CURRENT OPINION

Intelligence and obesity: which way does the causal direction go?

Satoshi Kanazawa

Purpose of review
The negative association between intelligence and obesity has been well established, but the direction of causality is unclear. The present review surveys the recent studies on the topic with both cross-sectional and longitudinal data in an attempt to establish causality.

Recent findings
Most studies in the area employ cross-sectional data and conclude (without empirical justification) that obesity causes intellectual impairment. The few studies that employ prospectively longitudinal data, however, uniformly conclude that lower intelligence leads to BMI gains and obesity. A close examination of three such studies, from three different nations (Sweden, New Zealand, and the UK), leaves little doubt that the causality runs from low intelligence to obesity.

Summary
The conclusion in previous studies that obesity impairs cognitive function stems from improper interpretation of a negative association between intelligence and obesity from cross-sectional studies. Results from the analyses of high-quality, population-based, prospectively longitudinal data firmly establish that low intelligence increases the chances of obesity.

Keywords
cross-sectional data, Dunedin Multidisciplinary Health and Development Study, National Child Development Study, prospectively longitudinal data, Westmannia Cardiovascular Risk Factors Study

INTRODUCTION

Researchers have long known that intelligence is correlated with a large number of health outcomes, including obesity and its comorbid conditions [1,2]. But they do not yet know why [3,4]. On the association between intelligence and obesity specifically, and the direction of causality between them, cross-sectional studies and prospectively longitudinal studies reach opposite conclusions. Cross-sectional studies usually conclude that obesity impairs cognitive functions, whereas prospectively longitudinal studies usually demonstrate that childhood intelligence strongly influences adult obesity.

CROSS-SECTIONAL STUDIES

During the past 18 months covered by the present review, researchers have continued to compile data that demonstrate the negative association between intelligence and obesity in different populations and subpopulations. For example, a study of Chilean preschool children (ages 4–5; $n = 215$) finds that, net of age, sex, social-emotional well-being, and 6-min walk test score, BMI z-score has a significantly ($P = 0.012$) negative association with intelligence, measured by the Wechsler Preschool and Primary Scale of Intelligence, among the children from mid-low SES families ($n = 48$), although not among those from low ($n = 83$) or very-low ($n = 84$) SES families [5]. A study of the elderly in Spain ($n = 1949$) finds that overweight and obese individuals perform significantly ($P < 0.001$) worse than normal-weight individuals on the 37-item version of the Mini-Mental State Examination [6]. In a study of adolescents (ages 14–20), those without metabolic syndrome ($\mathrm{BMI} = 27.09 \pm 9.59$; $n = 62$) have marginally significantly ($P = 0.09$) higher intelligence, measured by the Wechsler Abbreviated Scale of Intelligence, than those with metabolic syndrome

Department of Management, London School of Economics and Political Science, London, UK

Correspondence to Satoshi Kanazawa, Managerial Economics and Strategy Group, Department of Management, London School of Economics and Political Science, Houghton Street, London WC2A 2AE, UK. Tel: +44 (0)20 7955 7297; e-mail: S.Kanazawa@lse.ac.uk

Curr Opin Endocrinol Diabetes Obes 2014, 21:339–344

DOI:10.1097/MED.0000000000000091

KEY POINTS

- Most cross-sectional studies conclude that obesity impairs cognitive functions, whereas most prospectively longitudinal studies conclude that low intelligence leads to obesity and weight gain.
- Careful examinations of prospectively longitudinal studies with large population samples from three different nations (Sweden, New Zealand, and the UK) clearly show that causal direction goes from low intelligence to obesity and weight gain.
- There is no credible scientific evidence that obesity impairs cognitive functions, whereas there are both credible scientific theory and evidence that lower childhood intelligence leads to obesity and weight gain in adulthood.

(BMI = 38.43 ± 7.17; n = 49) (105.95 ± 12.35 vs. 102.00 ± 11.63) [7].

