Original Article
CLINICAL TRIALS: BEHAVIOR, PHARMACOTHERAPY, DEVICES, SURGERY

Obesity

Childhood Intelligence and Adult Obesity

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Objective: Recent studies conclude childhood intelligence has no direct effect on adult obesity net of education, but evolutionary psychological theories suggest otherwise.
Design and Methods: A population ($n = 17,419$) of British babies has been followed since birth in 1958 in a prospectively longitudinal study. Childhood general intelligence is measured at 7, 11, and 16, and adult BMI and obesity are measured at 51.
Results: Childhood general intelligence has a direct effect on adult BMI, obesity, and weight gain, net of education, earnings, mother's BMI, father's BMI, childhood social class, and sex. More intelligent children grow up to eat more healthy foods and exercise more frequently as adults.
Conclusion: Childhood intelligence has a direct effect on adult obesity unmediated by education or earnings. General intelligence decreases BMI only in adulthood when individuals have complete control over what they eat.

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Introduction

Obesity is an international epidemic. It now affects not only the developed nations like the United States (1) and Europe (2) but also, increasingly, developing nations like China (3). Yet, the precise cause(s) of the recent increase in the prevalence of obesity are not yet well understood (4,5).

One of the individual predictors of obesity is childhood intelligence. Less intelligent children are more likely to grow up to be overweight or obese than more intelligent children (6). Obesity is just one of a large number of health problems that afflict less intelligent individuals, increase their mortality, and decrease their life expectancy (7-9).

In a recent, comprehensive review of the literature on the association between childhood intelligence and adult obesity, Yu et al. (10, p. 666) conclude that ''childhood intelligence is inversely associated with adult obesity. However, after adjustment for educational attainment the association between childhood intelligence and later adult obesity became null.'' In a study of young Danish men, Halkjær, Holst, and Sørensen (11) find that the effect of intelligence in early adulthood on subsequent weight gain and obesity disappears completely when education is controlled for. In their study of the National Child Development Study (NCDS: the same data set that I use in this article), Chandola et al. (12, pp. 1427-1428) conclude that ''whereas the childhood IQ–obesity association persisted following adjustment for a range of childhood characteristics, which included paternal social class, foetal growth and maturation, it was markedly attenuated in both men and women when adjustment was made for the subjects' educational attainment.'' In NCDS, intelligence is measured with 11 different cognitive tests administered at three different ages (7, 11, and 16). Chandola et al. only use two of the five cognitive tests administered at Age 11 (verbal general ability test and nonverbal general ability test) and disregard the three other cognitive tests administered at the same time or those administered at other ages. As I demonstrate in this article, Chandola et al.'s conclusion that education entirely mediates the effect of childhood intelligence on adult obesity is tenable *only* if one uses these two particular measures of intelligence and *not* if one uses all 11 cognitive tests at all ages.

Recent theoretical and empirical developments in evolutionary psychology suggest that childhood intelligence may have a genuine causal effect on adult obesity independent of its effect through education. The Savanna-IQ Interaction Hypothesis (13,14) proposes that, because general intelligence evolved to solve evolutionarily novel problems, more intelligent individuals may be more likely to acquire and espouse evolutionarily novel preferences and values that our ancestors did not possess than less intelligent individuals are. Thus, relative to their less intelligent counterparts, more intelligent children are more likely to grow up to espouse the evolutionarily novel values of left-wing liberalism or atheism (13); to be nocturnal (15); to consume the evolutionarily novel substances of alcohol, tobacco, and psychoactive drugs (16); to prefer evolutionarily novel instrumental music such as classical and light music (17); and regardless of their genetic and hormonal predisposition, to engage in evolutionarily novel homosexual behavior (18).

Because food was scarce and its supply precarious in the ancestral environment, humans are designed to prefer sweet and fatty foods that contain high calories (19, pp. 144-147). Anyone in the ancestral environment who eschewed high-calorie sweets and fats is not likely to have survived long enough and reproduced successfully enough to become our genetic ancestor. So the preference for sweet and fatty food is evolutionarily given and universally shared by all humans.

