

RESEARCH REPORT

Combination of low birth weight and high adult body mass index: at what age is it established and what are its determinants?

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J Epidemiol Community Health 2003;57:969–973

Objective: To investigate growth trajectories and predictive factors for those with low birth weight and high adult BMI.

Design: Birth cohort study.

Setting: England, Scotland, and Wales.

Participants: All born 3–9 March, 1958.

Main outcome measures: People at “high risk” of adult disease were defined as having a combination of lower birth weight (in the lowest third of the distribution) and high BMI (in the highest third of the distribution at age 33).

Results: 284 of 3462 men and 338 of 3555 women were identified as “high risk”. This group was shorter than other cohort members at age 7, on average by 1.2 cm (boys) and 1.8 cm (girls), with a deficit of about 3 cm in adult height. The “high risk” group had a similar mean weight to other subjects at age 7, but were heavier thereafter through to age 23. BMI was increased at all ages in the “high risk” group. Independent predictors include paternal BMI, maternal height and smoking in pregnancy, and social class. For each SD increase in father’s BMI the odds of low birth weight/high BMI increased by about 20%. For maternal height, a 1 cm increase reduced the odds of low birth weight/high BMI by about 5%. Increased ORs for “high risk” were found for those with manual social origins (1.61 for men; 1.49 for women) and for maternal smoking in pregnancy (1.79 and 2.27 respectively).

Conclusions: Maternal short stature, low social class, and smoking during pregnancy influence the development of “high risk” for adult chronic disease. The causes of high risk therefore seem to reside in utero and even earlier, in the mother’s lifetime, with adverse conditions having a detrimental affect and favourable conditions protecting against high risk.

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Accepted for publication 16 May 2003

The past decade has witnessed growing acceptance of the “fetal origins of adult disease” hypothesis.¹ Although the mechanisms have yet to be determined, it is now thought that poor intrauterine growth is associated with greater risk of cardiovascular disease and non-insulin dependent diabetes (NIDDM) in adult life.² However, some studies suggest that the risks associated with poor early growth depend on disease markers in adulthood, in particular adult obesity.^{3–6} People with a combination of poor early growth and high adult body mass index (BMI) have been found to be at the greatest risk of adult disease. For example, the increase in blood pressure seen among 50 year old Swedish men who were lighter at birth was especially pronounced in those with a high adult BMI.⁵ Similarly, insulin resistance was found to be greatest among men born with low birth weight who attained a high BMI in adulthood.³ An analogous interaction was demonstrated for coronary heart disease incidence in the Caerphilly Study⁶ and low birth weight followed by a high BMI at age 11 years, was related to coronary heart disease in a Finnish study.⁷ Several studies therefore suggest that it is high BMI in late middle age which is of importance,^{3–6} although recent work also suggests that attaining a high BMI in childhood influences later risk.^{8–10}

So far, however, research has not identified what factors either generate or are associated with the combination of low birth weight and high BMI at later stages of life, and furthermore, little is known about the growth trajectories of those with this high risk combination. There is some evidence that later BMI is positively associated with weight at birth,¹¹ and we would therefore expect those with low birth weight to

be leaner rather than fatter in adulthood. To our knowledge, no studies have yet been able to establish whether the group who have low birth weight and high BMI in middle age have already become comparatively obese in their pre-adult years. Clearly an understanding of the aetiological processes involved depends upon determining the stage of life at which the trajectory of low birth weight to adult obesity is established. Adverse socioeconomic conditions in early life may underlie this trajectory, given that such social circumstances are consistently related to adult BMI¹² and with gain in BMI from childhood to early adulthood.¹³ The aim of this study is therefore to investigate the growth trajectories of those with low weight at birth and high BMI in adulthood. For this group with “high risk” of chronic disease in adulthood we seek to identify, firstly, the life stage at which compensatory growth occurs, and secondly, whether factors related to social circumstances in early life are involved in the development of “high risk” status, taking account also of parental size, which is an established determinant of offspring growth. The study uses data from the 1958 British cohort,¹⁴ followed up from birth to age 33 years.

