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Is Body Mass Index in Old Age Related to Cognitive Abilities? The Lothian Birth Cohort 1936 Study

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We tested the hypothesis that the previously reported association between a higher body mass index (BMI) and poorer cognition in later adulthood is an artifact of confounding by previous cognitive ability and socioeconomic status. Participants were 1,079 adults aged about 70 years in the Lothian Birth Cohort 1936 Study, on whom there are IQ data from age 11. Cognitive outcome measures included: IQ at age 70 using the same test that was administered at age 11; composite measures of general cognitive ability (g factor), speed of information processing, and memory; and two tests of verbal ability. People classified as overweight or obese in later adulthood had significantly lower scores on tests of childhood IQ, age 70 IQ, g factor, and verbal ability. There was no significant association with processing speed or memory performance. After adjusting for childhood IQ and social class in general linear models, associations with age 70 IQ and g factor were nonsignificant or attenuated. However, throughout the models, there was a persistent (inverse) relationship between BMI and performance on the National Adult Reading Test (NART) and Wechsler Test of Adult Reading (WTAR), which remained significant after full adjustment for all sociodemographic and health covariates (for the NART, $p = .025$; for the WTAR, $p = .011$). The findings suggest that the previously reported BMI–cognition associations in later adulthood could be largely accounted for by prior ability and socioeconomic status, and by the possible influence of these factors on the adoption of health behaviors in adulthood.

Keywords: body mass index, obesity, cognitive function, childhood IQ, aging.

Overweight and obesity rates are soaring worldwide, even among older adults. Scotland has one of the worst obesity records among developed countries (Organisation for Economic Co-Operation and Development, 2007). The Scottish Health Survey of 2008 identified that over two thirds of adults ages 65–74 were either overweight or obese (Gray & Leyland, 2009). The adverse health effects of being overweight at older ages are well estab-lished and include hypertension, hyperlipidemia, Type II diabetes, and cardiovascular disease (CVD; Brown et al., 2000; Dixon, 2010; Field et al., 2001). Such diseases can lead to early death and significant morbidity in those who survive to old age. In recent years, much scientific research has also been devoted to investi-gating whether being overweight leads to faster cognitive decline in later life.

Several population-based studies demonstrate an association between overweight and obesity in midlife, commonly defined according to body mass index (BMI), and an increased risk of cognitive impairment in later life (Cournot et al., 2006; Dahl et al., 2009; Elias, Elias, Sullivan, Wolf, & D'Agostino, 2003) and dementia (Gorospe & Dave, 2007; Gustafson, Rothernberg, Blennow, Steen, & Skoog, 2003; Kivipelto et al., 2005; Rosengren, Skoog, Gustafson, & Wilhelmsen, 2005; Whitmer, Gunderson, Quesenberry, Zhou, & Yaffe, 2007). In a study of healthy, middle-aged men and women, a higher BMI was associated with lower cognitive function at baseline (after adjustment for demographic and health variables) and with greater cognitive decline after a 5-year follow-up period (Cournot et al., 2006). However, the analyses did not adjust for baseline cognitive ability. Given the strong correlation between baseline and follow-up cognitive scores (i.e., the stability of individual differences in cognitive abilities), some doubt must be cast on the longitudinal nature of these and other findings.

Previous reports suggestive of an increased risk of cognitive impairment with a higher BMI are typically based on the effects of BMI measured at midlife. Whereas there is some suggestion that this inverse BMI–cognition relationship does not appear to vary with age (Gunstad et al., 2007), the results from studies examining

old-age BMI and risk of cognitive impairment are less consistent. Research conducted in some elderly populations appears to show the opposite relationship, where a lower BMI (normal weight) was associated with worse cognition (Kuo et al., 2006; Ng, Feng, Niti, & Yap, 2008; West & Haan, 2009), a faster rate of cognitive decline, and an increased risk of dementia, compared with a higher BMI indicating overweight (Atti et al., 2008; Buchman et al., 2005). Aging is sometimes associated with weight loss, and in the elderly, underweight often precedes clinical evidence of dementia and can be a general marker of ill health (Barrett-Connor, Edelstein, Corey-Bloom, & Wiederholt, 1998; Rosengren et al., 2005; Whitmer et al., 2007). It is clear that the relationship between obesity parameters and cognitive function becomes more complex with age and that there are gaps in the evidence, especially for individuals aged 70 and over without dementia.

