-report their racial category, based on questions used by the U.S. Census Bureau, as previously reported (14). After review of all clinical data, a clinician with expertise in the evaluation of persons with dementia classified each participant with respect to dementia status (13). Data on diabetes and medical conditions were available (13,14). Annual follow-up clinical evaluations were identical to the baseline evaluation in all essential components.

Assessment of BMI

Height and weight were measured and recorded annually. For each individual, BMI at baseline was calculated by dividing baseline weight (kg) by height (median of repeated measurements, m^2). For analyses, the BMI at baseline variable was centered at the mean (mean = 28). Analyses were also done with longitudinal BMI data (see Supplementary Material).

Assessment of Cognitive Function

A battery of neuropsychological tests was selected to assess a broad range of cognitive abilities in aging, as previously described (16). The Minority Aging Research Study and the Memory and Aging Project have 20 neuropsychological tests in common (17). Nineteen neuropsychological tests were grouped to form composite measures of global cognition and five cognitive domains (see Supplementary Material). To create each composite score, including the global cognition and individual domains, individual tests were converted to *z* scores, using the baseline mean and standard deviation from the entire cohort, and *z* scores for all tests in a given domain were averaged. Further information about the individual tests and composite scores is published elsewhere (16–18).

Data Analyses

We first used mixed-effects models to examine associations of baseline BMI with change in global cognition and each of the five cognitive domain scores (five outcome measures: semantic memory, episodic memory, working memory, perceptual speed, and visuospatial ability). Each model included terms for age, sex, education, race, BMI at baseline, and their interaction with time. Terms for age, education, and BMI at baseline were centered, based on data from the combined subgroups. Data were truncated for analyses, to have a similar number of follow-up observations for whites and blacks. We also examined the relation of longitudinal BMI (range) with change in cognition (see Supplementary Material). In additional models, we then tested for racial differences in relations of BMI to change in cognition, by adding interaction terms of Race × BMI at baseline and Time × Race × BMI at baseline to the six core models. In order to examine for factors with a potential for affecting the relation of BMI to cognitive decline, we also conducted analyses controlling for baseline diabetes and, separately, medical conditions. Analytic programming was done in SAS version 9.4 (SAS Institute Inc, Cary, NC). Models were validated graphically and analytically.

Results

Participants

Analyses included a total of 2,134 older persons, of whom 704 (33%) were black (from either of the two cohorts) and 1,430 (67%) were white (all from the Memory and Aging Project). The mean number of annual visits was 5.9 (*SD* = 3.8) visits for blacks and 6.5 (*SD* = 4.0) for whites. Clinical characteristics of the total group, and of blacks and whites separately, are shown in Table 1. BMI was increased in black compared to white persons (30.3 in blacks [95% CI: 27.2–27.7]; 27.4 in whites [95% CI: 29.8–30.7]; $t(2,132) = -11.21$, $p < .001$), in keeping with published data (8).

BMI and Decline in Cognitive Function

We constructed a mixed-effects model, controlling for age, sex, education, and race, to examine the relationship of baseline BMI to change in cognitive function in global cognition, in the total group of participants (blacks and whites, grouped together). The model was repeated for each of the five cognitive domains (Bonferroni corrected α of 0.01). As shown in Table 2, persons with a lower BMI had a faster rate of decline in the measure of global cognition (see Figure 1). We also found that lower BMI was related to a faster decline in both semantic memory and episodic memory domains (Table 2). Data suggest there may be a relation of lower BMI to a faster rate of decline in working memory, although this was not significant ($p = .087$), and data showed no relation of BMI with change in perceptual speed or visuospatial ability.

