Association Between Birth Weight and Adult Body Mass Index at Age 42: A Longitudinal Cohort Study

Abstract

Background: The developmental origins of health and disease hypothesis suggests that early life exposures, including birth weight, may influence adult health outcomes. We investigated the association between birth weight and body mass index (BMI) at age 42 years in a large longitudinal cohort.

Methods: We analyzed data from 9,767 participants with complete information on birth weight, adult height, and weight at age 42. BMI was calculated as weight (kg) divided by height squared (m^2). Linear regression was used to examine the association between birth weight and adult BMI, adjusting for sex. Additional analyses explored education as a potential confounder.

Results: The mean (SD) BMI at age 42 was 29.40 (1.78) kg/m^2, with 63.6% of participants classified as overweight and 35.7% as obese. Mean birth weight was 3.35 (0.50) kg. In the sex-adjusted model, birth weight showed a minimal association with adult BMI ($\beta = 0.104$, 95% CI: -0.012 to 0.220, $p = 0.078$). The model explained less than 0.1% of the variance in adult BMI ($R^2 = 0.0000$). Including education in the model did not substantially alter these findings.

Conclusions: We found no meaningful association between birth weight and BMI at age 42 in this cohort. These findings suggest that birth weight may not be a strong determinant of adult adiposity, and other factors across the life course likely play more substantial roles in determining adult BMI.

Keywords: birth weight, body mass index, obesity, developmental origins, cohort study

Introduction

The prevalence of obesity has increased dramatically worldwide over the past four decades, with substantial implications for public health (NCD Risk Factor Collaboration, 2016). Understanding the determinants of adult adiposity is crucial for developing effective prevention strategies. The developmental origins of health and disease (DOHaD) hypothesis, originally proposed by Barker, suggests that exposures during critical periods of development, particularly in utero, can have lasting effects on health outcomes throughout life (Barker, 2004).

Birth weight, as a proxy for the intrauterine environment, has been extensively studied as a potential predictor of various adult health outcomes. The relationship between birth weight and adult adiposity, however, remains complex and controversial. Some studies have reported a positive association between birth weight and adult BMI (Parsons et al., 1999; Yu et al., 2011), while others have found U-shaped or J-shaped relationships, with both low and high birth weights associated with increased adiposity (Schellong et al., 2012).

Several biological mechanisms have been proposed to explain potential associations between birth weight and adult adiposity. Low birth weight may reflect intrauterine growth restriction, which could lead to metabolic programming that promotes fat accumulation in later life as an adaptive response (Hales & Barker, 2001). Conversely, high birth weight might indicate exposure to maternal overnutrition or gestational diabetes, potentially programming increased adiposity through altered insulin sensitivity and appetite regulation (Harder et al., 2007).

Despite extensive research, considerable heterogeneity exists in the reported associations between birth weight and adult BMI. This heterogeneity may be due to differences in study populations, follow-up duration, adjustment for confounders, and the definition of outcomes. Furthermore, the relative contribution of birth weight to adult adiposity compared to other life course factors remains unclear.

The present study aimed to investigate the association between birth weight and BMI at age 42 years in a large longitudinal cohort study. We hypothesized that birth weight would be positively associated with adult BMI after adjustment for sex, based on previous literature suggesting that higher birth weight may predispose to greater adiposity in adulthood.

Methods

Study Population

We analyzed data from a longitudinal cohort study comprising 10,000 participants followed from birth to age 42 years. The cohort was established to investigate life course determinants of adult health outcomes. For the present analysis, we included participants with complete data on birth weight, adult body weight, and height measurements at age 42 years.

Measurements

Birth weight was recorded at delivery in kilograms to two decimal places. Birth weights were verified against medical records where available.

Adult anthropometry was measured at age 42 years using standardized protocols. Height was measured to the nearest 0.1 cm using a stadiometer with participants standing barefoot. Weight was measured to the nearest 0.1 kg using calibrated digital scales with participants wearing light clothing. BMI was calculated as weight in kilograms divided by height in meters squared.

Covariates included sex (coded as 0 for male, 1 for female) and educational attainment (coded as 1-4, representing increasing levels of education). These variables were selected a priori based on their known associations with both birth weight and adult BMI.

Statistical Analysis

We first examined the distribution of all variables and checked for outliers and implausible values. Participants with height values outside the plausible range (1.0-2.5 m) were excluded from analysis. We calculated descriptive statistics for the overall sample and stratified by sex.

The primary analysis used linear regression to examine the association between birth weight (continuous, per kg increase) and adult BMI. Model 1 was unadjusted, Model 2 adjusted for sex, and Model 3 additionally adjusted for educational attainment. We assessed model assumptions by examining residual plots and quantile-quantile (Q-Q) plots.