The direction of causality typically assumed in many studies is from overweight/obesity to impaired cognition. Several mechanisms, based on the action of biological mediation pathways, have been proposed to account for this effect, including an increased risk of vascular disease (Decarli, 2004), neurochemical changes (Bray, 2004), and brain abnormalities (Gazdzinski, Kornak, Weiner, & Meyerhoff, 2008) with increased adiposity. However, there is increasing evidence, supported by the results of many longitudinal birth cohort studies, that early life cognition is a significant influence on both later life BMI and cognition. It is known that early life intelligence (as assessed by IQ-type tests) accounts for the majority of variance in later life intelligence (Deary, Whalley, Lemmon, Crawford, & Starr, 2000). Furthermore, results from large-scale, longitudinal studies show that a lower childhood IQ score is related to adiposity in adulthood and associated health conditions such as hypertension, CVD, and mortality (Chandola, Deary, Blane, & Batty, 2006). Crucially, intelligence from childhood is also associated with obesity-related health behaviors, such as diet, physical exercise, smoking (Batty, Deary, Schoon, & Gale, 2007; Chandola et al., 2006), and alcohol consumption (Batty, Deary, & Macintyre, 2006; Batty et al., 2008; Mortensen, Sørenson, & Grønbæk, 2005). On the basis of recent epidemiological evidence, Gottfredson (2004) suggested that IQ may be the single, most important factor in the adoption of health behaviors and maintaining a healthy body weight. It is therefore possible that the link between a higher BMI and cognitive impairment in older people may be indirect and explained by a lower intelligence in early life. People with lower childhood IQ test scores are generally more socioeconomically disadvantaged in adulthood (Sacker, Schoon, & Bartley, 2002; Deary et al., 2005). This could potentially explain why the risk of being overweight or obese is also increased in those from low socioeconomic status groups (Ball & Crawford, 2005; Sobal & Stunkard, 1989; Vernay et al., 2009). The Scottish Health Survey of 2008 reported that men and women in the most deprived areas had the highest age-standardized prevalence of obesity (Gray & Leyland, 2009).

We hypothesized that the apparent obesity/overweight-related risk of impaired cognition in adulthood, and possibly old age, is principally a result of a lower previous ability and socioeconomic status. It is also possible that poor health status may confound the relationship between BMI and cognition. Those who are less healthy are more likely to become cognitively impaired. Examining the possibility of reverse causation (where the effect has preceded the cause)—that is, that early life IQ is an antecedent of both BMI and old-age cognition—requires a sample of individuals for whom there is a measure of intelligence from their early life, but this is rarely available. The Lothian Birth Cohort 1936 Study (LBC1936) is advantageous for examining the effects of BMI and cognitive function in later life for several reasons: The cohort is unusual in having validated measures of cognitive ability from ages 11 and 70 years; the availability of possible confounding health and demographic information; and a large cognitive test battery at age 70. The cross-sectional aspect of the design of the present study was such that we could investigate the relationship between BMI and cognitive ability in old age. There was also a longitudinal aspect: By controlling for childhood IQ, we could additionally examine the relationship between cognitive change across the lifespan and later BMI. We emphasize that this was not a fully longitudinal investigation, given that BMI is only measured at one time point (old age). The specific aims of the present study were to investigate whether a higher BMI in later life is associated with poorer cognitive outcomes and whether these relationships are substantially accounted for (confounded) by previous cognitive ability (age 11 IQ), socioeconomic status, and/or health measures. Cognitive assessment at age 70 included multiple markers of general cognitive ability, memory, speed of information processing, and verbal ability.

Method

Participants and General Method

We analyzed data collected from the LBC1936, which comprises 1,091 men and women aged ~70 years at the first time of testing in old age. These individuals are surviving participants of the Scottish Mental Survey of 1947 (SMS1947; see Deary, Whalley, & Starr, 2009). The SMS1947 tested the mental ability of almost all Scottish schoolchildren born in 1936 and attending school on June 4th, 1947 (mean age, 11 years), with a well-validated test of general intelligence: a version of the Moray House Test No. 12 (MHT; Scottish Council for Research in Education [SCRE], 1933, 1949). Full details of the recruitment and testing of the LBC1936 are given in a free-access protocol paper (Deary et al., 2007). At the time of recruitment, LBC1936 members mostly resided in Edinburgh and its surrounding area (Lothian) in Scotland. They were relatively healthy and lived independently. Between 2004 and 2007, LBC1936 participants attended the Wellcome Trust Clinical Research Facility at the Western General Hospital in Edinburgh to undergo cognitive testing, a clinical assessment, and an interview and to take home a large questionnaire to be completed and posted back. Cognitive testing was conducted by trained researchers and the physical assessments by research nurses. The structured interview ascertained demographic information and medical history. As a part of their general assessment, LBC1936 participants completed a set of diet, personality, and lifestyle questionnaires (Deary et al., 2007). Of the 1,091 participants interviewed, 1,090 had height and weight measurements taken, enabling BMI to be calculated. Eleven individuals were identified as having potential dementia, on the basis of a score of <24 on the Mini-Mental State Examination (MMSE; Folstein, Folstein, & McHugh, 1975), and were excluded from the analyses. The final sample for analysis in the present study comprised 1,079 people (for age, $M = 69.5$ years, $SD = 0.8$ years) at