METHODS

Study sample

A total of about 17 000 singletons born in England, Scotland, and Wales were enrolled in the Perinatal Mortality Survey in the week 3–9 March 1958.¹⁴ Surviving children were studied at ages 7, 11, 16, 23, and 33 years. At age 33, 11 405 people were included. Despite sample attrition, those remaining in

the study were found to be generally representative of the original sample.^{14 15}

We use information from each follow up from birth to age 33: 5047 men and 5238 women had data on birth weight and 33 year BMI. To avoid reductions in the numbers available for analysis, all people with relevant data were included for each age. The multivariable analysis of early life factors were restricted to those subjects with complete information ($n = 7017$). This sample was compared with the original sample to establish whether it was representative of those enrolled into the study. Differences were small: for example, mean birth weight in the analysis sample was 3434 g (males) and 3281 g (females), compared with 3400 g and 3263 g respectively in the original sample; and for social class, 30% of the analysis sample had non-manual backgrounds, compared with 27% in the original sample.

Measures

Birth weight was recorded in pounds and ounces by the midwives in charge of the delivery, and has been converted to kilograms. Heights and weights were measured by trained medical personnel at ages of 7, 11, and 16, but self reported at 23 years. At age 33, height was measured without shoes using a stadiometer reading to the nearest centimetre, and weight was measured in indoor clothing using Salter portable scales. BMI was derived as weight (kg)/height(m)².

"High risk": weight at birth and BMI at 33 years were classified into thirds, using centile cut offs shown in table 1. These centile cut offs were used to ensure an adequate sample (of about 10%) for the analysis of "high risk". A "high risk" group was defined as having a birth weight in the lowest third of the distribution for the whole cohort (that is, below 3147 g for males) and a BMI at age 33 in the highest third (that is, above 26.6 for men). This "high risk" group comprised 9.2% (466 of 5047) of men and 10.0% (524 of 5238) of women in the full sample; and 8.2% (284 of 3462) and 9.5% (338 of 3555) respectively in the multivariable analysis sample.

Predictors of high risk: measures identified as potential determinants of "high risk" status included parental BMI, height, age, social class, maternal smoking during pregnancy and infant feeding method and birth order. Maternal height without shoes was measured, and pre-pregnant weight self reported in categories of one stone, shortly after the birth of the cohort member, in 1958. Paternal measures were reported in most cases by the mother, in 1969 when the cohort member was aged 11. Father's height was reported to the nearest inch and weight was classified into one of 27 groups ranging from 6 stones 4 pounds (39.9 kg) to 19 stones 10 pounds (125.2 kg) in increments of seven pounds (2.7 kg). For the purpose of estimating BMI, parents were assigned a weight at the midpoint of their weight group. Social class of origin was based on the father's occupation in 1958, categorised as non-manual and manual. Maternal smoking during pregnancy was recorded at the time of birth and classified as smoker (≥ 1 cigarette/day) and non-smoker.

Table 1 Centile cut offs for birth weight and 33 year BMI used to identify high risk status

	33rd centile	66th centile
Birth weight (g)		
Male	3147	3600
Female	3033	3430
BMI at 33 (kg/m ²)		
Male	23.8	26.6
Female	22.1	25.3

Information on infant feeding was obtained at the seven year survey, and reported as: never breast fed, breast fed <1 month, or >1 month.

Data analysis

Mean standard deviation scores (SDS) for height, weight, and BMI were derived for each age and sex, thus allowing for differences in variance of these measures at different ages. SDS for height, weight, and BMI were plotted for the "high risk" group (that is, those with low birth weight and high 33 year BMI) and for all other cohort members, to identify the age at which persons at "high risk" gain in body size.

