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ORIGINAL ARTICLE

Childhood IQ in relation to obesity and weight gain in adult life: the National Child Development (1958) Study

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Objective: To examine the relation of childhood intelligence (IQ) test results with obesity in middle age and weight gain across the life course.
Methods: We analysed data from the National Child Development (1958) Study, a prospective cohort study of 17 414 births to parents residing in Great Britain in the late 1950s. Childhood IQ was measured at age 11 years and body mass index (BMI), an indicator of adiposity, was assessed at 16, 23, 33 and 42 years of age. Logistic regression (in which BMI was categorised into obese and non-obese) and structural equation growth curve models (in which BMI was retained as a continuous variable) were used to estimate the relation between childhood IQ and adult obesity, and childhood IQ and weight gain, respectively.
Results: In unadjusted analyses, lower childhood IQ scores were associated with an increased prevalence of adult obesity at age 42 years. This relation was somewhat stronger in women ($OR_{\text{per SD decrease in IQ score}}$ [95% CI]: 1.38 [1.26, 1.50]) than men (1.26 [1.15, 1.38]). This association remains statistically significant after adjusting for childhood characteristics, including socio-economic factors, but was heavily attenuated following control for adult characteristics, particularly education (women: 1.11 [0.99, 1.25]; men: 1.10 [0.98, 1.23]). When weight gain between age 16 and 42 years was the outcome of interest, structural equation modelling revealed that education and dietary characteristics in adult life mediated the association with childhood IQ.
Conclusions: A lower IQ score in childhood is associated with obesity and weight gain in adulthood. In the present study, this relation appears to be largely mediated via educational attainment and the adoption of healthy diets in later life.
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Keywords: child; IQ

Introduction

Findings from a series of population-based prospective cohort studies indicate that childhood intelligence, as assessed using psychometric tests (denoted here as IQ), is inversely associated with several somatic health outcomes ascertained in middle to later life, including coronary heart disease,[1,2] some cancers[3] and premature mortality.[3–8] For the most examined end point, all-cause mortality, the negative gradient with IQ appears to be strong, consistent across studies and independent of early life social conditions.[9] Mechanisms advanced to explain this relation include mediation via established mortality risk factors in adulthood,[4,10–13] such as smoking[14] and obesity.[15–18]

A link between early life IQ and adult smoking levels has been reported, with higher IQ scores related to a decreased risk of smoking initiation,[19,20] and increased rates of cessation.[21] One explanation for these associations is higher IQ-scoring individuals' differential interpretation of, and responses to, health advice;[21] an explanation that is in keeping with a working definition of IQ as the ability to learn, reason and solve problems.[22] By hypothesising similar associations between childhood IQ and other health behaviours, such as physical activity and diet, an inverse relation between childhood IQ and adult obesity would be anticipated.

We identified six studies,[3,23–27] which have examined the relation of IQ with obesity or body mass index (BMI) (Table 1). Although an inverse IQ–obesity/BMI association is evident in most, these reports are subject to a series of

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Table 1 Studies relating early life intelligence with obesity

Study[citation]	*Study description*	*IQ test*	*Outcome*	*Main result*
Danish Military Draft Study[27]	Case–control study (337 obese and 657 controls — all men)	Børge Priens Prøve at 18 years	Obesity (BMI $\geqslant$ 31 kg/m^2)	Cases had lower IQ scores than controls within strata of father's occupational social class
Danish Military Draft Study[23]	Case–control study (1117 obese and 2017 controls – all men)	Børge Priens Prøve at 18 years	Obesity (BMI $\geqslant$ 31 kg/m^2)	No difference in IQ scores between cases and controls within strata of education
Danish Military Draft Study[24]	Cross-sectional study (26 274 men)	Børge Priens Prøve at 18 years	BMI	Inverse relation with BMI; robust to adjustment for subjects' education
Chinese (Nanjing) Primary School study[25]	Cross-sectional study (102 girls and boys)	Wechsler IQ Scale for Children at 6–13 years.	Obesity	Inverse relation; no direct adjustment for socio-economic position, but parental education did not vary across childhood obesity groups.
Danish Military Draft Study[26]	Prospective case–control study (907 obese and 883 controls — all boys)	Børge Priens Prøve at 18 years	Obesity ($\geqslant$ 30 kg/m^2)	Inverse association with weight gain that was lost after adjustment for subjects' education
Scottish Mental Survey (1932)–Midspan Studies Linkage[2]	Prospective cohort study (937 girls and boys)	Version of the Moray House Test No. 12 at 11 years	Measured BMI	Inverse association; no adjustment for covariates

Abbreviation: BMI, body mass index; IQ, intelligence. Studies are arranged in ascending order of publication year.

limitations which complicate data interpretation. Firstly, four[23,24,26,27] of the six identified studies draw on cohorts of Danish military conscripts which are comprised entirely of men. The only two studies[2,25] which sampled men and women do not report sex-specific analyses. Secondly, only one study[2] examined the relation between *childhood* IQ and *adult* obesity but no adjustment was made for covariates. Thirdly, the findings of an inverse relation between IQ and obesity in cross-sectional[24,25] and case–control studies,[23,24,26] the predominant study designs, raise concerns about reverse causality. That is, rather than IQ predicting obesity, the development of this condition with its accompanying social stigma might result in a failure to realise cognitive potential.[23,27] Fourthly, only one study[26] has examined the link between IQ and weight gain, and this was hampered by a small number of study participants.