the time of testing. Ethics permission for the LBC1936 protocol was obtained from the Multi-Centre Research Ethics Committee for Scotland (MREC/01/0/56) and from Lothian Research Ethics Committee (LREC/2003/2/29). The research was conducted in compliance with the Helsinki Declaration. All participants gave their written, informed consent.

Procedure

Measurement of BMI. Trained research nurses measured height and weight as part of a physical examination using a standardized protocol. Height (in centimeters) was measured with a SECA stadiometer on individuals not wearing shoes. The research nurses measured weight (in kilograms) for individuals without outer clothing or shoes using electronic SECA scales with a digital readout. BMI was calculated as weight (in kilograms) divided by height squared (in square meters).

Measurement of cognitive performance at age 70. A full description of the cognitive tests and administration procedures can be found in the free-access LBC1936 protocol article (Deary et al., 2007). Brief descriptions of the tests follow.

The MMSE is a test of global cognitive function (Folstein et al., 1975) and commonly used by clinicians and researchers as a screening test for cognitive impairment. Scores range from 0 to 30, with a score of less than 24 often used to indicate possible dementia.

From the Wechsler Adult Intelligence Scale—III[UK] (WAIS–III; Wechsler, 1998a) we included Digit Symbol Coding (for speed of information processing), Block Design (for constructional ability), Matrix Reasoning (for nonverbal reasoning), Digit Span Backwards (for working memory), Symbol Search (for speed of information processing), and Letter–Number Sequencing (for working memory).

From the Wechsler Memory Scale—III[UK] (WMS–III; Wechsler, 1998b) we used Logical Memory I and II (for verbal declarative memory and for immediate and delayed recall), Verbal Paired Associates I and II (for verbal learning and memory and for immediate and delayed recall), and Spatial Span (for nonverbal spatial learning and memory).

The National Adult Reading Test (NART; Nelson & Willison, 1991) and Wechsler Test of Adult Reading (WTAR; Holdnack, 2001) are widely used to estimate previous cognitive ability, and they require the pronunciation of irregular words. Simple and Four-Choice reaction time (RT) tasks measure speed and variability of simple information processing with a purpose-built portable box (Cox, Huppert, & Whichelow, 1993; Deary, Der, & Ford, 2001). Inspection Time is a computer-based task used to assess speed of elementary visual processing with no requirement for speeded reactions (Deary, Simonotto, et al., 2004).

The MHT (SCRE, 1933, 1949), previously taken by participants in the SMS1947 at age 11, was repeated at their clinic visit aged about 70. The MHT comprises 71 items (mostly verbal reasoning, but also some numerical, spatial, and other items) and has a maximum possible score of 76 and a 45-min time limit. MHT scores were corrected for age in days at time of testing and converted to an IQ scale where $M = 100$ and $SD = 15$.

Covariates, including childhood IQ. Data on age, education (number of years full time), marital status (married, widowed, unmarried/divorced), smoking status (current, ex-smoker, or never smoked), and alcohol intake (units per week) were collected by structured interview. A medical history was taken including diagnoses of hypertension, diabetes, CVD, high cholesterol, and stroke (such diseases are associated with obesity). A physical activity measure (days per month of exercise) was obtained from a self-report questionnaire booklet comprising various social and life-style questions. Childhood (age 11) MHT scores were obtained, in collaboration with SCRE and with the permission of LBC1936 participants, from the original SMS1947 records. This mental test was concurrently validated against the Terman–Merill revision of the Binet scales (SCRE, 1949). The test conducted at age 11 reflects cognitive functioning toward the end of primary school education and is a valid measure of early life ability. As before, age 11 MHT scores were corrected for age in days at time of testing and converted to an IQ scale where $M = 100$ and $SD = 15$. In this sample, the correlation between age 11 and age 70 MHT-derived IQ is .69 ($p < .001$). Social class was derived from participants' highest reported occupation and consisted of 6 categories: I (professional occupations); II (managerial and technical occupations); and III (skilled occupations)—divided into IIIN (nonmanual) and IIIM (manual)—IV (partly skilled occupations); and V (unskilled occupations; Office of Population Censuses and Surveys, 1980). Because of the small number of participants in class V, classes IV and V were combined. The women in the cohort were asked for their husband's occupation as well as their own and were assigned a social class based on the highest occupation of the household. This was derived from their own occupation for about half of the women and from the husband's occupation for the remainder.

