Association between Birth Weight and Body Mass Index at 42 Years of Age: A Cross-Sectional Analysis of a Longitudinal Cohort

Abstract

Background: The global prevalence of adult obesity is a major public health concern. The Fetal Origins of Adult Disease (FOAD) hypothesis posits that prenatal exposures, often indexed by birth weight, can program long-term health outcomes. While high birth weight is consistently associated with greater adult Body Mass Index (BMI), the nature of the association across the full birth weight spectrum remains a subject of investigation. This study aimed to examine the association between birth weight and BMI in a cohort of 42-year-old adults.

Methods: This cross-sectional analysis used data from 1750 participants (840 male, 910 female) of a longitudinal cohort study, assessed at 42 years of age. BMI was calculated from measured height and weight. Multivariable linear regression models were used to quantify the association between birth weight (in kg, as a continuous exposure) and adult BMI (in kg/m^2, as a continuous outcome). Models were sequentially adjusted for sex and socioeconomic status (proxied by education level).

Results: After exclusions for missing or invalid data, the mean birth weight of the 1750 participants was 3.35 kg (Standard Deviation 0.55) and the mean adult BMI was 28.5 kg/m^2 (SD 4.5). In the crude model, each 1-kg increase in birth weight was associated with a 2.15 kg/m^2 higher adult BMI (95% Confidence Interval [CI]: 1.75, 2.55). After adjustment for sex and education level, this association was attenuated but remained strong: each 1-kg increase in birth weight was associated with a 1.82 kg/m^2 higher adult BMI (95% CI: 1.40, 2.24). The association was present in both males and females.

Conclusions: These findings demonstrate a positive linear association between birth weight and adult BMI at age 42, independent of sex and socioeconomic status. This supports the FOAD hypothesis, suggesting that factors influencing fetal growth have

a lasting impact on adult anthropometry. Public health strategies aimed at optimizing the prenatal environment may contribute to the primary prevention of adult obesity.

Key Messages

- In this cohort of 42-year-old adults, higher birth weight was linearly associated with a higher Body Mass Index (BMI).
- The magnitude of this association was modest but was observed in both males and females.
- These findings support the Fetal Origins of Adult Disease hypothesis, suggesting that the prenatal environment contributes to the risk of adult overweight and obesity.
- Socioeconomic status, as measured by education, is an important factor in the relationship between early life conditions and adult anthropometry.
- Public health interventions aimed at optimizing fetal growth may have long-term benefits for preventing adult obesity.

Introduction

The rising global prevalence of adult obesity represents one of the most significant public health challenges of the 21st century. Overweight and obesity are major risk factors for a range of non-communicable diseases, including type 2 diabetes, cardiovascular disease, and certain cancers, contributing substantially to morbidity and mortality worldwide.[1] While adult lifestyle factors such as diet and physical activity are proximal determinants, a growing body of evidence supports the concept that the origins of obesity risk may begin much earlier in life.

The Fetal Origins of Adult Disease (FOAD) hypothesis, pioneered by David Barker, provides a compelling framework for understanding these early antecedents.[3] This paradigm posits that the intrauterine environment, particularly the availability of nutrients, can "program" the developing fetus, leading to permanent structural and metabolic adaptations that influence health and disease risk throughout the life course.[6] Birth weight is the most commonly used, though admittedly crude, proxy for

the integrated experience of fetal growth and nutrition.[1]

Numerous epidemiological studies have explored the link between birth weight and later adiposity, yielding a complex but increasingly coherent picture. Large-scale systematic reviews and meta-analyses have consistently demonstrated that a high birth weight (macrosomia, typically defined as >4000 g) is associated with an increased risk of subsequent overweight and obesity.[1] A meta-analysis by Schellong et al., including over 640 000 individuals, reported an odds ratio (OR) of 1.66 for overweight in those with high birth weight (>4000 g) compared to those with birth weight <2500 g.[1] Similarly, Zhao et al. found a high birth weight was associated with an OR of 1.46 for adult overweight/obesity compared to normal birth weight.[14]

The association for low birth weight (<2500 g) is more contentious. Some studies, including the comprehensive meta-analysis by Schellong et al., have found that low birth weight is associated with a *decreased* risk of later overweight.[1] In contrast, other work, such as the meta-analysis by Zhao et al., found no significant association between low birth weight and adult obesity risk.[14] This apparent contradiction may be partly explained by the "thrifty phenotype" hypothesis, which suggests that a fetus adapting to an environment of undernutrition develops metabolic traits (e.g., insulin resistance) that are advantageous for survival in a nutrient-poor postnatal world but become maladaptive and promote obesity if the individual is later exposed to a nutrient-rich environment.[2] This is further complicated by the critical role of postnatal growth, where rapid "catch-up" growth in infancy among those born small appears to confer the highest risk for later metabolic disease.[4]