Table 1. Baseline Clinical Characteristics of Participants

Characteristics*	Blacks, *n* = 704	Whites, *n* = 1,430	Total, *n* = 2,134
Age, years	73.5 (6.6)	80.1 (7.2)	77.9 (7.7)
Female sex, *n* (%)	542 (77%)	1,065 (74%)	1,607 (75%)
Education, years	14.7 (3.4)	14.8 (3.2)	14.7 (3.3)
MMSE, median (IQR)**	28 (27, 29)	29 (27, 29)	28 (27, 29)
Global cognitive score	-0.055 (0.519)	0.156 (0.554)	0.087 (0.552)
Diabetes, *n* (%)	189 (27%)	162 (11%)	351 (16%)
BMI***	30.3 (6.3)	27.4 (5.0)	28.4 (5.6)

Note: *Values are mean (*SD*), unless otherwise specified. **MMSE = Mini-Mental State Examination, IQR = Interquartile range. ***BMI = Body mass index.

Table 2. Relationship of BMI at Baseline to Change in the Global Cognitive Score and Five Cognitive Domains, in 2,134 Participants*

Outcome	Estimate (*SE*), *p***
Global score	0.0015 (0.0005), .002
Domains***	
Semantic memory	0.0023 (0.0006), <.001
Episodic memory	0.0016 (0.0006), .004
Working memory	0.0008 (0.0005), .087
Perceptual speed	0.0006 (0.0004), .217
Visuospatial ability	0.0006 (0.0004), .172

Note: BMI = Body mass index.

*Six separate linear mixed-effects models, each adjusted for age, sex, education, and race. **Estimated effect of Time × BMI at baseline. ***Bonferroni corrected α = 0.01.

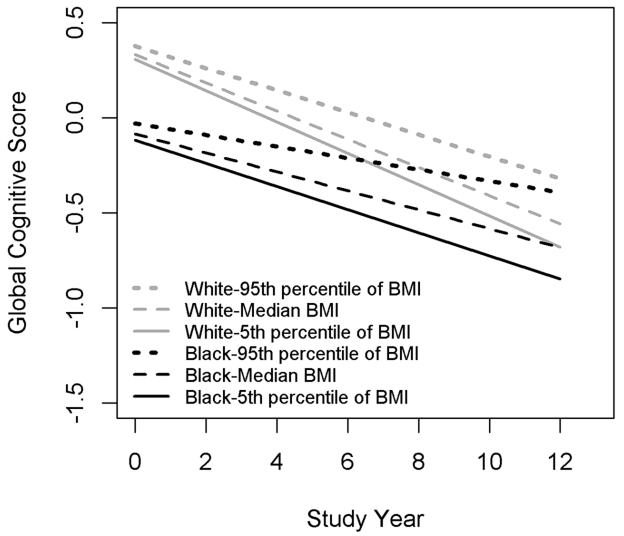

Figure 1. Estimated relationship of BMI at baseline to change in global cognitive function, for black and white persons at the 5th, 50th, and 95th percentiles of BMI. The 5th, 50th, and 95th percentiles of BMI are respectively 21.87, 29.34, 42.32 for blacks and 21.08, 26.58, 37.28 for whites. BMI = Body mass index.

We assessed whether the effect of BMI on change in cognitive function was modified by age, by adding interaction terms of Age × BMI and Time × Age × BMI. We found no interactions ($p > .31$), suggesting that the effect of BMI on rate of decline in global cognition, or semantic memory or episodic memory, does not differ by age.

Additional analyses examined for effect modification. Medical conditions, in particular vascular diseases such as diabetes, are increased in blacks compared to whites, and many of these conditions are known to be associated with impaired cognition (13,14). Also, medical conditions may be associated with lower BMI and be a confounder in the relation of BMI to cognition. We therefore conducted additional analyses to examine whether medical conditions might account for the relationship of BMI with cognitive decline. First, in a model adjusting for diabetes, in addition to age, sex, education, and race, the effect of BMI on decline in global cognition was essentially unchanged compared to that in the core model presented in Table 2 (estimate = 0.0014, *SE* = 0.0005, $p = .0034$), suggesting that diabetes does not play a substantial role in the relationship of BMI with cognitive decline. Similarly, in a separate analysis taking into account the number of medical conditions present, results were also essentially unchanged (data not shown).