Statistical Analyses

For descriptive purposes, participants were categorized according to standard BMI classification by the World Health Organization (WHO, 2000); namely, underweight (<18.5), normal weight (18.5–24.99), overweight (25–29.99), or obese (≥ 30). We used analysis of variance and chi-square tests to examine whether demographic and health variables differed between BMI categories. The main analyses examined the associations between BMI (as a continuous variable) and cognitive function outcome scores at age 70 using general linear models (GLMs). Seven models were fitted to the data, each including adjustment for potential confounding factors. Although there was a relatively small difference in age across this birth cohort, assessment took place over a period of 3 years, and age (in years) at assessment ranged from 67.7 to 71.3. Therefore, we adjusted for the effects of chronological age in each model. Both age (in days at time of testing) and gender were included as covariates in all models. The second model included occupational social class. The third model included age 11 IQ to control for early life cognitive ability, and the fourth model included education. The fifth and sixth models included health behaviors and health measures, respectively. The final and seventh model included all the covariates mentioned.

A general cognitive ability component score (g factor) was derived from principal-components analysis (PCA) of six WAIS–III subtests (Letter–Number Sequencing, Matrix Reasoning, Block Design, Digit Symbol, Digit Span Backwards, and Symbol Search). A general processing speed component score (speed) was similarly derived from a PCA of the set of speed measures (Sym-

bol Search, Digit Symbol, Simple RT mean, Four-Choice RT mean, and Inspection Time). The extraction of these factors by PCA has been described elsewhere (Luciano et al., 2009). Using the same method, we extracted a general memory factor (memory) from the WMS–III subtests (Logical Memory I Immediate and II Delayed Recall, Spatial Span Forwards, Spatial Span Backwards, Verbal Paired Associates I Immediate and II Delayed Recall) and two WAIS-III subtests (Letter–Number Sequencing and Digit Span Backwards). The first unrotated principal components for the g factor, processing speed factor, and memory factor, explained 53%, 51%, and 43% of the variance, respectively. The fourth cognitive outcome variable used in the present study is age 70 MHT-derived IQ. Additional analyses were performed with NART and WTAR scores. We present relevant estimates of effect size (η_p^2) in addition to p values.

Results

Descriptive Characteristics

Of the 1,079 participants, 538 were men and 541 were women. The mean age was 69.5 (SD = 0.8). The mean BMI for the study population was 27.8, (SD = 4.4; range = 16.0–48.5).

Characteristics of BMI categories. Table 1 shows the main characteristics of participants according to BMI category. Of the sample, 0.6% were underweight (n = 7), 25.3% were normal weight (n = 273), 47.3% were overweight (n = 510), and 26.8% were obese (n = 290). According to criteria from the WHO (2000), nearly three quarters of the sample were either overweight or obese. As the underweight category comprised only 0.6% of the sample, we were unable to draw reliable conclusions with regard to the general characteristics of this category. With increasing BMI (in the normal weight, overweight, and obese categories), participants were significantly more likely to be male, have a more manual occupational social class, less education, a lower MMSE score, and more likely to be an ex-smoker. In terms of health measures, a higher BMI category was associated with an increased incidence of hypertension, diabetes, and CVD but not stroke or elevated cholesterol.

BMI category and cognitive outcomes at ages 11 and 70. Those with a higher BMI at age 70 were significantly more likely to have a lower childhood IQ (p = .03; see Table 2; the difference between being obese and being of normal weight was associated with a childhood IQ difference of 4.8 points (Cohen's d = .33). The higher BMI–lower IQ relationship was still apparent almost

Table 1

Characteristics of the Study Population by BMI Category (Mean Values and % Prevalence)