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TABLE 1 The effect of childhood general intelligence on adult BMI at Age 51

	(1)	(2)	(3)	(4)
Childhood general intelligence	−0.037***	−0.030***	−0.020*	−0.017*
	(0.005)	(0.007)	(0.008)	(0.008)
	−0.096	*−0.076*	*−0.050*	*−0.043*
Education		−0.211**	−0.145	−0.078
		(0.073)	(0.082)	(0.083)
		−0.054	*−0.036*	*−0.019*
Earnings			0.003	0.003
			(0.002)	(0.002)
			0.030	*0.026*
Mother's BMI			0.226***	0.089***
			(0.023)	(0.025)
			0.157	*0.061*
Father's BMI			0.274***	0.109***
			(0.030)	(0.031)
			0.147	*0.058*
Childhood social class			−0.301**	−0.270**
			(0.103)	(0.104)
			−0.050	*−0.044*
Sex			0.824***	1.355***
			(0.179)	(0.183)
			0.075	*0.121*
BMI at 16				0.834***
				(0.034)
				0.424
Constant	31.001	30.892	18.109	7.508
	(0.526)	(0.654)	(1.210)	(1.296)
R^2	0.009	0.014	0.074	0.227
Number of cases	5,558	4,617	3,630	3,026

Main entries are unstandardized regression coefficients. Entries in parentheses are standard errors. Entries in italics are standardized regression coefficients.
*$P < 0.05$;
**$P < 0.01$;
***$P < 0.001$ (two-tailed).

Anyone who blindly acts on this evolutionarily familiar preference for high-calorie foods today, in industrial and postindustrial societies with abundant and relatively inexpensive food, is destined to become overweight and obese. One, therefore, must have an evolutionarily novel value of voluntarily controlling caloric intake, of not preferentially eating sweet and fatty foods (as our ancestors did) in order not to be overweight and obese today, although it has been difficult to estimate the precise causal effect of food consumption and energy surplus on obesity development (20).

At the same time, exercise for its own sake, to control weight and remain healthy, is also evolutionarily novel. Our ancestors were nomadic hunter-gatherers, with high levels of physical activity and subsistence-level nutrition. Therefore, it would not have been necessary for our ancestors to exercise for its own sake. The available evidence suggests that more intelligent individuals are more likely to exercise more frequently than less intelligent individuals (21,22).

The Savanna-IQ Interaction Hypothesis would therefore predict that less intelligent children are more likely to grow up to be overweight and obese than more intelligent children through both their food consumption and exercise activity. It further posits that the effect of childhood intelligence on adult obesity is direct and independent of its effect through education or income. Consistent with the prediction of the Hypothesis, Teasdale et al. (23) report that, among a sample of 26,274 young Danish men, intelligence and body-mass index (BMI) are significantly negatively correlated even net of education.

In this article, I will test the prediction of the Hypothesis with a large, nationally representative, and prospectively longitudinal sample from NCDS. I will demonstrate that Chandola et al.'s earlier conclusion with the NCDS data that education entirely mediates the effect of childhood intelligence on adult obesity does not hold if one uses a more comprehensive measure of childhood general intelligence available in the data. I will instead suggest that childhood intelligence has a direct effect on adult obesity and weight gain net of education and other confounds.

Methods and Procedures

Participants and study design

The NCDS is a large-scale prospectively longitudinal study, which has followed a *population* of British respondents since birth for more than half a century. The study includes all babies ($n = 17,419$) born in Great Britain (England, Wales, and Scotland) during 1 week (03-09 March 1958). The respondents are subsequently reinterviewed in 1965 (Sweep 1 at Age 7; $n = 15,496$), in 1969 (Sweep 2 at Age 11; $n = 18,285$), in 1974 (Sweep 3 at Age 16; $n = 14,469$), in 1981 (Sweep 4 at Age 23; $n = 12, 537$), in 1991 (Sweep 5 at Age 33; $n = 11,469$), in 1999-2000 (Sweep 6 at Age 41-42; $n = 11,419$), in 2004-2005 (Sweep 7 at Age 46-47; $n = 9,534$), and in 2008-2009 (Sweep 8 at Age 50-51; $n = 9,790$). There are more respondents in Sweep 2 than in the original sample (Sweep 0), because Sweep 2 sample includes eligible children who were in the country in 1969 but not in 1958. In each sweep, personal interviews and questionnaires are administered to the respondents, to their mothers, teachers, and doctors during childhood, and to their partners and children in adulthood. Virtually all (97.8%) of the NCDS respondents are Caucasian. Because I use measures from Sweeps 0-4 and 8, it reduces the sample to only those respondents who participated in all of these sweeps. Listwise deletion for missing data further reduces the sample size in any given regression analysis.