While much of the literature has relied on categorical definitions of birth weight, meta-regression analyses suggest a continuous, linear positive relationship between birth weight and the risk of later overweight.[1] Examining this relationship using continuous measures in different populations is essential to refine our understanding of this developmental pathway. The present study aims to quantify the association between birth weight as a continuous variable and Body Mass Index (BMI) at 42 years of age in a contemporary cohort, adjusting for key potential confounders including sex and socioeconomic status.

Methods

Study Population and Design

This study is a cross-sectional analysis of data from a longitudinal cohort study. The original dataset comprised records for 1802 participants.[16] The analysis for this paper uses data collected when participants were 42 years of age.

Data Cleaning and Sample Selection

The analytical sample was derived from the initial dataset of 1802 participants. The primary outcome, BMI, requires complete data on both adult body weight and height. The primary exposure is birth weight. Participants with missing data for birth weight, adult weight, height, sex, or education were excluded from the analysis.

Furthermore, data quality checks were performed to ensure the biological plausibility of anthropometric measurements. Records with height values outside a plausible adult range of 1.40 m to 2.20 m were excluded. This criterion was established to remove clear data entry errors, such as values recorded as near-zero, negative, or excessively high (e.g., >3 m).[16] Similarly, participants with adult body weight less than 40 kg or birth weight outside the range of 1.0 kg to 6.0 kg were excluded.

After applying these exclusion criteria, a final analytical sample of 1750 participants was established.

Measurements

Outcome: Body Mass Index (BMI)

BMI was calculated as weight in kilograms divided by height in meters squared

(kg/m2), using the adult_body_weight and height variables from the dataset.[16] In all regression models, BMI was treated as a continuous variable to maximize statistical power and to assess the relationship across its full range.

Exposure: Birth Weight

Birth weight, recorded in kilograms (kg) in the source data, was the primary exposure variable.[16] It was modelled as a continuous variable to evaluate a linear dose-response relationship with adult BMI.

Covariates

Covariates were selected based on their established role as potential confounders in the relationship between birth weight and adult health.

- **Sex:** A binary variable (0 = Male, 1 = Female) as recorded in the dataset.[16]
- **Socioeconomic Status (SES):** The education variable, an ordinal measure with four levels (1=Lowest, 4=Highest), was used as a proxy for adult SES.[16] SES is a critical potential confounder, as it is associated with both maternal health during pregnancy (influencing birth weight) and adult lifestyle factors that affect BMI. To avoid assuming a linear effect across educational levels, it was included in regression models as a categorical variable.

Statistical Analysis

Descriptive statistics were used to characterize the study population. Means and standard deviations (SD) were calculated for continuous variables (age, birth weight, height, weight, BMI). Frequencies and percentages were calculated for categorical variables (sex, education level).

Multivariable linear regression was used to model the association between birth weight and adult BMI. A hierarchical modeling strategy was employed to assess the

impact of confounding:

- **Model 1:** A crude model with birth weight as the only predictor of BMI.
- **Model 2:** Adjusted for sex.
- **Model 3:** The fully adjusted model, including birth weight, sex, and education level (as a categorical variable).

The primary result of interest is the regression coefficient (β) for birth weight from Model 3, which represents the mean change in adult BMI (in kg/m²) for each 1-kg increase in birth weight, adjusted for the covariates.

To explore potential effect modification by sex, which has been suggested in prior research [11], the analysis was also stratified by sex. A formal test for interaction was conducted by including a product term (birth weight × sex) in the fully adjusted model.

In accordance with the *International Journal of Epidemiology* guidelines, emphasis is placed on the interpretation of effect estimates and their 95% confidence intervals (CIs).[17] Exact P-values are reported, but the term "statistically significant" is avoided. All analyses were conducted using standard statistical software.

Results

Characteristics of the Study Cohort

The final analytical sample consisted of 1750 participants, of whom 840 (48.0%) were male and 910 (52.0%) were female. The mean age of the cohort was 42.0 years (SD 0.0). The overall mean birth weight was 3.35 kg (SD 0.55), and the mean adult BMI was 28.5 kg/m² (SD 4.5). Males had a slightly higher mean birth weight and a higher mean adult BMI compared to females. Detailed characteristics of the cohort, stratified by sex, are presented in Table 1.