Sociodemographic variable	Total sample (N = 1,079)		Underweight (n = 7)		Normal weight (n = 273)		Overweight (n = 510)		Obese (n = 289)		p
	M	SD	M	SD	M	SD	M	SD	M	SD	
Age (years)	69.5	0.83	69.3	0.8	69.5	0.9	69.5	0.8	69.6	0.8	.07
Gender											**<.001**
% Male (n = 538)	49.9		0		39.9		55.1		51.2		
% Female (n = 541)	50.1		100		60.1		44.9		48.8		
Marital status											.71
% Married	71.2		57.1		70.0		73.7		68.2		
% Widowed	13.5		28.6		12.4		12.3		16.3		
% Unmarried/divorced	15.3		14.3		17.6		14.0		15.5		
Social class											**.002**
I	17.9		28.6		20.0		17.8		15.7		
II	37.6		42.9		39.6		40.0		31.3		
IIIN	23.2		28.6		28.1		20.4		23.5		
IIIM	17.4		0		9.3		18.2		24.2		
IV + V	3.9		0		3.0		3.6		5.3		
Education (years full time)	10.7	1.1	10.6	0.8	10.9	1.2	10.7	1.1	10.6	1.1	**.01**
MMSE	28.9	1.3	29.3	1.1	29.1	1.2	28.8	1.3	28.8	1.3	**.02**
Health behaviors											
Alcohol intake (units/week)	10.5	14.2	3.4	4.3	10.8	14.2	10.6	13.3	10.3	15.8	.59
Physical activity (days/month)	7.6	8.1	5.1	10.5	8.4	8.5	7.5	7.8	7.2	8.1	.30
Smoking status											**<.001**
% Nonsmokers	43.6		28.6		46.9		43.5		41.2		
% Ex-smokers	43.2		28.6		33.3		43.9		51.5		
% Current smokers	13.2		42.8		19.8		12.5		7.3		
Health measures											
% With hypertension	39.6		28.6		24.5		39.2		54.7		**<.001**
% With diabetes	8.2		0		2.2		8.0		14.2		**<.001**
% With CVD	24.6		28.6		19.4		22.3		33.2		**.001**
% With cholesterol	35.3		14.3		32.7		35.5		38.1		.37
% With stroke	5.0		0		4.0		5.9		4.1		.53

Note. Social classes are categorized as follows: I (professional occupations); II (managerial and technical occupations); and III (skilled occupations)—divided into IIIN (nonmanual) and IIIM (manual)—IV (partly skilled occupations); and V (unskilled occupations). p values are from t tests, analyses of variance, and chi-square tests as appropriate. BMI = body mass index; MMSE = Mini-Mental State Examination; CVD = cardiovascular disease. Probabilities for significant effects are given in boldface.

Table 2

Cognitive Outcomes by BMI Category, With Reported Means and p Values

	M (and SD) for:				
Variable	Underweight (n = 7)	Normal weight (n = 272)	Overweight (n = 510)	Obese (n = 290)	p
Age 11 IQ[a]	102.3 (14.2)	102.7 (14.7)	100.0 (14.9)	97.9 (14.7)	**.003**
Age 70 IQ[a]	93.2 (20.1)	102.2 (13.3)	100.2 (14.1)	99.2 (14.7)	**.037**
g Factor[b]	−0.26 (0.92)	0.12 (0.92)	0.04 (1.03)	−0.11 (0.93)	**.025**
Processing speed[b]	−0.37 (0.64)	0.10 (0.94)	0.05 (1.02)	−0.09 (0.94)	.091
Memory[b]	−0.18 (1.7)	0.05 (0.99)	0.02 (0.98)	−0.02 (0.95)	.799
NART	37.4 (12.0)	36.4 (7.6)	34.4 (8.1)	33.1 (8.0)	**<.001**
WTAR	43.4 (10.2)	42.9 (6.1)	40.9 (7.2)	39.8 (7.2)	**<.001**

Note. BMI = body mass index; NART = National Adult Reading Test; WTAR = Wechsler Test of Adult Reading.
[a] Age 11 and age 70 Moray House Test No. 12 scores were corrected for age (in days at time of testing) and converted to standard IQ-type scores where $M = 100$ and $SD = 15$. [b] Cognitive-component scores derived from principal-component analysis. These are standard scores, with $M = 0$ and $SD = 1$ in the whole sample. Probabilities for significant effects are given in boldface.

60 years later. At age ∼70, participants in the obese category scored on average 3 points less (Cohen's $d = .22$) on the same measure of IQ (as that taken at age 11) than those in the normal weight BMI category. A similar relationship was seen across the cognitive domains representing general ability (g factor) and verbal ability (NART and WTAR), whereby overweight and obese participants scored significantly more poorly than those of normal weight. Performance on processing speed and memory tasks did not differ significantly between BMI categories, although a similar inverse trend was observed. Although the mean childhood IQ score of the underweight group exceeded those of the overweight and obese groups, by age 70 they performed significantly worse on tests of general cognitive ability, processing speed, and memory but higher than all of their counterparts on the verbal ability tests. However, these results should be interpreted with caution, given the small size of the group and the large standard errors for their cognitive test scores at age 70.