The negative association between intelligence and BMI or obesity appears so strong that it exists even in some extreme clinical populations with a limited range of BMI. For example, researchers find that, among 141 adolescents (ages 14–17) with severe obesity (BMI > 40 or BMI > 35 with serious comorbid conditions), BMI is significantly negatively correlated with full-scale intelligence, measured by the Wechsler Abbreviated Scale of Intelligence ($r = -0.250$, $P = 0.005$) and its vocabulary subtest ($r = -0.245$, $P = 0.006$), though not its matrix reasoning subtest ($r = -0.126$, *ns*) [8]. Given, the small sample and the extremely limited variance in BMI in the sample, such significantly negative correlations are remarkable. Researchers also find that, in a sample of Chinese outpatients with schizophrenia, recruited from 10 sites throughout China ($n = 896$; ages 18–50), BMI is significantly negatively correlated with intelligence, measured by five different cognitive tests (partial $r = -0.082$, $P = 0.018$, net of age and sex) [9]. Once again, given both that more than half (53.7%) of the participants are either overweight or obese, and participants with severe obesity (BMI > 45) are excluded from the study, thereby restricting the range and variance on BMI, such a negative correlation is notable. An analysis of euthymic individuals with Type I or II bipolar disorder shows that overweight or obese individuals ($n = 48$) are significantly less intelligent than their normal-weight controls ($n = 19$), measured by the Digit Symbol Substitution Test ($P = 0.091$) or the Verbal Fluency Test ($P = 0.013$) [10]. The group differences in intelligence measured by most of the other tests go in the same direction but are statistically non-significant because of small sample sizes.

The findings on the association between intelligence and obesity have not been uniform, however. A small study of adolescents (ages 14–18) seeking laparoscopic adjustable gastric banding surgery (BMI > 40) finds that their BMI is not at all associated with their intelligence, measured either by the Wechsler Intelligence Scale for Children (if the study participant is younger than 16) or the Wechsler Adult Intelligence Scale (if the study participant is 16 or older), despite the fact that 20.6% of the study participants had intelligence quotients (IQs) below 80 and none had IQs above 120 [11]. However, given the extremely small sample size ($n = 29$), caution is necessary in interpreting their null finding. At least one study of eighth-graders and community college and university students with relatively large samples finds that, even though overweight and obese students receive lower grades, they do not differ from normal-weight students in their intelligence, once sex, ethnicity, geographic location, socioeconomic status, conscientiousness, and their emotional well-being are statistically controlled [12]. The authors' conclusion is that overweight and obese individuals are 'just as smart but not as successful'. Another study of middle-aged individuals (ages 40–65; $n = 184$) claims to find no significant association between BMI and 'overall cognitive scores' on a large number of cognitive tests [13]. However, their multiple regression analysis controls for verbal intelligence, in addition to age and sex. Given that all measures of intelligence are very highly correlated with each other [14], their inclusion of verbal intelligence as a control in a multiple regression equation predicting cognitive function entirely invalidates their conclusion.

A study of intelligence and somatotypes among older Chilean school children (ages 11–16; $n = 1015$) finds that both endomorphy ($r = 0.074$, $P = 0.02$) and ectomorphy ($r = 0.067$, $P < .05$) are significantly correlated with intelligence, measured by Raven's Progressive Matrices, whereas mesomorphy is not [15]. A study of school children (ages 6–12; $n = 6746$) from four Southeast Asian nations (Indonesia, Malaysia, Thailand, and Vietnam) shows that the severely obese students are significantly more likely to have lower intelligence (measured by Raven's Progressive Matrices or Test of Nonverbal Intelligence), even though standardized BMI for age and intelligence are in general positively associated [16]. These studies suggest that the association between intelligence and obesity or BMI may not exist or may even be positive in economically developing nations where malnutrition may still be prevalent, as it indeed was in the post-World War II UK (D.W. Belsky, *Psychologist*, 2014, personal communication) [17■■].

Remarkably, all of the authors of the cross-sectional studies summarized above, who find a significantly negative correlation between intelligence and obesity, *without a single exception* interpret their findings to mean that *obesity impairs cognitive function*. In other words, they unquestioningly assume that the causal direction goes from obesity to lower intelligence. Such an uncritical conclusion might potentially reflect the unquestioned and strongly held assumption – among both scientists and civilians alike – that intelligence is the ultimate hallmark of human worth and thus that, because all humans, be they obese or otherwise, are equally worthy human beings, they must necessarily have on average equal intelligence to begin with [14]. Of course, the specification of the causal order is strictly impossible with cross-sectional data and instead requires either experimental data (which are probably unfeasible in obesity research because of ethical concerns) or prospectively longitudinal data. What then do prospectively longitudinal studies of intelligence and obesity show?