In further analyses, we examined associations between potential predictors (parental BMI, height, age, maternal smoking, infant feeding, social class) and high risk status. We examined these associations in both univariate and multivariate analyses: unadjusted odds ratios (OR) and 95% confidence intervals (CI) were obtained from logistic regression models, separately for men and women. Two way interactions were tested: none were significantly related to high risk status, so only main effects are included here. Next, we assessed whether the predictors of high risk status applied when prenatal growth was defined by birth weight for

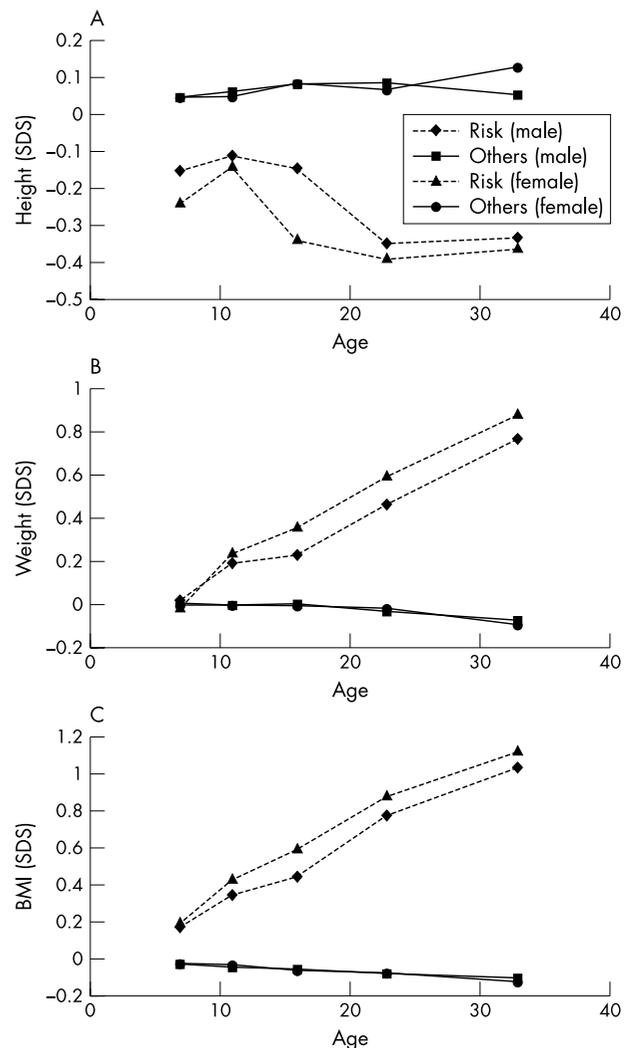


Figure 1 (A) Trend in height for high risk group compared with other cohort members. (B) Trend in weight for high risk group compared with other cohort members. (C) Trend of BMI for high risk group compared with other cohort members.

gestational age, rather than birth weight alone. Repeating the logistic regression models for high risk, defined as the lowest third of the centile distribution of birthweight for gestational age, combined with the highest third of 33 year BMI, showed a similar pattern of relations with predictor variables (data not shown). Finally, we estimated the variation in 33 year BMI explained by birth weight (0.6% and 0.4% for males and females respectively) from a separate regression model with a quadratic term.

RESULTS

By definition the high risk group had a lower weight at birth than other subjects: mean birth weight was 2813 g and 3476 g respectively for males; 2690 g and 3327 g for females. At age 7, boys in the high risk group were one sixth of a SD shorter than the mean height for their age, and 1.2 cm shorter than boys not at high risk. Similarly, girls in the high risk group were a quarter of a SD shorter than the mean 7 year height and 1.8 cm shorter than girls not at high risk. By age 11 the height deficit had reduced slightly (fig 1A). The trend in height between 7 and 11 years suggests an earlier growth spurt in the high risk group, possibly indicating a faster rate of maturation than among other children. After puberty, the deficit in height increased, such that by age 33 the high risk group was shorter by about a third of a SD compared with the mean, or by 2.7 cm (males) and 3.2 cm (females) compared with those not at high risk. (Discrepancies between 23 and 33 year heights are attributable largely to differences between self reported compared with measured heights and to small differences in sample size). In adolescence, the increase in height deficit occurred at an earlier age for females than males, corresponding to their earlier age of maturation.