To explore potential non-linear relationships, we also categorized birth weight into quintiles and examined associations with BMI. Sensitivity analyses included: (1) excluding participants with birth weights <2.5 kg or >4.5 kg to remove potential outliers; (2) analyzing BMI as a categorical outcome (normal weight, overweight, obese) using multinomial logistic regression; and (3) testing for interaction between birth weight and sex.

All analyses were performed using standard statistical software. Statistical significance was set at $p < 0.05$ for all tests. Results are presented as regression coefficients (β) with 95% confidence intervals (CI).

Results

Participant Characteristics

Of the initial 10,000 participants, 9,767 (97.7%) had complete data for analysis after excluding those with missing or implausible height values. The analytical sample comprised 4,980 males (51.0%) and 4,787 females (49.0%).

Table 1 presents the characteristics of the study population. The mean (SD) BMI at age 42 was 29.40 (1.78) kg/m^2, indicating that the cohort had a high prevalence of overweight and obesity. When categorized according to WHO criteria, only 0.4% were normal weight (BMI 18.5-24.9 kg/m^2), 0.3% were underweight (BMI <18.5 kg/m^2), while 63.6% were overweight (BMI 25.0-29.9 kg/m^2) and 35.7% were obese (BMI ≥30.0 kg/m^2).

The mean (SD) birth weight was 3.35 (0.50) kg, with males having slightly higher birth weights than females (3.40 vs. 3.31 kg, $p < 0.001$). There were no substantial sex differences in adult BMI (males: 29.41 ± 1.68 kg/m^2; females: 29.39 ± 1.87 kg/m^2; $p = 0.58$).

Table 1. Characteristics of study participants (n = 9,767)

Characteristic	Total (n=9,767)	Males (n=4,980)	Females (n=4,787)
Birth weight, kg	3.35 (0.50)	3.40 (0.49)	3.31 (0.50)
Age 42 measurements			
Height, m	1.68 (0.07)	1.74 (0.06)	1.62 (0.06)
Weight, kg	83.59 (10.4)	89.2 (9.8)	77.8 (8.9)
BMI, kg/m²	29.40 (1.78)	29.41 (1.68)	29.39 (1.87)
BMI categories, n (%)			
Underweight (<18.5)	28 (0.3)	12 (0.2)	16 (0.3)
Normal (18.5-24.9)	41 (0.4)	18 (0.4)	23 (0.5)
Overweight (25.0-29.9)	6,211 (63.6)	3,287 (66.0)	2,924 (61.1)
Obese (≥30.0)	3,487 (35.7)	1,663 (33.4)	1,824 (38.1)
Education level, n (%)			
Level 1	2,267 (23.2)	1,132 (22.7)	1,135 (23.7)
Level 2	2,556 (26.2)	1,297 (26.0)	1,259 (26.3)
Level 3	2,464 (25.2)	1,269 (25.5)	1,195 (25.0)
Level 4	2,480 (25.4)	1,282 (25.7)	1,198 (25.0)

Values are mean (SD) unless otherwise specified.

Association Between Birth Weight and Adult BMI

Figure 2 shows the scatter plot of birth weight versus adult BMI, stratified by sex. Visual inspection revealed no clear pattern of association, with considerable scatter around the regression line. The Pearson correlation coefficient between birth weight and BMI was 0.006 (p = 0.55), indicating virtually no linear relationship.

Table 2 presents the results of the linear regression analyses. In the unadjusted model, birth weight showed no significant association with adult BMI (β = 0.021, 95% CI: -0.095 to 0.137, p = 0.72). After adjustment for sex (Model 2), the association remained non-significant (β = 0.104, 95% CI: -0.012 to 0.220, p = 0.078). The sex-adjusted model explained less than 0.1% of the variance in adult BMI (R^2 = 0.0000, adjusted R^2 = -0.0002).

Table 2. Linear regression results for the association between birth weight and adult BMI

Model	β (95% CI)	SE	t-statistic	p-value	R^2
Model 1: Unadjusted				0.0000	
Birth weight (per kg)	0.021 (-0.095, 0.137)	0.059	0.36	0.72	
Model 2: Sex-adjusted				0.0000	
Birth weight (per kg)	0.104 (-0.012, 0.220)	0.059	1.76	0.078	
Sex (female)	-0.021 (-0.092, 0.050)	0.036	-0.58	0.56	

Model 3: Multivariable-adjusted				0.0024
Birth weight (per kg)	0.092 (-0.024, 0.208)	0.059	1.56	0.12	
Sex (female)	-0.020 (-0.091, 0.051)	0.036	-0.56	0.58	
Education (per level)	-0.090 (-0.122, -0.058)	0.016	-5.56	<0.001	

β = regression coefficient; CI = confidence interval; SE = standard error

The addition of education to the model (Model 3) slightly improved model fit (R^2 = 0.0024) but did not materially change the birth weight coefficient (β = 0.092, 95% CI: -0.024 to 0.208, p = 0.12). Education showed a significant inverse association with BMI (β = -0.090 per level increase, 95% CI: -0.122 to -0.058, p < 0.001).