PROSPECTIVELY LONGITUDINAL STUDIES

In clear contrast to the conclusions reached by all the cross-sectional studies reviewed above, all the prospectively longitudinal studies on intelligence and obesity published in the last 18 months conclude that the causal direction goes from lower intelligence to obesity.

Suggestive of the fact that obesity or diabetes may not have a causal impact on intelligence, a prospectively longitudinal study of Swedish men in a large population sample ($n = 723\,755$) from a large number of families ($n = 579\,857$) shows that, in between-family analyses, comparing men from different families, men whose mother had diabetes during their pregnancy have significantly lower intelligence than those whose mother did not have diabetes (by 1.36 IQ points) [18▪]. However, the significant difference disappears in within-family analyses, comparing brothers from the same families, suggesting that mothers' diabetes/obesity and men's intelligence may have a common factor (mothers' intelligence). On the other hand, a prospectively longitudinal study of British citizens ($n = 2268$) shows that maternal and paternal intelligence does *not* predict children's standardized BMI when they are on average 7–9 years old [19], but this may be largely because of the fact that childhood general intelligence is only weakly ($h^2 \sim 0.4$) heritable, compared with adult intelligence ($h^2 \sim 0.8$) [14], and maternal and paternal intelligence may not be strongly correlated with children's intelligence at these young ages.

An analysis of the prospectively longitudinal National Survey of Health and Development ($n = 2195$) shows that, although BMI is significantly negatively correlated with cognitive function at age 53, measured by verbal memory, verbal fluency, and letter cancellation, the significant correlations entirely disappear once childhood intelligence (measured at age 8) is statistically controlled, except in one case, in which BMI is significantly positively associated with verbal memory for men [20]. A path analysis of a Scottish sample in the Lothian Birth Cohort 1936 ($n = 1091$) shows that childhood intelligence, measured at age 11 with the Moray House Test No. 12, has a significantly positive effect (path coefficient $= 0.055$) on adult socioeconomic status (measured by education, occupational social class, and economic deprivation of the area of residence), which in turn has a significantly negative effect (path coefficient $= -0.17$) on BMI at an advanced age (68–71), which implies that childhood intelligence has an indirect effect of -0.09 on adult BMI 6 decades later [21]. (The authors' path model does not include a direct effect of childhood intelligence on adult BMI unmediated by SES.)

Three studies, all conducted with high-quality prospectively longitudinal data with large population samples from three different nations, deserve particular attention.

SWEDEN

Sweden had universal male conscription until 2006. At signup, at age 18, all Swedish men underwent an identical set of physical and mental examinations, including IQ tests, consisting of logic/inductive ability, verbal ability, spatiovisual perception, and technical/mechanical ability. It means that, until recently, the Swedish government had information on the intelligence of virtually all male citizens.

In the county of Västmanland, starting in 1989, all men and women at age 40 are given a free health examination at primary healthcare centers for the presence of cardiovascular disease risk factors. The examination includes measurement of height and weight. With the use of the Swedish personal identity numbers, researchers are able to link the BMI of 5286 males from Västmanland to their intelligence measured 22 years earlier at conscription signup [22].

Their results show a clear and monotonically negative association between intelligence at 18 and BMI at 40. Individuals with IQs below 74 at 18 have BMI of 26.59 at 40, whereas those with IQs above 126 have BMI of 25.75 ($P < 0.001$). Similarly, there is a clear and monotonically negative association between intelligence at 18 and the BMI change from

18 to 40. Individuals with IQs below 74 gain 5.19 in BMI in 22 years, whereas those with IQs above 126 gain 3.73 ($P < 0.001$). Their conclusion remains identical even when they control for systolic and diastolic blood pressure, resting pulse rate, birth place, birth year, and education at conscription. Their results from a large population sample of Swedish men make it clear that it is adolescent intelligence that influences BMI in middle age, not the other way around.