In this report, we explore the relation of scores on a childhood IQ test with adult obesity separately in men and women using data from a prospective study, the participants in which are well characterised for socio-economic position in early and adult life. Specifically, the purposes of the present analyses are to (i) examine the relation of childhood IQ with adult obesity in mid-life and weight gain across the life course and (ii) assess the influence, if any, of controlling for potential confounding and mediating factors on these associations.

Materials and methods

The National Child Development Study (NCDS) is an ongoing longitudinal study, which takes as its subjects over 17 000 live births occurring to parents residing in Great Britain between 3rd and 9th March 1958.[28] Following the initial 1958 survey, there have been six attempts to follow up members of the birth cohort to monitor their physical, educational and social development.[29] These were conducted in 1965 (when aged 7 years), 1969 (aged 11 years), 1974 (aged 16 years), 1981 (aged 23 years), 1991 (aged 33 years) and 1999 (aged 42 years). At age 42 years, 10 979 (approximately 63%) of the original subjects participated.

Assessment of intelligence, other early life characteristics and parental factors

Childhood IQ was measured using a general ability test.[30] Group administered at school when the study participants were 11 years of age, this test comprises 40 verbal and 40 non-verbal items. Children were presented with an example set of either four words or shapes or symbols that were linked either logically or semantically or phonologically. They were then given another set of three words or shapes or symbols with a blank. Participants were required to select the missing word from a list of five alternatives.[30,31] Testing took less than 2 h. In a separate study of primary school children ($N = 74$), the relation between scores from this test (range: 0–80) and those from an IQ-type test used by educational authorities to assess the type of secondary school to which a child should progress (an 11 plus selection test) was examined.[30] A strong, positive correlation was found ($r = 0.93$).[30] Further, in a recent report based on the present cohort,[32] scores from the test demonstrate the expected associations with birth weight[33] and childhood socio-economic position.[22] Thus, the test would appear to have a high degree of validity. As we report later, scores on the IQ

test were also positively correlated with later educational attainment ($r=0.57$, $P<0.001$) in the expected direction.

Based on existing evidence,[34] *a priori* we selected variables that could act as confounders in the association between IQ and obesity. These were parental BMI, pubertal development, birth weight and childhood socio-economic position. Parental BMI was based on self-reports of height and weight from the fathers (when the child was 11 years old) and the mothers (around the birth of the child when non-pregnant). Pubertal development was based on a medical examination at 16 years of age in which auxiliary hair development was recorded for boys (categorised as absent, sparse, intermediate and adult) and the age at menstruation reported by girls. Birth weight was extracted from maternity records. Fathers self-reported their occupation when the study participants were born, and when the child was 7 and 11 years of age. Occupational social class was then derived from the Registrar General's schema.[35,36]

Assessment of obesity and overweight and other adult characteristics

At age 16 years, the subjects' weight and height were measured by trained nurses; from age 23 years onwards, self-reported measures of height and weight were used. Both self-reported height and weight demonstrate a reasonable level of agreement with direct measurement.[37] Following conversion from imperial to metric units, BMI (weight (kg) divided by height squared (m^2)) was computed. Based on World Health Organization criteria,[38] we defined obesity as BMI $\geqslant 30.0\,kg/m^2$.

Educational qualifications at age 23 years was based on a six-point ordinal scale (no qualifications, CSE grades 2–5, 'O' levels, 'A' levels, professional qualifications, degree level or higher). At age 33 years, the study participants reported their own and, where applicable, their partners' occupation, from which occupational social class was derived.[39] Dietary characteristics were based on the reported consumption of chips (French fries), other fried food and fresh fruit. At age 33 years, respondents were asked: 'How often do you eat …. (chips/other fried food/fresh fruit)', to which enquiry there were six responses (more than once a day, once a day, 3–6 days a week, once or twice a week, less than once a week, never). A higher score was indicative of a more healthy consumption. The afore-mentioned covariates were conceptualised as potential mediating factors in the IQ–obesity/weight gain relation.

Statistical analyses

As we have utilised elsewhere,[3] in the present analyses we adopted two complementary statistical approaches: logistic regression and structural equation modelling (s.e.m.). Logistic regression was used to examine the relation of childhood IQ with adult obesity (see the above definition) at age 42 years. In these analyses, we computed odds ratios with accompanying 95% confidence intervals for the IQ–obesity relation with the 'exposure' both categorised into tertiles (tertile one: 0–33 IQ points; two: 34–50; three: 51–79), and utilised as a standard score (mean = 0; s.d. = 1). To assess the impact of potential confounding and mediating variables on this relation, we adjusted for these factors in a stepwise fashion.