BMI and Cognitive Decline as a Function of Race

Because the average BMI level differed among blacks and whites, we aimed to test whether the relation of BMI to change in cognition differed by race: specifically, we tested whether blacks, who have on average a higher BMI than whites, had a differential rate of decline in cognitive function, compared to whites. In order to test for effect modification by race, we created a third set of mixed-effect models, by building on the core model but adding two terms for the interaction of BMI with race (Race × BMI and Time × Race × BMI). As before, all model terms included age, sex, and education. As shown in Table 3, there was no significant interactions of BMI with black race for any of the cognitive outcomes, including global and domain scores (all p values > .09), suggesting that the association of BMI with change in cognition does not significantly differ between black and white persons.

Next, we conducted sensitivity analyses to verify if the lack of interaction of BMI at baseline and race (Race × BMI and Time × Race × BMI) was due to differences between blacks and whites on other important covariates, such as the differential mortality by race (19), lower baseline age of blacks, or higher BMI among blacks. In these analyses, we obtained a propensity score to balance the person-specific characteristics of the blacks and whites, specifically age at baseline, sex, education, BMI at baseline, death, and number of follow-ups. These scores were used to compute normalized inverse probabilities that were used to weigh models, giving higher weight for whites similar to blacks. Results showed that interactions between race and BMI remained nonsignificant, for models similar to those in Table 3 (data not shown). These analyses are consistent with the finding of no racial differences among blacks and whites, in the relation of BMI to cognitive decline.

Discussion

In this study of more than 2,100 older black and white persons, late-life BMI was related to change in cognitive function, with lower BMI being related to faster rates of decline in summary measures of global cognition, and specifically semantic memory and episodic memory. BMI was not related to decline in cognitive function in other cognitive domains, such as perceptual speed. And, in separate analyses examining for effect modification, the relation of BMI to cognitive decline did not change when accounting for diabetes or common medical conditions in aging. In analyses testing whether relations differed by race, there was no interaction of BMI with race, suggesting that the effect of BMI on cognitive decline is similar in both blacks and whites. Furthermore, results were similar in sensitivity analyses using propensity scoring to weigh models and minimize racial differences in key covariates.

BMI values outside the range of what is currently considered healthy, is increasingly common. Several factors are likely contributing to high or low BMI, such as current trends in lifestyle (eg, a sedentary lifestyle is associated with increasing BMI) and the aging population (eg, aging is associated with decreasing BMI) (20). Many clinicians consider BMI to be a potentially modifiable risk factor for adverse health outcomes, in particular for mortality due to vascular diseases (eg, myocardial infarction), as well as for morbidity associated with other medical conditions (eg, fractures in aging). Because vascular disease is a common cause of cognitive impairment and dementia, researchers are interested in understanding the relation of

Table 3. Examination for Potential Race Effects on the Relationship of BMI at Baseline to Change in the Global Cognitive Score and Five Cognitive Domains, in the Total Group of 2,134 Participants*

Model Term	Outcome Estimate (SE), *p*					
	Global Score	Domains**				
		Semantic Memory	Episodic Memory	Working Memory	Perceptual Speed	Visuospatial Ability
Race	−0.4301 (0.0240), <.001	−0.4592 (0.0323), <.001	−0.2795 (0.0307), <.001	−0.4157 (0.0338), <.001	−0.5183 (0.0336), <.001	−0.7195 (0.0331), <.001
BMI at baseline	0.0043 (0.0019), .025	0.0033 (0.0026), .203	0.0079 (0.0024), .001	0.0015 (0.0027), .568	0.0056 (0.0027), .038	−0.0036 (0.0026), .168
Time	−0.0729 (0.0034), <.001	−0.0883 (0.0044), <.001	−0.0599 (0.0041), <.001	−0.0583 (0.0033), <.001	−0.0995 (0.0032), <.001	−0.0364 (0.0031), <.001
Time × race	0.0200 (0.0061), .001	0.0132 (0.0080), .101	0.0085 (0.0073), .243	0.0203 (0.0060), .001	0.0231 (0.0057), <.001	0.0109 (0.0056), .050
Time × BMI at baseline	0.0012 (0.0006), .056	0.0020 (0.0008). .014	0.0015 (0.0008), .049	0.0001 (0.0006), .809	0.0004 (0.0006), .48	0.0003 (0.0006), .632
Time × race × BMI at baseline	0.0007 (0.0009), .446	0.0006 (0.0012), .644	0.0004 (0.0011), .745	0.0015 (0.0009), .091	0.0003 (0.0009), .727	0.0008 (0.0008), .357