Measures

Dependent variables: Adult BMI and obesity. Respondents' height and weight are measured at Sweeps 1-8, except for Sweep 7. In Sweeps 1-3, they are measured by a physician. In Sweeps 4, 6, and 8, they are self-reported. In Sweep 5, they are measured by an interviewer. From the recorded height and weight, I can compute the respondent's BMI and their obesity status (1 if BMI > 30).

For measures of adult BMI and obesity, I use the Sweep 8 measures of height and weight at Age 51, because they are the latest measures available in NCDS. Because Sweep 8 measures are self-reports, they are subject to systematic reporting errors (24,25). In NCDS, however, the self-reported Sweep 8 measures are very highly correlated with the interviewer-measured Sweep 5 measures (height: $r = 0.898$, $P < 0.001$, $n = 8,439$; weight: $r = 0.808$, $P < 0.001$, $n =$

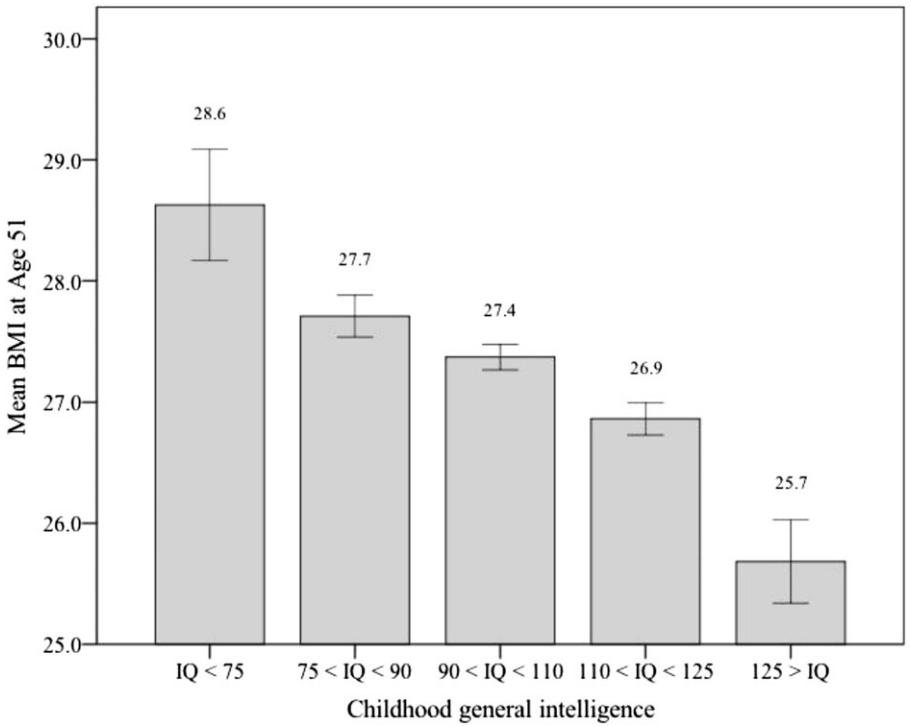

FIGURE 1 Bivariate association between childhood general intelligence and mean BMI at Age 51. Error bars represent standard errors.

8,072). As a result, all of my substantive conclusions reported later remain the same if I use the Sweep 5 measures at Age 33 instead of the Sweep 8 measures at Age 51.