NEW ZEALAND

The Dunedin Multidisciplinary Health and Development Study is a prospectively longitudinal and ongoing study of health and behavior in a complete birth cohort (all individuals born in Dunedin, New Zealand, between April 1972 and March 1973). The cohort members' intelligence was measured at age 3 with the Peabody Picture Vocabulary Test, at ages 7, 9, and 11 with the Wechsler Intelligence Scale for Children-Revised, and again at age 38 with the Wechsler Adult Intelligence Scale-IV. The sample for the analysis includes 913 cohort members with complete data [23▪▪].

The researchers show that, consistent with predominant findings from the cross-sectional studies reviewed above, cohort members who are obese at 38 have significantly lower intelligence than those who are nonobese (96.93 vs. 101.23, $P = 0.001$). However, this difference disappears entirely once childhood intelligence measured at 7–11 is statistically controlled. In fact, there is already a significant difference in intelligence at age 3 between those who eventually become obese before 38 and those who never do (97.8 vs. 100.91, $P = 0.006$). Consistent with earlier studies in intelligence research [24], the analysis shows that individuals' intelligence stays virtually identical after age 11.

Their analysis clearly demonstrates that, contrary to the conclusions reached by the authors of the cross-sectional studies, obesity does not at all impair cognitive function. The direction of causality clearly goes from childhood intelligence to adult obesity, not the other way around. Obesity does not decrease intelligence; less intelligent individuals become obese.

UNITED KINGDOM

The National Child Development Study is a prospectively longitudinal and ongoing study that includes all babies born during 1 week (3–9 March 1958) in Great Britain (England, Wales, and Scotland), who have been followed for more than half a century. The participants' childhood intelligence was measured by 11 different cognitive tests administered at three different ages (7, 11, and 16). Their adult BMI was measured at the latest sweep at age 51.

The analysis ($n = 3026$) shows that childhood intelligence has a direct negative effect on BMI and obesity at 51, and its effect is not at all mediated by education or earnings [17▪▪]. Childhood intelligence has a significantly negative effect on BMI at 51 even net of education, earnings, mother's BMI, father's BMI, childhood social class, sex, and even

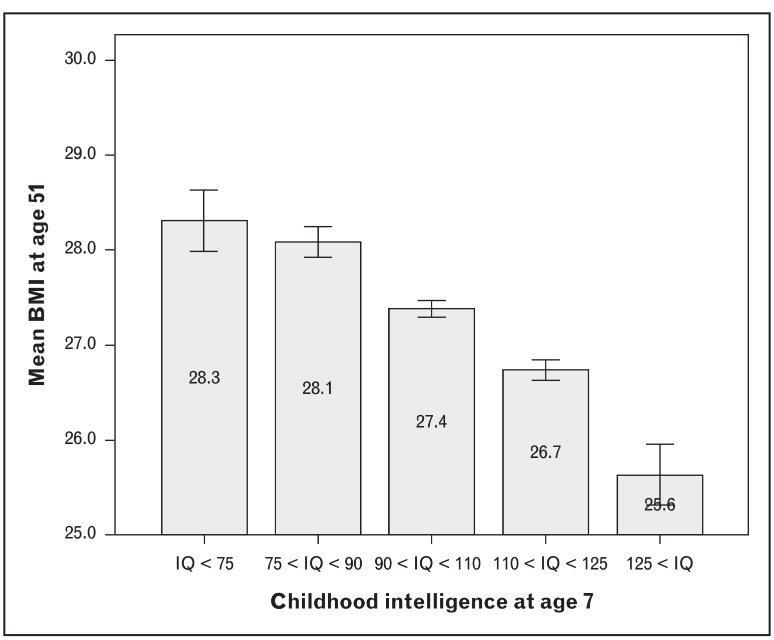

FIGURE 1. The association between childhood intelligence at age 7 and BMI at age 51. National Child Development Study (UK).