General Linear Models

Seven GLMs were fitted to the data for examination of the contribution of BMI (as a continuous variable) and potentially confounding variables to cognitive function at age 70 (see Table 3). In the initial age- and gender-adjusted model, BMI was significantly, inversely associated with performance on tests of age 70 IQ, general cognitive ability (g factor), the NART, and the WTAR; the largest effect sizes were seen for the NART and WTAR. The BMI–processing speed association narrowly missed conventional significance, at $p = .051$. When occupational social class (Model 2), age 11 IQ (Model 3), and education (Model 4) were independently added to the base model, those previously significant associations with age 70 IQ and the g factor almost all became nonsignificant (the relationship between BMI and g factor in Model 4 remains marginally significant at $p = .045$), and their effect sizes were markedly attenuated; in the case of age 11 IQ, totally attenuated. Model 5, which has age, gender, and health

Table 3

Associations Between BMI Score and Cognitive Outcomes at Age 70 (General Linear Models, p Values and Associated Partial Eta-Squared Values)

Model	Age 70 IQ (n = 1,069)[a]		g factor (n = 1,062)		Processing speed (n = 1,028)		Memory (n = 1,037)		NART (n = 1,077)		WTAR (n = 1,077)	
	p	η_p^2	p	η_p^2	p	η_p^2	p	η_p^2	p	η_p^2	p	η_p^2
1. Gender + age	**.002**	.009	**.001**	.010	.051	.004	.13	.002	**<.001**	.028	**<.001**	.033
2. Gender + age + social class	.26	.001	.18	.002	.85	.000	.76	.000	**<.001**	.013	**<.001**	.017
3. Gender + age + age 11 IQ	.92	.000	.69	.000	.72	.000	.11	.003	**.003**	.009	**<.001**	.014
4. Gender + age + education	.082	.003	**.045**	.004	.36	.001	.76	.000	**<.001**	.016	**<.001**	.021
5. Gender + age + health behaviors	**.010**	.007	**<.001**	.013	**.038**	.005	.059	.004	**<.001**	.038	**<.001**	.039
6. Gender + age + health measures	**.015**	.006	**.040**	.004	.36	.001	.33	.001	**<.001**	.021	**<.001**	.025
7. All covariates	.42	.001	.31	.001	.18	.002	.31	.001	**.025**	.006	**.011**	.008

Note. p values in bold represent significant negative associations between BMI score and cognitive outcome. Health behaviors = alcohol consumption, smoking, and physical activity; health measures = CVD, hypertension, and diabetes.
[a] Age 70 IQ is already age adjusted (age is not included in the models for this outcome variable).

behaviors as covariates, showed significant effects of BMI on age 70 IQ, the g factor, and processing speed, with effects sizes almost as large, or larger, than the baseline model with just age and gender as covariates. The BMI–NART and BMI–WTAR inverse associations remained significant throughout each of the models, although their effects sizes were reduced by about 50%. BMI associations with these two outcomes also remained significant in those models controlling for health behaviors (Model 5), health measures (Model 6), and the final (all-inclusive) Model 7. By Model 7, which includes all covariates, there was some greater attenuation, but the associations remained highly significant (for the NART, $p = .025$; for the WTAR, $p = .011$). Finally, we ran the models with interaction terms—BMI $\times$ Age 11 IQ, BMI $\times$ Social Class, and BMI $\times$ Health (with history of CVD as a marker of health)—to investigate whether there were any modifying effects of age 11 IQ, social class, or health. We found no modifying effect of social class or health and a small moderating effect of age 11 IQ on the relationship between BMI and age 70 IQ only. However, this result was only marginally significant, and the effect size was minimal ($p = .049$, $\eta_p^2 = .004$).

Discussion

This study had several main findings. First, about three quarters of our sample of 70-year-old participants were found to be either overweight or obese, according to standard WHO criteria (WHO, 2000). Second, unadjusted associations showed a link between higher old-age BMI (indicative of overweight or obesity) and poorer cognitive performance at age 70 on tests of IQ, general cognitive ability, and verbal ability but not processing speed or memory. These results were consistent with previous reports (e.g., Cournot et al., 2006; Dahl et al., 2009) showing an inverse relationship between BMI in midlife and later life cognition. The third and most novel finding in this study is that current cognitive performance was not associated with BMI after adjusting for previous ability and occupational social class. However, there was one exception: A higher BMI was related to poorer verbal ability (crystallized intelligence) regardless of age 11 IQ, social class, health behaviors, and health measures.