Following these analyses, s.e.m. was employed in order to explicitly examine mediating pathways between childhood IQ and weight gain in adulthood by incorporating the repeated measures of BMI at ages 16, 23, 33 and 42 years, in addition to various covariate data. In all these analyses, BMI was retained as a continuous variable, allowing us to examine incremental increases in BMI values (denoted here as 'weight gain') as the cohort aged, and what predicts these incremental increases. The s.e.m. model (Figure 1) consists of two parts, the growth model and the structural pathways linking the variables (manifest (observed) variables are denoted by rectangles; latent variables by ovals). The growth model for weight gain comprises two latent variables: the intercept (BMI at the first time point, which is age 16 years) and the slope (change in BMI from age 16 to 42 years, incorporating all intermediate data collection points). The model is based on temporality of data, with childhood characteristics allowed to have an association with those collected in adolescence (IQ and puberty), both of which may also influence later life indices. An exception was made for the intercept of the growth model, which was correlated with later life data (educational qualifications, social class and diet). This takes into account the bi-directional association between obesity and psychosocial factors (childhood obesity could affect adult psychosocial conditions, whereas childhood psychosocial conditions could affect adult obesity). In order to maximise data use, latent variable models were utilised to capture socio-economic position throughout childhood (indicated by father's social class when the study participants were aged 0, 7 and 11 years) and diet using several indices of food intake (based on the enquiries regarding the consumption of chips, fried food and fruit).

Because Figure 1 represents a very large number of possible pathways, only those that had statistical significance (at the 5% level) are displayed in the resulting figures. Hu and Bentler[40] suggest the following cutoffs for good fitting models, with continuous and categorical outcomes: Tucker Lewis index (TLI) >0.95, comparative fit index (CFI) >0.95 and the root mean squared error of approximation (RMSEA) <0.06.

Using s.e.m., missing data were accounted for using maximum likelihood (ML) estimation in Mplus. This works by estimating a likelihood function for each individual based on the variables that are present, so that all the available data are used. This is conceptually analogous to generating predicted scores for the missing data by regressing it on other variables used in the analysis. This estimation method does not impute missing values, but directly estimates model parameters and standard errors using all available raw data.

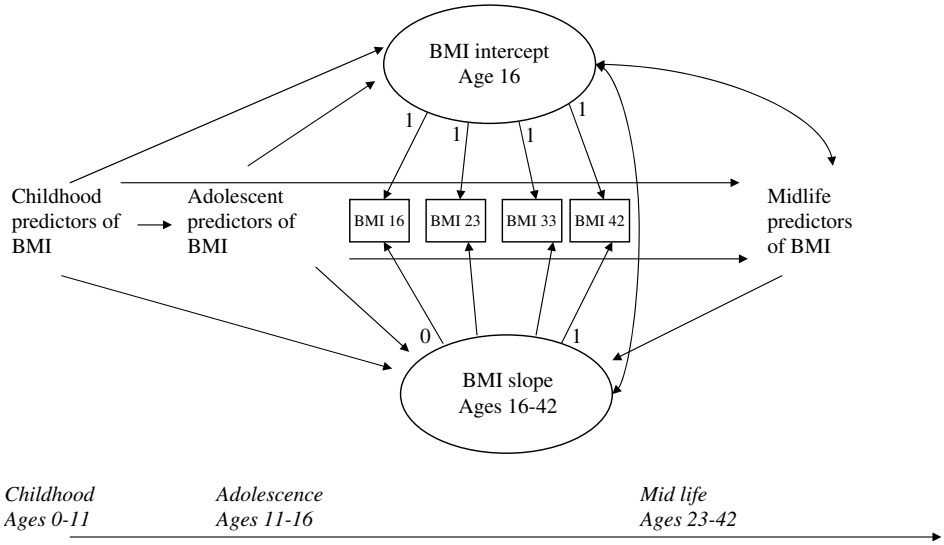

Figure 1 Full structural equation modelling pathway model, based on temporality of data measures.

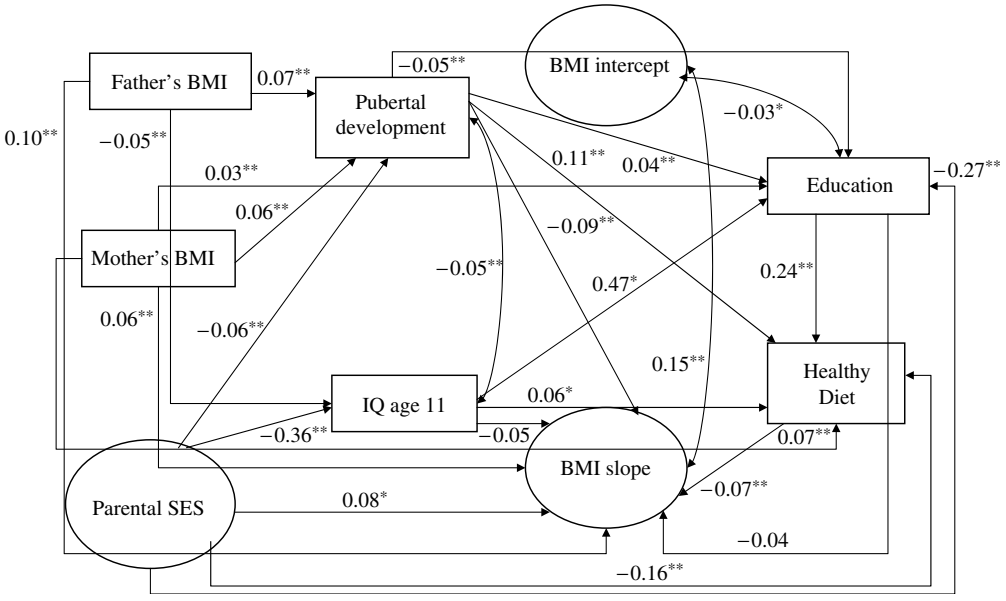

Figure 2 Structural equation modelling pathway model for men (χ^2/df = 264.6/66, CFI = 0.99, RMSEA = 0.03).