In contrast with height, there was no weight (SDS) deficit for the high risk group at age 7: indeed their mean weight was similar to that of other subjects (fig 1B). But thereafter, the high risk group was heavier at age 11 years and thereafter. The trend in increasing weight was monotonic for both sexes, and thus seems to suggest a gradual excess weight gain in the high risk group over time. Figures 1A and 1B show that, in general, the high risk group was both heavier and shorter from age 11 to 33 years. As the trends for weight and height suggest, BMI in the high risk group was

increased at all ages, and becomes increasing divergent over time (fig 1C).

Table 2 shows the predictors of high risk status. Most factors examined were significantly associated with high risk before adjustment for other factors and all but parent's age were included in the adjusted model. Predictors remaining significant after adjustment for other factors, include paternal BMI, maternal height and smoking in pregnancy, social class and birth order. The adjusted estimates indicate positive associations for parental BMI, such that for each SD increase in father's BMI the odds of low birth weight/high BMI increase by 18% in men and 23% in women. For maternal height, a 1 cm increase reduced the risk of low birth weight/high BMI by 5% and 4%, respectively for males and females. Odds ratios for high risk status were also increased among those with manual social class origins (adjusted estimates indicate an increased risk of 61% and 49% respectively in males and females) and among those whose mothers smoked during pregnancy (OR = 1.79 for males; 2.27 for females). In general, odds ratios were slightly larger for women than men, albeit not significantly. Finally, in a further series of analyses, we examined whether the predictors in table 2 were associated separately with birth weight and with adult BMI among the population not at "high risk" (that is, excluding the latter group from these analyses). Most predictors were related in unadjusted analyses to both birth weight and adult BMI (data not presented), although maternal height and birth order were related to birth weight but not to adult BMI and no associations were seen for breast feeding. Thus, some early factors predicting "high risk" tend to influence both dimensions (birth weight and adult BMI) of high risk.

DISCUSSION

Low birth weight and high BMI are, each separately, predictive of multiple health conditions from childhood through to adulthood. Thus, in their own right they provide a focus for public health concern. Recent research suggests, however, that the combination of low birth weight and high BMI has particularly detrimental effects on chronic disease in adult life, notably increased blood pressure,¹⁶ coronary heart disease,^{6,7} and impaired glucose tolerance.^{3,4} A combination of low weight at birth and high BMI represents dispropor-

Table 2 Predictors (odds ratios and 95% CI) of high risk status (low birth weight and high BMI at 33 years)

Predictors	Men (n = 284/3462)			Women (n = 338/3555)		
	N	OR(95% CI)		N	OR(95% CI)	
		Unadjusted	Adjusted†		Unadjusted	Adjusted†
Maternal BMI		1.03 (1.00 to 1.07)	1.02 (0.99 to 1.06)		1.03 (1.00 to 1.06)	1.03 (0.99 to 1.06)
Paternal BMI		1.06 (1.01 to 1.10)	1.07 (1.01 to 1.10)		1.08 (1.04 to 1.12)	1.07 (1.03 to 1.11)
Maternal height (cm)		0.95 (0.93 to 0.97)	0.95 (0.93 to 0.97)		0.95 (0.93 to 0.97)	0.96 (0.94 to 0.98)
Paternal height (cm)		0.99 (0.97 to 1.00)	1.00 (0.99 to 1.02)		0.97 (0.96 to 0.98)	0.99 (0.97 to 1.00)
Mother's age at birth		0.99 (0.97 to 1.01)	—		0.99 (0.97, 1.01)	—
Father's age at birth		0.99 (0.97 to 1.01)	—		1.00 (0.98 to 1.02)	—
Social class						
non-manual*	1050	—	—	1031	—	—
manual	2412	1.90 (1.39 to 2.59)	1.61 (1.16 to 2.21)	2524	1.88 (1.41 to 2.51)	1.49 (1.11 to 2.02)
Maternal smoking during pregnancy						
non-smoker*	2377	—	—	2413	—	—
smoker	1085	1.85 (1.44 to 2.37)	1.79 (1.37 to 2.29)	1142	2.31 (1.83 to 2.90)	2.27 (1.79 to 2.86)
Birth order						
1st born*	1236	—	—	1311	—	—
2nd born or greater	2226	0.80 (0.62 to 1.03)	0.72 (0.55 to 0.93)	2244	0.72 (0.57 to 0.90)	0.64 (0.50 to 0.81)
Breast feeding						
never	1035	1.30 (0.98 to 1.73)	1.15 (0.86 to 1.54)	1018	1.46 (1.12 to 1.91)	1.26 (0.96 to 1.66)
<1 month	848	0.99 (0.72 to 1.37)	0.90 (0.65 to 1.25)	892	1.22 (0.91 to 1.63)	1.03 (0.77 to 1.39)
>1 month*	1579	—	—	1645	—	—