Independent variable: Childhood general intelligence. The NCDS respondents take multiple intelligence tests at Ages 7, 11, and 16. At Age 7, the respondents take four cognitive tests: Copying Designs Test (the respondent is instructed to copy geometric shapes and a sentence as carefully as possible), Draw-a-Man Test (the respondent is instructed to ''make a picture of a man''), Southgate Group Reading Test (the respondent identifies a word out of five candidates), and Problem Arithmetic Test. At Age 11, they take five cognitive tests: Verbal General Ability Test (Raven's-type logic test using words), Nonverbal General Ability Test (Raven's-type logic test using shapes), Reading Comprehension Test, Mathematical Test, and Copying Designs Test. At Age 16, they take two cognitive tests: Reading Comprehension Test and Mathematics Comprehension Test. I first perform a factor analysis at each age to compute their general intelligence score for each age. All cognitive test scores at each age load only on one latent factor, with reasonably high factor loadings (Age 7: Copying Designs = 0.671, Draw-a-Man = 0.696, Southgate Group Reading = 0.780, and Problem Arithmetic = 0.762, Cronbach's α = 0.596; Age 11: Verbal General Ability = 0.920, Nonverbal General Ability = 0.885, Reading Comprehension = 0.864, Mathematical = 0.903, and Copying Designs = 0.486, Cronbach's α = 0.867; and Age 16: Reading Comprehension = 0.909, and Mathematics Comprehension = 0.909, Cronbach's α = 0.791). The latent general intelligence scores at each age are converted into the standard IQ metric, with a mean of 100 and a standard deviation of 15. Then, I perform a second-order factor analysis with the IQ scores at three different ages to compute the overall childhood general intelligence score. The three IQ scores load only on one latest factor with very high factor loadings (Age 7 = 0.867; Age 11 = 0.947; Age 16 = 0.919; Cronbach's α = 0.897). I use the childhood general intelligence score in the standard IQ metric as my main independent variable.

Independent variable: Education. Educational attainment is measured at Age 23 by a six-point ordinal scale, reflecting the highly complex system of examinations, qualifications, and certifications in the British school system: 0 = no qualifications; 1 = Certificate of Secondary Education 2-5/NVQ (National Vocational Qualifications), 1; 2 = O (Ordinary) levels/NVQ, 2; 3 = A (Advanced) levels/NVQ, 3; 4 = higher qualification/NVQ, 4, and; 5 = degree/NVQ, 5-6. It is important to note that this is the identical measure of education that Chandola et al. (12) use when they conclude that education entirely medicates the effect of childhood intelligence on adult obesity.

Control variables. In my multiple regression analyses, I control for the following variables: earnings (measured at Age 51 in 1K GBP); mother's BMI; father's BMI (both measured at Age 11 and intended to control for the respondent's genetic tendency toward obesity); childhood social class (measured at birth by the father's occupational class: 0 = unemployed, dead, retired, or no father present; 1 = unskilled; 2 = semiskilled; 3 = skilled; 4 = white-collar; and 5 = professional); and sex (0 = female and 1 = male; measured at birth).

Statistical analysis. Adult BMI at Age 51 is regressed on childhood general intelligence, education, and the control variables in a stepwise manner in OLS regression. Adult obesity at Age 51 (1 if

TABLE 2 The effect of childhood general intelligence on adult obesity at Age 51

	(1)	(2)	(3)	(4)
Childhood general intelligence	−0.018***	−0.011***	−0.010**	−0.008*
	(0.002)	(0.003)	(0.004)	(0.004)
	0.763	*0.848*	*0.861*	*0.887*
Education		−0.132***	−0.124***	−0.122**
		(0.032)	(0.037)	(0.001)
		0.828	*0.838*	*0.840*
Earnings			0.002	0.002
			(0.001)	(0.001)
			1.111	*1.111*
Mother's BMI			0.073***	0.035**
			(0.010)	(0.012)
			1.337	*1.149*
Father's BMI			0.080***	0.027
			(0.013)	(0.015)
			1.287	*1.089*
Childhood social class			−0.093*	−0.098
			(0.046)	(0.053)
			0.909	*0.904*
Sex			0.046	0.254**
			(0.080)	(0.094)
			1.023	*1.135*
BMI at 16				0.283***
				(0.018)
				2.267
Constant	0.645	0.327	−3.340	−7.278
	(0.222)	(0.278)	(0.531)	(0.675)
Cox & Snell R^2	0.012	0.017	0.050	0.134
Number of cases	5,558	4,617	3,630	3,026