Note: BMI = Body mass index.
*Six separate linear mixed-effects models, each adjusted for age, sex, and education. ** Bonferroni corrected $\alpha = 0.01$.

BMI to cognition in aging. Yet, the relationship of BMI to biologic processes is complex. Indeed, the relationship of BMI to mortality follows a U-shaped curve, with persons at either end of the spectrum, with low or high BMI, having an increased risk of death (20).

The published literature on late-life BMI (in persons older than 65 years of age) and cognition is mixed. Several cross-sectional studies have found that late-life BMI is associated with dementia and level of cognitive function, and some findings point to high BMI and others to low BMI, as being associated (21–23). Some studies have examined the role of late-life BMI in cognitive decline within restricted samples, such as persons with dementia or mild cognitive impairment (3,5). Few longitudinal studies have examined the prospective relation of late-life BMI to incident cognitive impairment or dementia, and results from these studies suggest that lower, compared to higher BMI, is a predictor of poor cognitive outcomes (24–26). There is very little data on late-life BMI and change in cognition in different racial groups or across different cognitive functions. In the Cardiovascular Health Study, higher late-life BMI was associated with less cognitive decline in the digit symbol modalities test, a measure of executive function (4). But, other cognitive domains were not assessed. Our findings are in keeping with this prior study, in that higher BMI was associated with slower cognitive decline, and lower BMI was associated with faster cognitive decline, including in a global measure. But our study extends the literature by examining five different cognitive domains, each based on two or more individual neuropsychological tests, and by also considering longitudinal BMI data. We found that lower BMI is related with faster decline in both semantic memory and episodic memory, but not working memory, perceptual speed, or visuospatial ability. Future work will need to replicate our findings.

As indicated previously, most research on BMI, whether in midlife or late-life, and cognitive decline is among largely white populations (25,26). Yet, body mass indices such as BMI and body mass composition, differ among racial groups, and BMI is higher among several minority groups, including blacks (27–30). Further, while BMI tends to decline with aging (19), a recent study of both blacks and whites found that persons who are obese have a faster rate of decline in BMI (31). Racial differences in BMI, along with the mixed findings in minority populations of high BMI being protective but obesity being a risk for decline, point to the importance of examining race as a potential modifier in the relation of BMI to cognitive decline in aging. But while some studies have examined midlife and late-life BMI and cognition among black persons, data has been limited due to a range of factors, such as studies only including black but no other races, or studies limited to cross-sectional analyses (32). In one large prospective study of 1331 older black participants followed longitudinally for 6 years, authors found that a declining late-life BMI was associated with incident cognitive impairment, both mild cognitive impairment and, separately, dementia (33). But differential effects of race among blacks and whites could not be determined. In cross-sectional analyses using data from the Health and Retirement Study, and including about 550 blacks, results showed that the relation between late-life BMI (based on self-report weight and height) and a general test of cognition administered by phone, varied by race (6). But findings in blacks were similar to those in whites, for BMI not in the obese range. Another study which included both blacks and whites and was longitudinal, using data from the National Alzheimer's Coordinating Center, showed that among persons with mild cognitive impairment, late-life BMI and race (black vs white) were differentially related to a faster rate of decline to dementia (34). More recently, a population-based study of blacks and whites

showed that the relation of late-life BMI to cognitive decline differed by a genetic factor (7). We are not aware of any prior study examining the relation of BMI to cognitive decline in different cognitive domains, which directly examined racial differences among blacks and whites in the association. Our data extends the knowledge in the field by showing that while late-life BMI is related to cognitive decline, and memory decline in particular, that this relation does not appear to be influenced by race. Our main results were supported by sensitivity analyses that used scores based on normalized inverse probabilities to weigh models so that racial differences across key covariates were minimized.