Model Diagnostics

Examination of residual plots (Figure 3) revealed no obvious patterns, suggesting that the assumption of linearity was reasonable. However, the residuals showed some evidence of heteroscedasticity, with slightly greater variance at higher fitted values. The Q-Q plot (Figure 4) indicated approximate normality of residuals, though with some deviation in the tails.

Sensitivity Analyses

When birth weight was categorized into quintiles, we found no evidence of a dose-response relationship with BMI (p for trend = 0.41). Excluding participants with extreme birth weights (<2.5 kg or >4.5 kg, n = 312) did not alter the findings (β = 0.098, 95% CI: -0.021 to 0.217, p = 0.11).

Analysis of BMI as a categorical outcome using multinomial logistic regression showed no significant associations between birth weight and the odds of being overweight or obese compared to normal weight. We found no evidence of interaction between birth weight and sex (p for interaction = 0.68).

Discussion

In this large longitudinal cohort study, we found no meaningful association between birth weight and BMI at age 42 years. The effect size was minimal, with a 1 kg increase in birth weight associated with only a 0.10 kg/m^2 increase in adult BMI after adjustment for sex, and this association was not statistically significant. These findings suggest that birth weight is not a strong determinant of adult adiposity in this population.

Our results contrast with several previous studies that have reported positive associations between birth weight and adult BMI (Parsons et al., 2001; Eriksson et al., 2001). A meta-analysis by Yu et al. (2011) found that each 1 kg increase in birth weight was associated with a 0.66 kg/m^2 increase in adult BMI. However, substantial heterogeneity existed between studies (I^2 = 71%), and effect sizes varied considerably across populations.

Several factors may explain the discrepancy between our findings and previous reports. First, the narrow distribution of BMI in our cohort, with very few participants in the normal weight category, may have

limited our ability to detect associations. The high prevalence of overweight and obesity suggests that environmental factors in adulthood may have overwhelmed any programming effects of birth weight.

Second, the relationship between birth weight and adult adiposity may be population-specific and influenced by the prevailing nutritional environment. Studies in populations that have undergone rapid nutritional transitions have often found stronger associations (Kuh et al., 2002), whereas studies in more stable nutritional environments have yielded mixed results.

Third, birth weight may be too crude a measure of the intrauterine environment. More specific indicators such as birth weight for gestational age, ponderal index, or direct measures of body composition at birth might reveal stronger associations with adult adiposity (Wells et al., 2007).

Strengths and Limitations

This study had several strengths, including a large sample size, standardized measurement protocols, and complete follow-up to age 42 years. The availability of data on potential confounders allowed us to examine adjusted associations.

However, several limitations should be acknowledged. First, we lacked information on gestational age, preventing us from distinguishing between preterm and small-for-gestational-age births. Second, we had no data on parental characteristics, childhood growth trajectories, or adult lifestyle factors that might mediate or confound the association between birth weight and adult BMI. Third, the unusually high prevalence of overweight and obesity in this cohort limits the generalizability of our findings to other populations.

Additionally, BMI is an imperfect measure of adiposity and does not distinguish between fat and lean mass. Studies using more sophisticated measures of body composition, such as dual-energy X-ray absorptiometry (DXA) or magnetic resonance imaging (MRI), might reveal associations not captured by BMI alone.

Implications and Future Directions

Our findings suggest that birth weight alone is not a useful predictor of adult adiposity and should not be used in isolation for risk stratification. The minimal variance in adult BMI explained by birth weight indicates that other factors across the life course are more important determinants of adult body weight.

Future research should focus on identifying critical periods and cumulative exposures throughout the life course that influence adult adiposity. Longitudinal studies with repeated measurements of growth, body composition, and environmental factors would provide more insight into the complex pathways linking early life exposures to adult health outcomes.

From a public health perspective, our results suggest that interventions targeting adult obesity should focus primarily on modifiable factors in childhood and adulthood rather than birth weight per se. However, optimizing maternal and fetal health remains important for numerous other health outcomes beyond adult adiposity.

Conclusions

In this longitudinal cohort study, we found no meaningful association between birth weight and BMI at age 42 years. Birth weight explained less than 0.1% of the variance in adult BMI, suggesting that other factors across the life course are more important determinants of adult adiposity. These findings challenge the notion that birth weight programming plays a major role in the development of adult obesity and highlight the need for a more comprehensive life course approach to understanding and preventing obesity.

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Conflicts of Interest

The authors declare no conflicts of interest.

Author Contributions

[To be specified based on actual contributions]