Main entries are unstandardized regression coefficients. Entries in parentheses are standard errors. Entries in italics are effects on odds associated with one standard deviation increase.
*$P < 0.05$;
**$P < 0.01$;
***$P < 0.001$ (two-tailed).

BMI > 30) is similarly regressed on the same set of variables in a stepwise manner in binary logistic regression. For the purpose of graphic presentation only, mean adult BMI and mean proportion obese are computed for five ordinal categories of cognitive classes (IQ < 75, 75 < IQ < 90, 90 < IQ < 110, 110 < IQ < 125, IQ > 125).

Results

Table 1 presents the results of OLS regression of adult BMI at Age 51. Column (1) shows that childhood general intelligence before Age 16 is strongly and statistically significantly ($P < 0.001$) negatively correlated with adult BMI at Age 51. One standard deviation (15 IQ points) increase in childhood general intelligence decreases adult BMI by 0.555 points, which is more than a tenth of BMI points separating normal weight and obesity. Column (2) further shows that the negative correlation between childhood intelligence and adult BMI remains strong and statistically significant ($P < 0.001$) even when educational attainment is controlled for. Education only very slightly mediates the effect of childhood intelligence on adult BMI.

Column (3) shows that the negative association between childhood general intelligence and adult BMI remains large and statistically significant even when earnings, mother's BMI, father's BMI, childhood social class, and respondent's sex are statistically controlled. In sharp contrast, net of these further control variables, educational attainment is no longer significantly correlated with adult BMI.

The statistical model presented in Column (4) further controls for the respondent's BMI at Age 16. The dependent variable in this model is essentially adult weight gain from Age 16 to Age 51. The results show that more intelligent children gain less weight in their adulthood than less intelligent children. Childhood general intelligence has a significantly ($P < 0.05$) negative effect on adult BMI at Age 51 net of BMI at Age 16. Earnings have no association at all with either adult BMI or adult weight change.

Figure 1 presents the bivariate association between childhood general intelligence, represented by five ordinal categories, and the mean adult BMI at Age 51. It shows that there is a monotonic negative association between childhood general intelligence and adult BMI. Children whose IQs are above 125 have a mean adult BMI at Age 51 nearly three points lower than those whose IQs are below 75 (25.7 vs. 28.6).

Table 2 presents the results of binary logistic regression of adult obesity at Age 51. Column (1) shows that, as with adult BMI, childhood general intelligence is significantly ($P < 0.001$) negatively associated with adult obesity at Age 51. One standard deviation increase in childhood general intelligence decreases the odds of adult obesity by 24%. Column (2) further shows that educational attainment only very slightly mediates the effect of childhood general intelligence on adult obesity. Net of education, childhood general intelligence still significantly decreases the likelihood of adult obesity. One standard deviation increase in childhood general intelligence or educational attainment, net of each other, has roughly the same effect on adult obesity (15% and 18% decrease in the odds of obesity, respectively).

Column (3) shows that, even net of earnings, mother's BMI, father's BMI, childhood social class, and sex, childhood general intelligence has a significantly negative effect on the likelihood of adult obesity. Net of these control variables, one standard deviation increase in childhood general intelligence still decreases the odds of adult obesity by 14%. Education has a comparable negative effect on the odds of adult obesity (one standard deviation increase decreasing the odds by 16%). It is interesting to note that, although men have significantly higher BMI than women, they are no more likely to be obese.