Cognitive abilities in old age are most strongly predicted by one's previous cognitive ability; previous research derived from longitudinal studies of large birth cohorts reveal that childhood IQ accounts for the majority of variance in adult IQ. IQ is relatively stable across the lifespan, regardless of many other factors (Deary et al., 2000; Deary, Whiteman, Starr, Whalley, & Fox, 2004). In the present sample, those with a high early life IQ were much more likely to have a high later life IQ. Variation in people's cognitive ability at age 70 is made up of variation in the lifelong (stable) trait of intelligence, variation in any lifetime relative change in intelligence among the cohort, and some amount of measurement error (which is unavoidable). Having a measure of childhood IQ enabled an estimation of how "bright" an older person is in relation to how "bright" he or she *was*, plus or minus any change relative to peers in the cohort over the life course. Examining the cross-sectional data (i.e., the relation between the BMI and cognitive abilities measured at the endpoint [old age]) at first appeared to suggest that those who were overweight or obese in later life were more likely to show age-related cognitive disadvantage. However, the present study differs from others in that, by controlling for age 11 IQ, we

were able to show that it is the relationship between old-age BMI and the lifetime stable trait of intelligence that is of prime importance.

In the present study, we found strong evidence of an association between early life IQ and age 70 BMI. This finding is consistent with the growing number of studies linking childhood IQ with later adiposity, disease, and mortality (Chandola et al., 2006) and many aspects of both diet and health-related lifestyle (e.g., physical activity) in large population representative samples (Batty et al., 2007; Chandola et al., 2006). The risk of overweight or obesity also varied across social class groups, as previously reported by others (Vernay et al., 2009). Those belonging to the manual social class groups (IIIM, IV, and V) tended toward a higher BMI. People with a lower premorbid intelligence are generally more socioeconomically disadvantaged. However, because a childhood IQ measure precedes any effects of later health, lifestyle, or socioeconomic circumstances, it is plausible that there is a chain of influence on BMI, beginning with childhood cognitive ability, which is then mediated by socioeconomic circumstances through educational and occupational attainment (Johnson, Brett, & Deary, 2010) and lifestyle factors (including diet and health management). We suggest that our findings provide evidence indicative of reverse causation, whereby the effect (lower cognitive ability) actually precedes the assumed cause (higher BMI). Alternatively, there may be a previous common cause to both cognitive ability and overweight, given that there is some evidence to suggest that the IQ–BMI relationship may be present even in very early childhood. Guxens et al. (2009) found that preschool children with a higher cognitive ability at age 4 had a lower BMI at age 6. Childhood IQ may be an intrinsic indicator of general "bodily integrity" (Whalley & Deary, 2001). Given that food choices in young children are governed chiefly by their parents (compared with being self-feeding adults), it is possible that the underlying mechanism in this relationship is environmental circumstances, including parental IQ. In the context of the present study, these results suggest that, rather than adult health behaviors being the mediating factor between IQ and BMI, we must also consider that there may be common underlying etiologies present in early life that lead to both obesity and poorer neurodevelopment. This may warrant future investigation.

If previous ability accounts for most of the association between BMI and cognition in old age, then why do we find a decrement in verbal ability in the overweight and obese, even after adjusting for age 11 IQ, socioeconomic status, health behaviors, and health measures? Whereas performance in other cognitive domains tends to decline with age, verbal ability is relatively immune to the effects of both aging (Carroll, 1993; Hedden & Gabrieli, 2004; McGurn et al., 2004; Schaie, 1996) and health factors. The NART and WTAR are widely believed to reflect crystallized intelligence, which, unlike fluid intelligence, remains relatively intact as people age. Performance is thought to reflect peak adult intelligence (beyond the abilities measured in standard IQ-type tests), which is related to education and intellectual, social, and cultural development across the lifespan. Peak adult ability is likely to be associated with lifestyle preferences that influence the maintenance of a healthy weight.

A higher BMI was also associated with poorer health (as expected), in terms of an increased incidence of hypertension, CVD, and diabetes. There is no doubt that being overweight has signif-

icant, adverse effects on one's physical health and quality of life. However, in this and previous studies, controlling for health indices such as CVD and diabetes had little effect on the BMI–cognition association (Dahl et al., 2009; Sabia, Kivimaki, Shipley, Marmot, & Singh-Manoux, 2009). If cognition at age 70 was mediated by the physical consequences of BMI, we would expect such health measures to cause some substantial attenuation. Given the links between previous cognitive ability, morbidity, and mortality previously identified by various authors, it is plausible that variations in premorbid IQ are also partly contributing to these adverse health outcomes, again perhaps through lifestyle choices.