*Reference category; †adjusted model includes all variables except parental age.

Key points

- Increases in coronary heart disease have been reported for people with a “high risk” combination of low weight at birth and obesity in adulthood, yet the development of “high risk” has been neglected.
- We found that “high risk” people started their disproportionate weight gain early in childhood and continued on a faster weight trajectory to adulthood despite being shorter than others.
- Those at “high risk” had shorter mothers and fatter fathers, they were more likely to have manual social origins and to have mothers who had smoked during pregnancy.
- Conditions in the prenatal period and in the mother’s lifetime seem to influence growth trajectories associated with adverse adult disease outcome.

tionate weight gain, which has been related to glucose intolerance in childhood.^{8–10} Factors predisposing people to, or protecting them from, this pattern of growth are therefore of interest in this context. Yet to our knowledge, there is no other study that examines why some people have this combination of risks. Catch up growth in early life has been related to subsequent adiposity, but only to age 5,¹⁷ while our study extends to a measure of obesity in adulthood. In the 1958 cohort those identified as high risk status, with low birth weight and high BMI, were more likely to have shorter mothers and fatter fathers, lower social class origins and mother’s who smoked during pregnancy. The growth trajectories of the high risk group showed that, while they were comparatively short in stature, they gained weight steadily through childhood to adulthood. Less regular trends may have occurred between the ages available in the study, but we have no information on this.

Methodological considerations

Other methodological issues should be considered. The first concerns sample attrition: 7010 people had complete data for multivariable analysis. There were only small differences between this group and the original sample, at least in respect of weight and social class at birth, and thus we regard the analysis sample as generally representative of the original cohort. It is unlikely that the small biases observed would change the associations from multivariate analysis on predictors of “high risk”. Secondly, height and weight were reported at age 23 and measured at all other ages. This probably accounts for differences between 23 and 33 year heights. Thirdly, regarding the definition of high risk, the 33rd centile cut offs used for birth weight and BMI do not equate with standard definitions for these measures, but none the less ensured an adequate analysis sample of about 10%. Given the argument that fetal growth rate rather than size at birth is the important causal factor, at least for ischaemic heart disease¹⁸ we examined high risk status using birth weight for gestational age as well as for birth weight in itself. A similar pattern of relations with predictor variables was found. Finally, we focused on early life predictors because the childhood growth measures suggested that the high risk group had already caught up in weight by age 7, thereby implicating factors occurring before this age.

Growth trajectories

Birth weight and BMI are themselves not entirely independent, with several studies reporting a positive relation,¹¹

although the relation is weak in this cohort, especially in adulthood.¹⁹ The general tendency is for those with a high adult BMI to have had a high, rather than low, weight at birth. Our high risk group had, on average, a shorter stature from childhood through to adulthood, which is consistent with studies reporting a positive birth weight/height relation.^{20–23} Some early linear growth acceleration was evident for the high risk group, between 7 and 11 years. (This may also have occurred at younger ages, but our study is unable to detect this.) Even so, men and women in the high risk group were, on average, 2.7 cm and 3.2 cm shorter in adulthood than other subjects in the cohort. Accelerated rate of maturation as indexed by various measures, including stage of puberty and adiposity rebound, has been shown to increase the risk of fatness in adolescence and adulthood^{24–26}; while in early life, catch up growth increased the risk of obesity at age 5.¹⁷ It is of interest that for the high risk group identified in our study, there seemed to be no critical stage of weight gain, for example around puberty, rather the increase in BMI relative to other subjects was steady over the first three decades of life.