Mechanisms relating BMI to cognition are unclear. One plausible consideration is that life-stage and age-related factors may play a role. Indeed, higher midlife BMI has been related to increased risk of dementia (26) and to faster decline in cognitive function (35). But some findings suggest a more complex relationship, with also normal or low mid-life BMI predicting cognitive decline and some data not showing any relation (36,37). Data examining this question among older persons are also available (24). One study across the adult life span found an interaction of BMI with age, with high BMI in older age groups being associated with poor cognitive performance (38). In the Baltimore Longitudinal Study of Aging which included longitudinal follow-up data of adults with a mean age of 55 years at baseline, the relation of obesity to change in cognition, both decline and incline depending on the cognitive domain, varied by age (39). We did not find evidence for an interaction of BMI with age, although the age range in our study was restricted to older adults. Another consideration is whether BMI is related to cognition via known risk factors for cognitive decline, and vascular risk factors in particular. One study reported that the association of apolipoprotein E epsilon 4 to cognitive decline is reduced in obese older persons, compared to those with normal BMI (7). And, because we and others have shown that the common vascular risk factor, diabetes, is related to cognition, including in these cohorts (14), we considered diabetes, and separately a range of medical conditions. Our result of a relation of BMI to cognitive decline was unchanged when controlling for these factors. Other mechanisms, including involving neurodegenerative and cognitive reserve, need more exploration (40). And, while much attention has been given to the weight component of BMI, and some research focuses on overweight as the central features of interest relating BMI to cognition, underweight and height itself (eg, height loss in aging, commonly associated with low vertebral bone mass), may also be of importance, either in direct or indirect processes, and warrant more consideration. Future research on these and other factors, including inflammation, neuroimmunity, and other plausible biologic processes, is needed to elucidate mechanisms linking late-life BMI to cognitive decline in aging. Because abnormal BMI is common and BMI is modifiable, a better understanding of mechanisms linking BMI to cognitive decline is a promising avenue for research to potentially have a positive impact on healthy aging.

This study has weaknesses and strengths. One limitation is that BMI was considered in late-life and it may be that BMI earlier in life or factors related to BMI that are not available in our study (eg, waist circumference), would be more meaningful predictors of decline in cognition in late life. Nonetheless, our previous work has shown that most of the contribution of late-life BMI range is in the midpoint of decline (19), and here we used data from across the study to center BMI by height and results showed relations of BMI to cognitive decline, and additional analyses used longitudinal BMI. A second limitation is that we were not able to consider incident cognitive conditions, such as dementia, given that relatively fewer blacks compared to white, developed dementia over the course of the follow-up (with younger age at baseline likely playing a role). A third limitation is that underlying mechanisms linking BMI to cognitive decline were not elucidated in this study and are in need of further examination. Yet, we also note several strengths to our study. We included a large number of both blacks and white persons, allowing us to explore the potential for differences of the relation of BMI to cognition by race. Also, cognitive assessments included a detailed battery of nearly twenty neuropsychological tests administered annually. Data were very complete, with missing data in less than 1% of participants at baseline on each of the individual neuropsychological tests except one (for which missing data was <10%). These assessments allow for determination of change in cognition over more than two time points, thus adding additional precision to the estimates for change in cognition. Further, examination of five different cognitive domains provides additional data about which brain networks are most affected by BMI, and thus gives some insight into directions for future research in this field.

Supplementary Material

Supplementary data is available at *The Journals of Gerontology, Series A: Biological Sciences and Medical Sciences* online.

Funding

This work was supported by the National Institutes of Health, grants R01 AG17917, RF1 AG22018, R01 AG40039, and R01 NS084965.

Conflict of Interest

None reported.

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