Finally, the statistical model presented in Column (4) controls for BMI at Age 16 and thus transforms the dependent variable into the likelihood of becoming obese after Age 16. Naturally, BMI at Age 16 has a very large and statistically significantly ($P < 0.001$) positive effect on adult obesity. One standard deviation increase in BMI at Age 16 (2.891) more than doubles the odds of adult obesity at Age 51. Nevertheless, net of BMI at Age 16 and all the other control variables, childhood general intelligence still significantly decreases the likelihood of adult obesity at Age 51. One standard deviation increase in childhood general intelligence decreases the

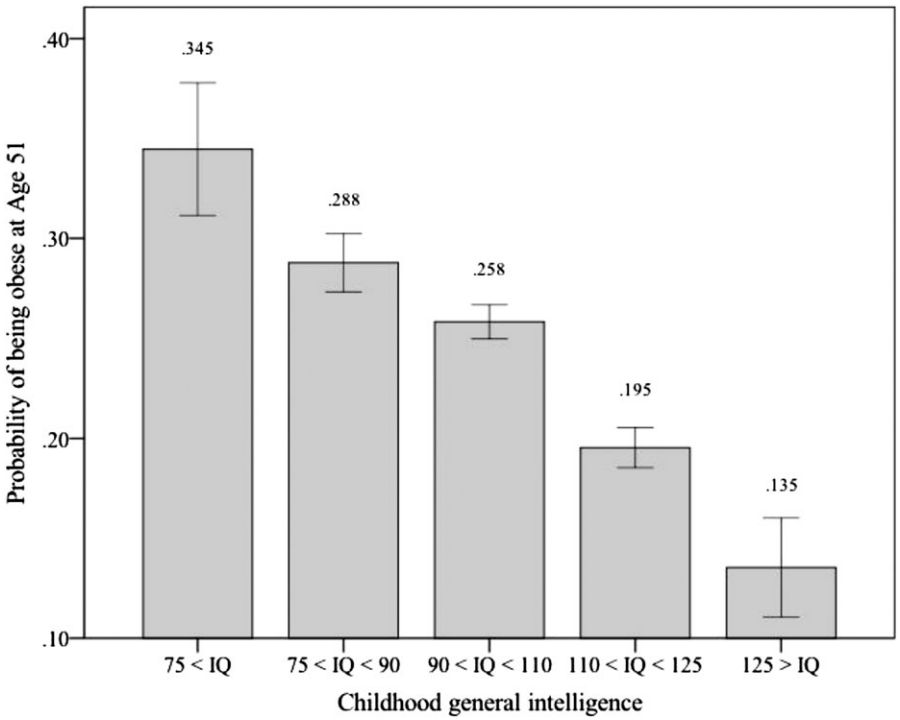

FIGURE 2 Bivariate association between childhood general intelligence and the probability of being obese at Age 51. Error bars represent standard errors.

odds of adult obesity at Age 51 by 11%. Once adolescent BMI is controlled, men are significantly more likely to be obese than women; they have 29% higher odds of being obese.

Figure 2 presents the bivariate association between childhood general intelligence and the probability of being obese at Age 51. It shows that there is a monotonic negative association between childhood general intelligence and the probability of being obese. Children whose IQs are below 75 are more than two and half times as likely to be obese at Age 51 as those whose IQs are above 125.

One may argue that my measure of childhood intelligence, extracted from cognitive tests administered at Ages 7, 11, and 16, may at least partially reflect (and be causally subsequent to) formal education. It may, therefore, be partly measuring educational attainment rather than innate intelligence. As Table 3 shows, however, using a measure of childhood intelligence computed only from the four cognitive tests administered at Age 7, before the respondents have had much exposure to formal education, does not alter any of my substantive conclusions. In fact, the effect of childhood general intelligence at Age 7 on adult BMI and obesity is much stronger than that of childhood general intelligence computed from Ages 7, 11, and 16. It is remarkable that general intelligence measured at Age 7 still has a statistically significant effect on adult BMI and obesity 44 years later.

Discussion

One possible objection to my analyses earlier is that it is not ''fair'' to compare the relative effects of childhood intelligence measured on a continuous scale and education measured on a six-point ordinal scale, because, *ceteris paribus*, a continuous variable is expected to account for greater variance in a dependent variable than an ordinal variable. In NCDS, education is measured continuously only once, in Sweep 6, as the age at which the respondent left full-time continuous education. Despite the fact that the continuous measure of education is not extremely highly correlated with the ordinal measure (r= 0.491), all my substantive conclusions remain identical if I use the continuous measure.