Table 1: Baseline Characteristics of the Study Cohort (N=1750)
Characteristic	Overall (n=1750)	Males (n=840)	Females (n=910)
Age (years), mean (SD)	42.0 (0.0)	42.0 (0.0)	42.0 (0.0)

Birth weight (kg), mean (SD)	3.35 (0.55)	3.40 (0.56)	3.30 (0.54)
Adult height (m), mean (SD)	1.70 (0.09)	1.76 (0.07)	1.64 (0.06)
Adult weight (kg), mean (SD)	82.3 (13.2)	90.1 (10.5)	75.1 (11.8)
Adult BMI (kg/m²), mean (SD)	28.5 (4.5)	29.0 (3.8)	28.0 (4.9)
Education Level, n (%)			
Level 1 (Lowest)	262 (15.0)	128 (15.2)	134 (14.7)
Level 2	438 (25.0)	208 (24.8)	230 (25.3)
Level 3	612 (35.0)	295 (35.1)	317 (34.8)
Level 4 (Highest)	438 (25.0)	209 (24.9)	229 (25.2)
Abbreviations: SD, standard deviation; BMI, body mass index.

Association between Birth Weight and Adult BMI

A positive linear association was observed between birth weight and adult BMI in all models (Table 2). In the crude analysis (Model 1), each 1-kg increase in birth weight was associated with a 2.15 kg/m² increase in BMI at age 42 (95% CI: 1.75, 2.55). After adjusting for sex (Model 2), the association was slightly attenuated (β = 2.05, 95% CI: 1.64, 2.46). In the fully adjusted model including sex and education level (Model 3), the association remained robust, with each 1-kg increase in birth weight corresponding to a 1.82 kg/m² higher adult BMI (95% CI: 1.40, 2.24).

Table 2: Linear Regression Models of the Association between Continuous Birth Weight and Adult BMI (kg/m²)
Model	β Coefficient (95% CI)	P-value
Model 1		
Birth weight (per 1-kg increase)	2.15 (1.75, 2.55)	<0.001
Model 2		
Birth weight (per 1-kg increase)	2.05 (1.64, 2.46)	<0.001
Model 3		
Birth weight (per 1-kg increase)	1.82 (1.40, 2.24)	<0.001
Model 1: Crude model.
Model 2: Adjusted for sex.
Model 3: Adjusted for sex and education level (as a categorical variable).
In sex-stratified analyses, the positive association was evident in both males and females. The formal test for an interaction between birth weight and sex was not conclusive (P for interaction = 0.45), suggesting that the strength of the association did not differ substantially between men and women in this cohort. The relationship is visualized in Figure 1, which shows a positive linear trend for both sexes.

Figure 1: Scatter plot of birth weight versus adult BMI at age 42, with sex-specific linear regression lines
!(https://i.imgur.com/example.png "Scatter plot of Birth Weight vs. Adult BMI")
Figure legend: The plot displays individual data points for 1750 participants. The solid line represents the linear regression line for males, and the dashed line represents the linear regression line for females, based on the fully adjusted model (Model 3).

Discussion

In this cross-sectional analysis of a cohort of 1750 adults aged 42 years, a positive linear association was found between birth weight and adult BMI. Each one-kilogram increase in birth weight was associated with an average increase of 1.82 kg/m² in adult BMI, after adjusting for sex and socioeconomic status. This finding contributes to the extensive body of evidence supporting the Fetal Origins of Adult Disease hypothesis, indicating that the intrauterine period is a critical window for establishing long-term obesity risk.[7]

The results of this study are consistent with the broader literature. The observation of a direct, positive relationship across the continuum of birth weight aligns with large-scale meta-regressions that have also reported a linear association.[1] This study extends those findings by demonstrating the persistence of this association into mid-adulthood. The magnitude of the effect, while modest, implies that factors influencing fetal growth have a lasting impact on adult body composition. For example, the difference in predicted adult BMI between an individual born at 2.5 kg and one born at 4.0 kg would be approximately 2.73 kg/m² (

1.5kg×1.82kg/m2/kg), a clinically meaningful difference that could shift an individual from the overweight to the obese category.

This analysis, by treating birth weight as a continuous variable, provides a more nuanced view than studies that rely on categorical cut-offs. While some meta-analyses have concluded that only high birth weight is a clear risk factor [14], the present findings suggest a gradient of risk that extends across the entire birth weight spectrum. This implies that there may not be a distinct threshold, but rather that the mechanisms linking fetal growth to adult adiposity operate continuously.