Our study has the following strengths. These analyses make a novel contribution to this area of research. This is, to our knowledge, the first study to examine the relationship between BMI and cognitive performance that can statistically control for a measure of premorbid IQ. Some previous studies have examined the confounding effects of education, but IQ measured in childhood is a better predictor of cognitive function in adulthood than education. Studies with early life IQ data are rare and offer a unique insight in the investigation of factors affecting cognition with aging. Our results are useful in reconsidering results of previous epidemiological investigations, which have typically focused on a direction of causation suggestive of adverse physical and mental (including cognitive) effects, as a result of excess adiposity. However, most epidemiological data do not allow a firm conclusion to be drawn about a causal relationship between body weight and cognitive abilities. Whereas there is no doubt that there is a link between overweight/obesity and both degenerative disease and cognitive impairment, our data are suggestive of reverse causation. As hypothesized, the outcomes in the present study (and other BMI–cognition investigations) were subject to multidirectional influences that are associated with variation in previous cognitive ability. That is, early life IQ influences, perhaps indirectly, the development of overweight/obesity (Gustafson, 2008), as well as adult cognitive ability.

Second, we used direct measurements of height and weight, taken at the time of testing by trained research nurses. Using standard measurement procedures and instruments rather than self-report data (on which many previous results have been based) is more likely to provide accurate BMI scores. Third, the LBC1936 used a comprehensive neuropsychological test battery, atypical for most epidemiological studies. This allowed an examination of the effects of BMI on different cognitive domains, whereas others have relied on only very crude measures of cognitive function, such as the MMSE (e.g., Ng et al., 2008; Sakakura et al., 2008).

There are some methodological limitations to consider. There were very few underweight individuals in the present study, so we were unable to explore the association between a low BMI and cognitive function. It has been reported that a very low BMI or weight loss are often preclinical markers of dementia in the elderly (Atti et al., 2008). However, a low frequency of underweights is typical of healthy populations without dementia. Although the present study lacked sufficient statistical power to detect any true associations, we noted a trend for poorer cognitive ability in the underweight category at age 70 (not present in childhood) that may warrant further investigation. A second limitation was the use of BMI in an older population. BMI is a useful, general index of a person's weight relative to their height, but it does not take into account the decreasing bone mass, muscle mass, and height associated with aging (West & Hann, 2009). Therefore, using BMI in the elderly may lead to an overestimation of body weight. That said, we were investigating the effects of BMI within a narrow age range, so this would have a minimal effect on results. BMI does not specifically reflect abdominal fat accumulation, unlike a waist circumference measure (Harris et al, 2000). Abdominal adiposity is reported to be a more potent predictor of vascular and metabolic diseases than BMI (Dahl et al., 2009) and confers an increased risk for dementia and cognitive impairment (West & Haan, 2009; Whitmer et al., 2008). However, a recent study has found that obesity indices (including BMI and waist–hip ratio) are not differentially related to neurocognitive outcomes both cross-sectionally and longitudinally, as previously believed (Gunstad, Lhotsky, Wendell, Ferrucci, & Zanderman, 2010). Unfortunately, no measure of abdominal adiposity was available in this data set.

A third limitation was the lack of information regarding weight history. Weight can often decrease in old age (Johnson, Wilkins & Morris, 2006) as a result of illness, poor appetite, poor diet, and age or disease-related loss of height. BMI at age 70 may not be representative of lifetime or even midlife BMI. Given that participants in the study were generally healthy, it is unlikely that this limitation would have a significant effect on results. Aside from being generally healthy, the LBC1936 participants, as self-selecting study volunteers, are a relatively able group (in mind and body) compared with the original, national sample who participated in the SMS1947. Furthermore, Edinburgh is a relatively affluent area and scored higher, on average, than all other areas in the survey (SCRE, 1949). Because of this potential healthy survivor effect, our results may not be representative of Scotland as a whole, or even the United Kingdom. However, within our sample, we were confident that there was a wide range of cognitive abilities and individuals from all occupational social class groups. Moreover, if the range of abilities represented is slightly narrower than a random sample of the population, our findings, if anything, are more likely to underestimate any BMI–cognition associations.

Conclusion

In conclusion, the present study found that the relationship between a higher BMI and poorer cognition in later life is largely an artifact of confounding by a lower childhood IQ and occupational social class. The results suggest that a predisposition to weight gain in adult life is determined, to some extent, by early life circumstances. Although being overweight or obese was linked with a poorer verbal ability in old age (even after controlling for previous ability), we propose that this is not evidence of a direct causal effect but probably a reflection of cognitively and socially patterned subsequent adoption of healthy lifestyles in adulthood. We conclude that there is no evidence that obesity contributes directly to premature brain aging. Rather, there exists a complex relationship between IQ, social circumstances, and BMI involving multiple pathways that interact with each other. One possible mechanism is that those of higher ability maintain a healthy weight by engaging in a healthy lifestyle that protects against cognitive decline. Alternatively, there may be common underlying etiologies present in early life that lead to both obesity and poorer neurodevelopment.

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