The data are assumed to be either missing completely at random or missing at random (MAR).[41] The latent outcomes and the latent variables are normally distributed. The latter assumption corresponds to the standard s.e.m. assumption and, as such, is not restrictive. The MAR assumption is more realistic if factors strongly associated with non-response or drop out are included in the model. In the present cohort, socio-economic deprivation and low birth weight were predictive of attrition.[42] The inclusion of these variables in the model as covariates thereby makes the MAR assumption more plausible, and the ML estimation more robust.

Results

Table 2 describes the physical, social and behavioural characteristics of the study participants across the life course according to childhood IQ. In general, the more favourable levels of each characteristic were apparent in the higher scoring IQ groups. Thus, in comparison to their lower IQ-scoring childhood peers, higher test performers were more likely to be heavier babies, have higher educational qualifications, less likely to be overweight in early adulthood, less likely to have a partner in, or themselves be in, a manual

occupation, and less likely to have a parent from the manual social classes. Higher IQ-scoring girls were also more likely to eat fresh fruit as adults, whereas in boys the gradient was weaker. Appendix A1 depicts a correlation matrix for all variables featured in the present analyses.

Table 3 displays the results of the logistic regression analysis of the relation between childhood IQ and obesity at age 42 years. In unadjusted analyses (model A), there was an inverse IQ–obesity gradient such that study participants with lower childhood IQ scores had a higher risk of obesity in middle age. This relation was somewhat stronger for women than men (model A). Thus, in women, each standard deviation disadvantage in childhood IQ was associated with a 38% increase in the odds of being obese at age 42 years. For men, the corresponding risk was 26%. Effect modification by sex did not, however, reach statistical significance, as evidenced by the overlapping confidence intervals of the point estimates on the IQ–obesity slope in male and female subjects.

The addition of early life factors, including father's socio-economic position, had essentially no effect on these associations (model B). Marked attenuation was, however, evident in both sexes following additional control for educational attainment at age 23 years (model C). Further adjustment for midlife characteristics — socio-economic disadvantaged and diet — attenuated the gradient somewhat further (model D). When social class and diet were added separately to the logistic regression models, IQ remained significantly associated with obesity (results not shown).

Further analysis using s.e.m. allowed us to examine the predictive value of childhood IQ for weight gain using continuous BMI values. The results of the growth model for weight gain in adulthood are displayed in Appendix B1. The regression coefficients for the intercept were fixed at '1', which enabled us to interpret the intercept as the initial BMI level (age 16 years). For the slope, the regression coefficients were fixed at zero at age 16 years and at 1 at age 42 years, leaving the coefficients at ages 23 and 33 years to be freely estimated. This coding enables us to conceptualise the BMI growth as being 0% complete at age 16 years and 100% complete at age 42 years. The mean intercept for men was 20.29, that is, at age 16 years, the average BMI for men. The mean slope was 6.23, that is, the average increase in BMI from age 16 to 42 years. For women, the equivalent statistics were 20.90 and 4.59, respectively. Although the χ^2 statistic

Table 2 Childhood IQ score in relation to socioeconomic, physical and behavioural characteristics across the life course