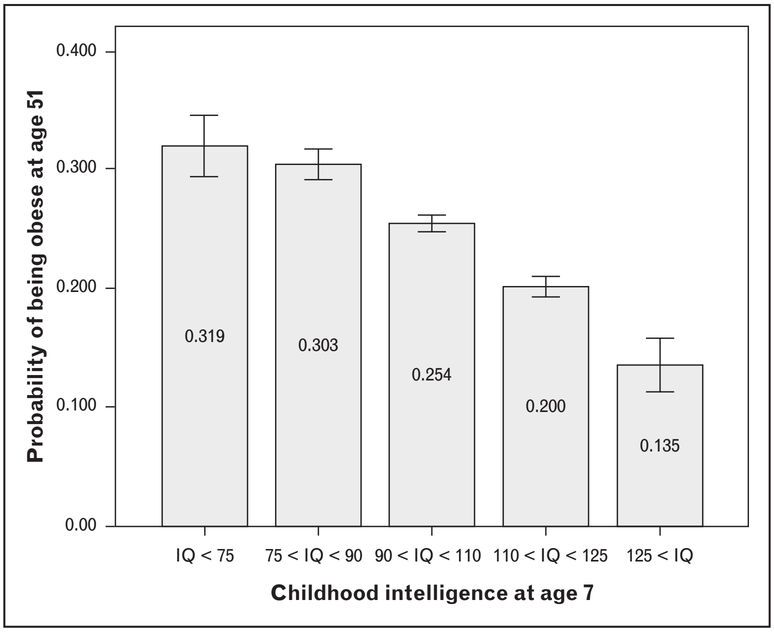

FIGURE 2. The association between childhood intelligence at age 7 and probability of being obese at age 51. National Child Development Study (UK).

BMI at age 16. In other words, childhood intelligence has a direct effect on both adult BMI at 51 and BMI change from 16 to 51.

Similar to the New Zealand study, the UK study shows that childhood intelligence measured as early as age 7 strongly predicts adult BMI and obesity at 51. As Fig. 1 shows, childhood intelligence at age 7 is strongly and monotonically negatively associated with BMI more than 40 years later. Individuals whose IQs at 7 are above 125 have BMIs on average nearly three points lower than their classmates whose IQs are below 75 (25.6 vs. 28.3). Similarly, Fig. 2 shows that childhood intelligence at 7 is strongly and monotonically negatively associated with the probability of being obese more than 40 years later. Individuals whose IQs at 7 are above 125 have 13.5% chance of being obese at 51, whereas those whose IQs at 7 are below 75 have 31.9% chance. In the National Child Development Study data, no one is obese at age 7. Figures 1 and 2 make it abundantly clear, once again, that it is low intelligence in childhood that causes obesity later in life.

CONCLUSION

The close examination of prospectively longitudinal studies on the association between intelligence and obesity, particularly those that use large population samples in Sweden, New Zealand, and the UK, makes it clear that the conclusion unanimously reached in cross-sectional studies that obesity impairs cognitive function is incorrect. As is clear particularly in the New Zealand study, obesity does not change intelligence. The direction of causality goes from intelligence to obesity. Less intelligent individuals are more likely to gain weight and become obese throughout adulthood than more intelligent individuals. The strongly negative effect of intelligence on obesity explains the association between the two.

Acknowledgements

I thank Daniel W. Belsky and Terrie E. Moffitt for helpful conversations on the topics covered in the review.

Conflicts of interest

There are no conflicts of interest.

REFERENCES AND RECOMMENDED READING

Papers of particular interest, published within the annual period of review, have been highlighted as:

- ▪ of special interest
- ▪▪ of outstanding interest

1. Yu ZB, Han SP, Cao XG, Guo XR. Intelligence in relation to obesity: a systematic review and meta-analysis. Obes Rev 2009; 11:656–670.
2. Cawley J, Spiess CK. Obesity and skill attainment in early childhood. Econ Hum Biol 2008; 6:388–397.
3. Deary IJ. Why do intelligent people live longer? Nature 2008; 456:175–176.
4. Gottfredson LS, Deary IJ. Intelligence predicts health and longevity, but why? Curr Dir Psychol Sci 2004; 13:1–4.
5. Galván M, Uauy R, López-Rodríguez. Kain J. Association between childhood obesity, cognitive development, physical fitness and social-emotional well-being in a transitional economy. Ann Hum Biol 2014; 41:101–106.
6. Benito-León J, Mitchell AJ, Herńandez-Gallego J, Bermejo-Pareja F. Obesity and impaired cognitive functioning in the elderly: a population-based cross-sectional study (NEDICES). Eur J Neurol 2013; 20:899–906.
7. Yau PL, Castro MG, Tagani A, Tsui WH, *et al.* Obesity and metabolic syndrome and functional and structural brain impairments in adolescence. Pediatrics 2012; 130:e856.
8. Freidl EK, Sysko R, Devlin M, *et al.* School and cognitive functioning problems in adolescent bariatric surgery candidates. Surg Obes Relat Dis 2013; 9:991–996.