One alternative explanation for the significantly negative association between childhood intelligence and obesity is the ''general fitness factor'' approach (26), which suggests that both higher intelligence and health (including the ability to stay within the normal weight range) reflect underlying genetic quality. In this context, it is interesting to note that general intelligence is significantly *positively* associated with BMI at Age 7 ($r = 0.044$, $n = 8{,}331$, $P < 0.001$) and at Age 11 ($r = 0.040$, $n = 7{,}780$, $P < 0.001$), and it is not correlated with BMI at Age 16 ($r = -0.016$, $n = 7{,}177$, *ns*). In other words, general intelligence is significantly *negatively* associated with BMI only in adulthood (Age 23: $r = -0.124$, $n = 7{,}279$, $P < 0.001$; Age 33: $r = -0.109$, $n = 6{,}669$, $P < 0.001$; Age 42: $r = -0.108$, $n = 6{,}537$, $P < 0.001$; Age 51: $r = -0.096$, $n = 5{,}558$, $P < 0.001$), when individuals have complete control over what to eat and not to eat, as predicted by the Hypothesis.

The Hypothesis explains the significant association between childhood general intelligence and adult obesity with more intelligent individuals' evolutionarily novel values to avoid sweet and fatty

TABLE 3 The effect of general intelligence at Age 7 on adult BMI and obesity at Age 51

	BMI	Obesity
General intelligence at 7	−0.026***	−0.011**
	(0.007)	(0.003)
	−0.064	*0.848*
Education	−0.098	−0.121***
	(0.067)	(0.035)
	−0.025	*0.841*
Earnings	0.003	0.001
	(0.002)	(0.001)
	0.022	*1.054*
Mother's BMI	0.088***	0.032**
	(0.022)	(0.011)
	0.060	*1.136*
Father's BMI	0.093***	0.026
	(0.028)	(0.014)
	0.050	*1.086*
Childhood social class	−0.205*	−0.068
	(0.092)	(0.047)
	−0.034	*0.933*
Sex	1.163***	0.156
	(0.164)	(0.085)
	0.105	*1.081*
BMI at 16	0.818***	0.280***
	(0.030)	(0.017)
	0.425	*2.247*
Constant	9.080	−6.975
	(1.134)	(0.595)
(Cox & Snell) R^2	0.226	0.130
Number of cases	3,706	3,706

Main entries are unstandardized regression coefficients. Entries in parentheses are standard errors. Entries in italics are effects on odds associated with one standard deviation increase.
*$P < 0.05$;
**$P < 0.01$;
***$P < 0.001$ (two-tailed).

foods and to exercise for its own sake. The NCDS data allow me to test both these causal mechanisms directly.