	IQ test score at age 11 years			
	Tertile 1 (0–33)	*Tertile 2 (34–50)*	*Tertile 3 (51–79)*	*Total* N
Men		*Mean (s.d.)*		
Height at age 7 years (m)	1.4 (0.1)	1.4 (0.1)	1.5 (0.1)	6146
BMI at age 16 years (kg/m^2)	20.3 (2.9)	20.2 (2.6)	20.3 (2.7)	4935
Auxiliary hair development at age 16 years (range: 1–4)	2.7 (1.)	2.8 (1.0)	2.9 (1.0)	4966
Mother's BMI (kg/m^2)	16.7 (2.7)	16.4 (2.4)	16.4 (2.3)	6504
Father's BMI (kg/m^2)	17.9 (2.4)	17.7 (2.2)	17.6 (2.0)	6182
BMI at age 42 years (kg/m^2)	27.0 (4.4)	26.4 (3.8)	26.1 (3.8)	4759
		Proportion (%)		
Low birth weight (<2.6 kg[a])	9.9	8.2	7.0	5926
Father in manual social class at age 7 years[b]	84.2	72.3	55.6	6178
No educational qualifications at age 23 years	39.1	10.0	2.4	3921
Manual social class at age 33 year	75.3	53.5	29.8	4531
Partner in manual social class at age 33 years	41.0	33.6	22.4	2902
Fruit consumption (>1 item fruit/day) at age 33 years	9.8	10.5	10.7	4800
Women		*Mean (S.D.)*		
Height at age 7 years (m)	1.4 (0.1)	1.4 (0.1)	1.5 (0.1)	5840
BMI at age 16 years (kg/m^2)	21.2 (3.2)	21.0 (3.0)	20.8 (2.8)	4671
Age of menstruation (years)	13.4 (2.1)	13.1 (1.8)	13.1 (1.9)	4172
Mother's BMI (kg/m^2)	16.5 (2.6)	16.4 (2.5)	16.2 (2.3)	6166
Father's BMI (kg/m^2)	17.9 (2.4)	17.7 (2.2)	17.5 (2.0)	5876
BMI at age 42 years (kg/m^2)	26.2 (5.5)	25.4 (4.9)	24.7 (4.7)	4808
		Proportion (%)		
Low birth weight (<2.6 kg[a])	12.2	8.4	5.9	5622
Father in manual social class at age 7 years[b]	87.4	70.4	53.2	5941
No educational qualifications at age 23 years	46.9	11.6	2.3	4001
Manual social class at age 33 years	53.6	33.5	20.7	4832
Partner in manual social class at age 33 years	67.9	51.0	36.7	2779
Fruit consumption (>1 item fruit/day) at age 33 years	19.2	24.4	30.1	4980

Abbreviation: BMI, body mass index. [a]Corresponds to the lowest decile; [b]father's social class when study participant was 11 years of age was utilized if data at 7 years were unavailable.

Table 3 Odds ratios (95% CI) for the relation of childhood IQ score with obesity at age 42 years ($N=9567$)

	No. of cases/no. of subjects	*Model A*	*Model B*	*Model C*	*Model D*
Men					
Tertile 1 (0–33)	301/1454	1.59 (1.28, 1.97)	1.58 (1.25, 2.00)	1.13 (0.87, 1.48)	1.10 (0.84, 1.44)
Tertile 2 (34–50)	247/1618	1.11 (0.90, 1.38)	1.19 (0.95, 1.50)	0.98 (0.77, 1.25)	0.97 (0.76, 1.24)
Tertile 3 (51–79)	199/1736	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
Per 1 s.d. decrease[a]	—	1.26 (1.15, 1.38)	1.26 (1.15, 1.39)	1.10 (0.98, 1.23)	1.09 (0.97, 1.22)
P-trend	—	0.001	0.001	0.10	0.15
Women					
Tertile 1 (0–37)	268/1387	1.99 (1.62, 2.46)	1.95 (1.55, 2.44)	1.29 (0.99, 1.67)	1.21 (0.92, 1.57)
Tertile 2 (38–52)	237/1632	1.38 (1.12, 1.71)	1.36 (1.09, 1.71)	1.12 (0.88, 1.41)	1.08 (0.85, 1.37)
Tertile 3 (53–79)	229/1740	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
Per 1 s.d. decrease[b]	—	1.38 (1.26, 1.50)	1.34 (1.22, 1.48)	1.11 (0.99, 1.25)	1.08 (0.96, 1.21)
P-trend	—	0.001	0.001	0.06	0.21

Model A – no adjustment. Model B – adjustment for childhood factors: height at 7 years; BMI at 16 years; pubertal development; mother's and father's BMI; birth weight; sports activity; sweet consumption; father's occupational social class. Model C – adjustment for childhood factors (as above) and education attainment at 23 years. Model D – adjustment for childhood, education and other adult characteristics: partner's and subject's occupational social class; fruit consumption. [a]1 s.d. is 16.23 IQ points. [b]1 s.d. is 15.90 IQ points.

Table 4 Direct and indirect effects of childhood IQ on weight gain in adulthood estimated from Figures 2 and 3

	Direct	*Indirect via qualifications*	*Indirect via healthy diets*	*Total indirect*	*Total effect*
Men ($N=8987$)	0.002	−0.004	−0.002*	−0.011*	−0.006*
Women ($N=8403$)	−0.002	−0.010**	−0.003**	−0.011**	−0.013**

A unit increase in IQ (ranging from 0 to 80) increases (or decreases) the BMI slope by the number shown in the cell. *$P<0.05$; **$P<0.01$.

for the growth curve models for men and women was high relative to the degrees of freedom, the CFI and TLI statistics indicate that the models were an acceptable fit. Similarly, the RMSEA statistic for the model for men also indicates that this was an acceptable fit, although it was higher (i.e., of poorer fit) for women.

For ease of interpretation and brevity, only standardised pathways affecting the BMI slope are shown in Figures 2 (men) and 3 (women). In both figures, IQ did not have a statistically significant direct effect on weight gain. Parental size and social class, earlier pubertal development, and a healthy diet were associated with weight gain in both men and women. Additionally, in women only, education also had a significant effect. In both models, the intercept (BMI at age 16 years) was negatively correlated with education such that overweight children are less likely to obtain higher qualifications. The unstandardised indirect effects of a unit increase in IQ (ranging from 0 to 80) on weight gain in adulthood are presented in Table 4. For men and women, childhood IQ is associated with weight gain through a healthy diet, and in addition, for women, there is evidence of a pathway via educational qualifications.