9. Guo X, Zhang Z, Wei Q, *et al.* The relationship between obesity and neurocognitive function in Chinese patients with schizophrenia. BMC Psychiatry 2013; 13:109.
10. Yim CY, Soczynska JK, Kennedy SH, *et al.* The effect of overweight/obesity on cognitive function in euthymic individuals with bipolar disorder. Eur Psychiatry 2012; 27:223–228.
11. Phan. T-LT. Curran JL, Datto GA. Evaluation of intelligence in an adolescent bariatric population. Surg Obes Relat Dis 2013; 9:574–579.
12. MacCann C, Roberts RD. Just as smart but not as successful: obese students obtain lower school grades but equivalent test scores to nonobese students. Int J Obes 2013; 37:40–46.
13. Pedersen M, Pedersen KK, Bruunsgaard H, *et al.* Cognitive functions in middle aged individuals are related to metabolic disturbances and aerobic capacity: a cross-sectional study. PLoS One 2012; 7:e51132.
14. Kanazawa S. The intelligence paradox: why the intelligent choice isn't always the smart one. New York: Wiley; 2012.
15. Tapia LU, Lizana PA, Orellana YZ, *et al.* Somatotype and intellectual ability (Raven progressive matrices test) in Chilean school-age children. Nutr Hosp 2013; 28:1552–1557.
16. Sandjaja BKP, Rojroonwasinkul N, Nyugen BKL, Budiman B, *et al.* Relationship between anthropometric indicators and cognitive performance in Southeast Asian school-aged children. Br J Nutr 2013; 110:S57–S64.
17. Kanazawa S. Childhood intelligence and adult obesity. Obesity 2013;
■■ 21:434–440.
The article uses high-quality, prospectively longitudinal data with a large population sample to demonstrate that childhood intelligence before 16 strongly influences BMI and obesity at age 51. It also provides an evolutionary psychological theory on why intelligence affects obesity.
18. Fraser A, Almqvist C, Larsson HL, Långström N, *et al.* Maternal diabetes in
■ pregnancy and offspring cognitive ability: sibling study with 72 775 men from 579 857 families. Diabetologia 2014; 57:102–109.
The article shows that mother's obesity and diabetes have no impact on children's intelligence.
19. Whitley E, Gale CR, Deary IJ, *et al.* Influence of maternal and paternal IQ on offspring health and health behaviours: evidence for some trans-generational associations using the 1958 British Birth Cohort Study. Eur Psychiatry 2013; 28:219–224.
20. Albanese E, Hardy R, Wills A, *et al.* No association between gain in body mass index across the life course and midlife cognitive function and cognitive reserve–the 1946 British Birth Cohort Study. Alzheimers Dement 2012; 8:470–482.
21. Hagger-Johnson G, Mottus. Craig LCA, Starr JM. Pathways from childhood intelligence and socioeconomic status to late-life cardiovascular disease risk. Health Psychol 2012; 4:403–412.
22. Rosenblad A, Nilsson G, Leppert J. Intelligence level in late adolescence is inversely associated with BMI change during 22 years of follow-up: results from the WICTORY study. Eur J Epidemiol 2012; 27:647–655.
23. Belsky DW, Caspi A, Goldman-Mellor S, *et al.* Is obesity associated with a
■■ decline in intelligence quotient during the first half of the life course? Am J Epidemiol 2013; 178:1461–1468.
The article, using high-quality, prospectively longitudinal data from New Zealand, provides the most solid evidence to date that obesity does not impair cognitive functions, and the direction of causality goes from low childhood intelligence to adult obesity.
24. Deary IJ, Whiteman MC, Starr JM, *et al.* The impact of childhood intelligence on later life: following up the Scottish Mental Surveys of 1932 and 1947. J Pers Soc Psychol 2004; 86:130–147.