At Age 33, NCDS asks its respondents how often they consume various food items (fried food, fresh fruits, salads, French fries, sweets and chocolates, and cookies) on a six-point Likert scale from 1 = never to 6 = more than once a day. Childhood general intelligence is significantly positively associated with the frequency of consumption of fresh fruits ($r = 0.136$, $n = 6{,}680$, $P < 0.001$) and salads ($r = 0.129$, $n = 6{,}682$, $P < 0.001$) and significantly negatively associated with the frequency of consumption of fried food ($r = -0.135$, $n = 6{,}679$, $P < 0.001$) and French fries ($r = -0.202$, $n = 6{,}673$, $P < 0.001$). At Age 42, NCDS asks about an even larger number of food items (fresh fruits, eggs, salads, cooked vegetables, food fried in vegetable oil, food fried in hard fat, French fries, sweets and chocolates, cookies and cakes, whole-grain bread, other bread, red meat, poultry, fish, and legume) on a seven-point Likert scale from 1 = never to 7 = more than once a day. Childhood general intelligence is significantly positively associated with the frequency of consumption of fresh fruits ($r = 0.202$, $n = 6{,}705$, $P < 0.001$), salads ($r = 0.134$, $n = 6{,}705$, $P < 0.001$), cooked vegetables ($r = .131$, $n = 6{,}706$, $P < 0.001$), food fried in vegetable oil ($r = 0.111$, $n = 6{,}703$, $P < 0.001$), sweets and chocolates ($r = 0.060$, $n = 6{,}706$, $P < 0.001$), cookies and cakes ($r = 0.064$, $n = 6{,}706$, $P < 0.001$), whole-grain bread ($r = 0.158$, $n = 6{,}705$, $P < 0.001$), poultry ($r = 0.044$, $n = 6{,}706$, $P < 0.001$), and fish ($r = 0.123$, $n = 6{,}706$, $P < 0.001$) and significantly negatively associated with the frequency of consumption of French fries ($r = -0.147$, $n = 6{,}706$, $P < 0.001$), other bread ($r = -0.069$, $n = 6{,}706$, $P < 0.001$), and red meat ($r = -0.053$, $n = 6{,}706$, $P < 0.001$). With the exceptions of small positive correlations for sweets and chocolates and cookies and cakes at Age 42, it seems safe to conclude that more intelligent children grow up to consume more healthy foods and avoid sweet and fatty foods in adulthood.

At Ages 33, 42, 47, and 51, the NCDS asks its respondents how frequently they exercise on a seven-point Likert scale from 0 = never to 6 = every day. Childhood general intelligence is significantly positively associated with the frequency of exercises throughout adulthood (Age 33: $r = 0.073$, $n = 6{,}649$, $P < 0.001$; Age 42: $r = 0.061$, $n = 6{,}704$, $P < 0.001$; Age 47: $r = 0.125$, $n = 5{,}734$, $P < 0.001$; Age 51: $r = 0.068$, $n = 5{,}796$, $P < 0.001$). The NCDS data, therefore, support both the underlying mechanisms suggested by the Hypothesis for the association between childhood general intelligence and adult obesity.

Conclusion

The Savanna-IQ Interaction Hypothesis (13,14) suggests that more intelligent children are more likely to grow up to acquire and espouse evolutionarily novel preferences and values that our ancestors did not have. Voluntary control of caloric intake and exercise for its own sake are among such evolutionarily novel values, as the human mind is designed to crave sweet and fatty foods that contain higher calories and it was not necessary for our ancestors to exercise. Such evolutionarily familiar cravings for sweet and fatty foods allowed our ancestors to live longer, stay healthier, and reproduce more successfully in the ancestral environment with scarce food.

The Hypothesis would therefore predict that less intelligent children are more likely to grow up to have higher BMI and be obese. It further predicts that the effect of childhood general intelligence on adult obesity is direct and unmediated by education or income. The analysis of a large, nationally representative, and prospectively longitudinal sample from the NCDS strongly supports the prediction of the Hypothesis. Contrary to the earlier findings by Halkjær et al. (11), Chandola et al. (12), and the comprehensive review by Yu et al. (10), education appears to mediate the effect of childhood general intelligence on adult obesity only very slightly. However, given that the NCDS, while prospectively longitudinal with a large, nationally representative sample, is nonetheless correlational data, I cannot entirely rule out the possibility that some antecedent genetic or environmental factor can simultaneously account for the associations between childhood general intelligence, education, and adult obesity (27,28).

I hasten to add that the effect of childhood intelligence on adult BMI and obesity that I find in my analyses is relatively small, and it is much smaller than the effects of mother's BMI, father's BMI, and

BMI at Age 16, all of which are intended to measure the respondent's genetic tendency toward obesity and their early family environment. (However, the effect of general intelligence at Age 7 has a slightly larger effect on adult BMI and obesity than parental BMI; see Table 3.) There is little doubt that these factors exert strong influences on obesity. On the other hand, parental BMI may itself be a consequence of parental general intelligence, which the parents then pass on to their children. Careful behavior genetic studies are necessary to tease apart the influences of general intelligence, genes, and family environment on obesity. O

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Disclosure

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