As education and IQ are moderately correlated ($r=0.56$, $P<0.001$), the difference in the fit of two nested models was tested to examine whether the effect of education on weight gain is confounded by childhood IQ. The goodness of fit of the models for men and women was compared with the model that constrained the pathways from education to weight gain to be zero (i.e., the direct pathway from education to weight gain and the indirect pathway via healthy diet). The χ^2 difference was 160 on two degrees of freedom for men, and 116 on two degrees of freedom for women, indicating a significantly poorer fit (both $P<0.001$). This suggests that the education–weight gain association is not confounded by childhood IQ (Figure 3).

Discussion

In the present study, IQ test scores at 11 years of age were inversely related to obesity at age 42 years, an effect that was marginally stronger in women than men. These observations were made whether obesity was modelled as a dichotomous outcome using only BMI at age 42 years (logistic regression analyses), or as a growth curve using the whole range of individual differences in weight changes from age 16 to 42 years (s.e.m. analyses). 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

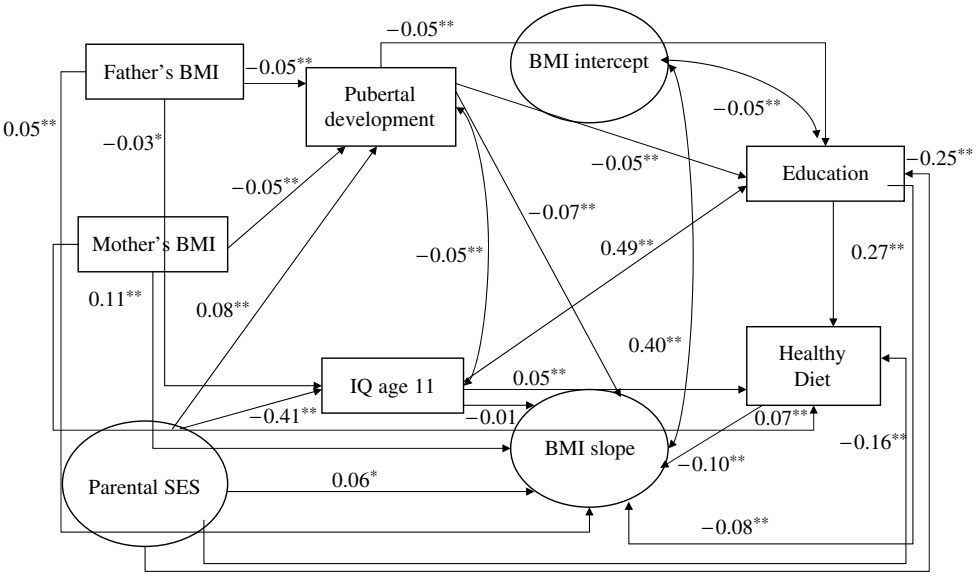

Figure 3 Structural equation modelling pathway model for women (χ^2/df = 617.7/65, CFI = 0.98, RMSEA = 0.03).

made for the subjects' educational attainment. Further control for other potential mediating variables, including subjects' adult occupational social class and dietary characteristics, had a small additional attenuating effect.

Our motivation for examining the relationship of childhood IQ with adult adiposity and weight gain was based on the suggestion that the apparent protective effect of high pre-adult IQ against premature mortality, evident across a series of studies, might be partially explained by mediation via obesity in later life, in addition to other established risk factors.[4] Although the link between obesity and total mortality,[16,43] cardiovascular disease[16,44] and some cancers[15,17] is well established, the influence of weight change, even when extreme, is less clear.[45–47] Nonetheless, we elected also to utilise weight gain as an outcome in the analyses in order to provide a comprehensive picture of the relationship, if any, between early IQ with adult body weight. The present study is one of very few to have the data to do so.

We found that the IQ–obesity gradient was eliminated at conventional levels of statistical significance when adult characteristics, including socio-economic disadvantage, were added to the multivariable model. This accords with the findings of some other studies where the same observation was made when all-cause mortality was the outcome of interest.[8] This is not, however, a universal finding.[6,48]

Comparison with previous studies

This is first prospective study to report on the relation between childhood IQ and adult obesity in men and women separately with control for a range of potential confounding and mediating variables across the life course. Although direct comparison with existing studies is therefore problematic, elements of some studies are relevant. The observation herein that statistical control for early life social factors has little impact on this IQ–obesity relation, but adult indicators such as education, occupational social class and diet markedly attenuated it, is consistent with some existing studies. Using a case–control design, two groups of invesigators[25,27] found that the lower IQ scores in obese cases in comparison to non-obese controls were not altered by stratification by parental social class (a marker of the study participants' early life socio-economic position), thus supporting our own findings. In three studies that utilised data on adult education, two[23,26] found that statistical control for this characteristic diminished the IQ–obesity gradient to the point of nonsignificance. In another,[24] the relation between IQ and the full range of BMI scores held. We also found that dietary characteristics in adulthood partially attenuated the IQ–obesity association. To our knowledge, this is the first study to examine the role of diet in this regard.