Predictors of high risk

High risk status was increased among cohort members from manual social origins, shorter mothers, fatter fathers, and mothers who smoked during pregnancy. Among these factors, short maternal stature is thought to index suboptimal lifelong nutritional status of the mother, while low social class and smoking during pregnancy would be associated with an intrauterine environment restricting fetal growth. These factors suggest that conditions constraining fetal growth play a part in the development of high risk status. Our findings are supported by previous research in which both lower social class and maternal smoking are associated with smaller size at birth.^{27–28} Recent reviews on obesity also implicate poor social conditions in early life¹²; while the growing number of studies on maternal smoking show a greater weight gain across differing periods of childhood among offspring of mothers who smoked during pregnancy.^{17–29–35} It was anticipated that parental body size would be among the important influences affecting high risk among offspring, as parent’s size is a principal determinant of both fetal growth and BMI. However, the differing effect of maternal and paternal factors is notable in this regard. While shorter maternal height increased the risk of having the combination of low birth weight and high adult BMI in the 1958 cohort, paternal height did not. This parallels previous research on fetal growth, which highlights the influence of maternal rather than paternal factors, including height and weight.^{27–28–36} Yet, our results contradict another study in which tallness of fathers, but not height of mothers, was associated with early catch up growth.¹⁷ Low birth weight among offspring of shorter mothers is thought to reflect maternal regulation of growth, and influenced in part by the mother’s own intrauterine environment.^{37–38} Thus, the effect of maternal short stature seen in our study suggests that high risk status is affected by unfavourable conditions constraining the growth of both the mother and her child.

Our study suggests that for parental BMI, it is the father rather than the mother that determines high risk. These parental differences relate to the components of high risk. Firstly for BMI, the parent-child association is a well replicated finding, but with no consistent differences for mothers and fathers,¹¹ but secondly, as mentioned above, maternal weight has a stronger effect than paternal weight on offspring birth weight. Heavier mothers have fatter adult offspring, and these offspring have a reduced risk of low weight at birth. For the high risk group with low birth weight and high adult BMI, these trends offset each other and

weaken the relation with maternal BMI. Whereas for fathers, the parent-child BMI relation is less affected by an opposing trend with weight at birth.

Neither breast feeding or parental age was an important determinant of high risk status. While it has been suspected that bottle fed infants have less ability to regulate their energy intake, evidence for a protective effect of breast feeding is inconclusive.¹¹ Parental age is of interest as a potential health influence on offspring because it is related both to the probability of gene mutations (particularly with respect to paternal age) and to the nature of the intrauterine environment.³⁹ Within this study, there was a range of parental ages, for example for mothers in 1958 the range was 15 to 47 years, with 5% aged <20 years, 61% aged 20–29 years, 31% aged 30–39 years, and 3% aged 40 years or more. Yet, we find no evidence that parental age is related to offspring being in the high risk group as adults in this study.

To conclude, maternal short stature, low social class, and smoking during pregnancy influenced the development of high risk status. The causes of high risk in this generation born in 1958 therefore seem to reside in utero and even earlier, in the mother's lifetime, with adverse conditions having a detrimental affect and favourable conditions protecting against high risk. The combination of low birth weight and high adult BMI is established during childhood, during which some linear growth acceleration occurs, though resulting in short adult stature. This pattern of linear growth is itself associated with adult disease^{40–42} and may represent an additional health burden for the low birth weight/high BMI group.

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Funding: this project was funded by the MacArthur Foundation Network on SES and Health.

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