The finding of an inverse relation between IQ and obesity in cross-sectional[24,25] and case–control studies[23,24,26] — the study designs utilised most frequently in examining this research question — raises the possibility that rather than IQ predicting obesity, the development of this condition, with its potential for precipitating social withdrawal, might lead to reduced cognitive function.[23,27] Longitudinal studies facilitate an examination of this issue of reverse causality in at least two ways: either by excluding persons with existing obesity at study induction so that new cases of obesity can be studied, or by controlling for obesity status. In taking the latter approach, we adjusted for BMI score at age 16 years. There was essentially no difference between the unadjusted effect estimates for childhood IQ and obesity at age 42 years (Table 3; model A) and those that were so adjusted (model B).

Plausible mechanisms of effect

It has repeatedly been shown that higher childhood IQ test scores predict favourable socio-economic position in later life (as indexed by high educational attainment, elevated income and raised occupational social status).[22,49,50] Similarly strong evidence points to a gradient between educational attainment and obesity and/or overweight in men and women.[51] Based on the data collected in the present study, it is plausible therefore that raised scores on childhood IQ tests set in motion a protective chain of events that lead to a reduction in later life obesity risk: high IQ scores are associated with higher educational success in early adulthood, and subsequent placement into a well-remunerated, high-status occupation in mid-life. That the early life IQ–adult obesity association seemed to be mediated via educational attainment in the present data set provides some support for this suggestion. This pathway may also be extrapolated further back into early life: for instance, pre-adult socio-economic disadvantage[5,52,53] (as indexed by parental occupation, and family size), impaired fetal growth[33] (as indexed by birth weight) and reduced parental IQ test scores[54] are all related to low pre-adult IQ. Interventions that may reduce socio-economic differentials in health include improved access to resources (e.g., education) and physical exposures in the working and living environment (e.g., workplace housing conditions). Such measures are also likely to favourably influence IQ, particularly when directed at early life, although this is not a universal finding.[55]

What is also plausible, but often ignored, is that educational attainment may represent a proxy for IQ.[12,56] As such, including education in our statistical models may be seen as over-adjustment. However, although educational qualifications and childhood IQ are moderately correlated in the present study, the data here suggest that education affects weight gain in adulthood independently of childhood IQ, and that including education in our models should not, in fact, be regarded as over-control. Whether educational qualifications might be acting partly as a proxy for individual differences in IQ evident after age 11 years, and in cognitive domains not examined in the test administered at age 11 years, could not be examined in the present data set.

Strengths and limitations

As described, the strengths of this study lie in its size, which exceeds that of most others; its prospective design; the sex-specific analyses; the repeated measurements of BMI from age 16 to 42 years; the wide range of covariate data, which were serially gathered across the life course; and the use of s.e.m., which takes into account the bi-directional association between obesity and psychosocial factors over the life course. However, the longitudinal nature of the NCDS has, inevitably, led to some attrition. At age 42 years, 63% of the original cohort responded to the questionnaire mailing, a figure that compares favourably with other long-term follow-ups of child cohorts, including the Aberdeen *Children of the 1950s* study[57] and the 1946 birth cohort.[58] In keeping with other findings,[59,60] analyses of non-response in the NCDS data[42] show that there are systematic differences between respondents and non-respondents at every sweep of the data collection. Thus, in comparison to current study members, non-respondents were more likely to have only basic educational qualifications, come from a more disadvantaged social background and have less stable employment patterns. Although these differences were statistically significant, there is, in fact, only a small difference in the predicted probabilities of responding and not responding,[42] supporting the assumption that the data are MAR. It is unlikely, therefore, that attrition in the present study has led to selection bias. Finally, the temporal sequencing of the s.e.m. analysis should not be taken to imply causality. There may be other variables, not incorporated in the present analysis or unmeasured, that may be on the causal pathway. For example, physical activity may be a mediator in the IQ–obesity association, but detailed measures of physical activity were not available in the present study.

The public health implications of our results are significant. Already obese people are subjected to felt and enacted stigma, and we do not wish to add the additional stigma of an association with lower IQ scores. Consequently, we emphasise that the current rise in the prevalence of obesity has not been accompanied by a fall in IQ levels during childhood.[61] Of similar relevance to our findings, rising levels of obesity have been accompanied by rising, not falling, levels of educational attainment. Of our main explanatory variables, therefore, nutrition (as indexed by healthy eating) is of particular relevance to the current challenge facing public health.

In conclusion, in the present study, a low child IQ test score was related to adult obesity and weight gain in both men and women. This effect was substantially mediated via education and healthy eating.

Contributions

David Batty generated the idea for this study. The analytical strategy was formulated following discussions between Tarani Chandola, Ian Deary and David Batty. Tarani Chandola conducted all data analyses. David Batty wrote the first draft of the manuscript, and all co-authors made significant contributions to subsequent revisions.

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Appendix A1

Inter-variable correlations are given in Table A1.

Table A1 Correlations above the diagonal refer to women; below the diagonal refer to men

	IQ	*BMI 16 years*	*BMI 23 years*	*BMI 33 years*	*BMI 42 years*	*Birth weight*	*Mum BMI*	*Father BMI*	*Axillary hair*	*Age menstruation*	*Father's class*	*Qualifications*	*Class 33*	*Partner's class*	*Fruit*	*Fried food*	*Chips*
IQ	1	−0.064	−0.125**	−0.120**	−0.127**	0.111**	−0.052**	−0.065**	0**	−0.070[a]	−0.312**	0.583**	−0.322**	−0.244**	0.181**	0.129**	0.183**
BMI 16	0.002	1	0.639**	0.547**	0.498**	0.089**	0.241**	0.201**	0**	−0.201[a]	0.053	−0.050	0.036*	0.043*	0.054**	0.054**	0.046**
BMI 23	−0.115**	0.615**	1	0.724**	0.708**	0.048**	0.225**	0.170**	0**	−0.114[a]	0.109**	−0.174**	0.119**	0.141**	0.015	−0.017	−0.024
BMI 33	−0.093**	0.509**	0.668**	1	0.795**	0.049**	0.215**	0.143**	0**	−0.130[a]	0.097**	−0.153**	0.095**	0.109**	0.031	−0.062	−0.051
BMI 42	−0.098**	0.507**	0.677**	0.741**	1	0.025**	0.212**	0.158**	0**	−0.133[a]	0.099**	−0.169**	0.089**	0.131**	−0.005	−0.046	−0.055
Birthweight	0.081**	0.111**	0.078**	0.062**	0.078**	1	0.142**	0.034	0	−0.008[a]	−0.054**	0.098**	−0.072*	−0.024	0.094**	0.056	0.064
Mum BMI	−0.050**	0.224**	0.205**	0.179**	0.189**	0.135**	1	0.084**	0**	−0.048[a]	0.051**	−0.072**	0.033**	0.041**	0.020	0.026	0.026
Father BMI	−0.054**	0.187**	0.206**	0.178**	0.193**	0.008	0.060**	1	0**	−0.042[a]	0.045	−0.051**	0.036**	0.051	0.014	0.026	−0.001
Axillary hair	0.071**	0.249**	0.114**	0.112**	0.095**	0.011	0.060**	0.062**	1	[a]	**	**	**	**	**	**	**
Age menstruation	[a]	[a]	[a]	[a]	[a]	[a]	[a]	[a]	[a]	1[a]	0.057[a]	−0.091[a]	0.045[a]	0.041[a]	−0.063[a]	−0.049[a]	−0.031[a]
Father's class	−0.259**	−0.006	0.090**	0.069**	0.061**	−0.048**	0.063**	0.025	−0.045**	[a]	1	−0.328**	0.210**	0.205**	−0.125**	−0.089**	−0.161**
Qualifications	0.551**	−0.025	−0.158**	−0.087**	−0.127**	0.068**	−0.089**	−0.049**	0.069**	[a]	−0.289**	1	−0.456**	−0.360**	0.257**	0.181**	0.255**
Class 33	−0.392**	0.042*	0.139**	0.080**	0.098**	−0.033*	0.081**	0.043**	−0.050**	[a]	0.244**	−0.463**	1	0.289**	−0.151**	−0.127**	−0.192**
Partner's class	−0.221**	0.049*	0.097**	0.087**	0.094**	0.001	0.054**	0.027	−0.062**	[a]	0.141**	−0.283**	0.281**	1	−0.133**	−0.109**	−0.183**
Fruit	0.080**	0.067**	0.019	0.005	0.006	0.057**	0.010	0.003	0.056**	[a]	−0.049**	0.132**	−0.078**	−0.085**	1	0.192**	0.253**
Fried food	0.138**	0.076**	0.007	−0.022	−0.007	0.013	−0.006	0.005	0.079**	[a]	−0.094**	0.187**	−0.147**	−0.141**	0.179**	1	0.454**
Chips	0.162**	0.083**	0.025	−0.001	0.011	0.023	−0.018	0.015	0.093**	[a]	−0.131**	0.224**	−0.188**	−0.165**	0.217**	0.417**	1

**Correlation is significant at the 0.01 level (two-tailed). *Correlation is significant at the 0.05 level (two-tailed). [a]Cannot be computed because at least one of the variables is constant.

Appendix B1

Estimates and goodness of fit for the growth model of weight gain (measured by BMI at ages 16–42 years) for men and women are given in Table B1.

Table B1

	Loadings							
Women	Age 16 years	Age 23 years	Age 33 years	Age 42 years	Mean	Variance	χ^2, df	CFI/TLI/RMSEA
Intercept	1[a]	1[a]	1[a]	1[a]	20.90**	6.39**	219.1, 3	0.98/0.96/0.10
Slope	0[a]	0.14	0.85	1[a]	4.59**	9.36**		
	Loadings							
Men	Age 16	Age 23	Age 33	Age 42	Mean	Variance	χ^2, df	CFI/TLI/RMSEA
Intercept	1[a]	1[a]	1[a]	1[a]	20.29**	4.73**	32.5, 3	1.00/0.99/0.04
Slope	0[a]	0.37	0.84	1[a]	6.23**	6.99**		

[a]Fixed coefficients. $P<0.01$.