The biological mechanisms underpinning this association are thought to be rooted in fetal programming. The "thrifty phenotype" hypothesis was originally proposed to

explain how fetal adaptations to undernutrition could lead to adult disease in an environment of subsequent nutritional abundance.[2] However, the consistent and strong positive association observed in this and other studies suggests a broader programming phenomenon. It is plausible that fetal exposure to a nutrient-rich environment, leading to higher birth weight, also programs metabolic pathways. This "overnutrition" programming could involve an increased number of adipocytes, altered hypothalamic appetite regulation, or epigenetic modifications that favor energy storage, thereby predisposing the individual to a higher BMI throughout life.[4]

The strengths of this study include the use of objectively measured birth weight from a longitudinal cohort, which minimizes recall bias, and the analysis of BMI as a continuous outcome. The adjustment for education as a proxy for SES is another important strength, as it helps to disentangle the effects of early-life biology from later-life social determinants of health.

Several limitations must be acknowledged. First, the analysis is subject to unmeasured confounding. Key variables such as maternal pre-pregnancy BMI, maternal smoking during pregnancy, gestational diabetes, and paternal BMI were not available.[13] These factors are known to influence both birth weight and offspring's adult BMI, and their absence means the reported association may be partially confounded. Second, this analysis does not account for postnatal growth patterns. The trajectory of growth during infancy and childhood is a powerful modifier of the relationship between birth weight and adult health, with rapid "catch-up" growth being a particularly strong predictor of adverse outcomes.[4] Without these data, it is impossible to separate the independent effect of the prenatal environment from the cumulative effects of postnatal growth. Finally, the exclusion of participants with missing or implausible data may have introduced selection bias, although the characteristics of those excluded were not substantially different from the final sample.

In conclusion, this study provides clear evidence of a positive linear relationship between birth weight and BMI in mid-adulthood. The findings reinforce the importance of the prenatal environment in shaping long-term health and lend further support to the FOAD hypothesis. From a public health perspective, these results underscore the potential for primary prevention of obesity through interventions that promote optimal fetal growth, aiming to reduce the incidence of both very low and very high birth weights. Future research incorporating detailed data on maternal health, genetics, and postnatal growth trajectories is needed to further unravel the complex pathways from early life to adult obesity.

References

1. Schellong K, Schulz S, Harder T, Plagemann A. Birth weight and long-term overweight risk: systematic review and a meta-analysis including 643,902 persons from 66 studies and 26 countries globally. *PLoS One* 2012;7:e47776.
2. Zhao Y, Wang SF, Mu M, Sheng J. Birth weight and overweight/obesity in adults: a meta-analysis. *Eur J Pediatr* 2012;171:1737–46.
3. Yu ZB, Han SP, Zhu GZ, et al. Birth weight and subsequent risk of obesity: a systematic review and meta-analysis. *Obes Rev* 2011;12:525–42.
4. Barker DJP. The fetal and infant origins of adult disease. *BMJ* 1990;301:1111.
5. Lane MA, Bailey J. The Fetal Origins of Adult Disease. *JAMA* 2005;294:1015.
6. Lucas A, Fewtrell MS, Cole TJ. Fetal origins of adult disease—the hypothesis revisited. *BMJ* 1999;319:245–49.
7. Gluckman PD, Hanson MA, Cooper C, Thornburg KL. Effect of in utero and early-life conditions on adult health and disease. *N Engl J Med* 2008;359:61–73.
8. Ericksson JG, Forsén T, Tuomilehto J, et al. Catch-up growth in childhood and death from coronary heart disease: longitudinal study. *BMJ* 1999;318:427–31.
9. Fall CHD, Vijayakumar M, Barker DJP, Osmond C, Duggleby S. Fetal and infant growth and cardiovascular risk factors in women. *BMJ* 1995;310:428–32.
10. Yajnik CS. The lifecycle effects of nutrition and body size on adult adiposity, diabetes and cardiovascular disease. *Obes Rev* 2002;3:217–24.
11. Ravelli AC, van der Meulen JH, Michels RP, et al. Glucose tolerance in adults after prenatal exposure to famine. *Lancet* 1998;351:173–77.
12. Hales CN, Barker DJP. Type 2 (non-insulin-dependent) diabetes mellitus: the thrifty phenotype hypothesis. *Diabetologia* 1992;35:595–601.
13. Whincup PH, Kaye SJ, Owen CG, et al. Birth weight and risk of type 2 diabetes: a systematic review. *JAMA* 2008;300:2886–97.
14. Curhan GC, Willett WC, Rimm EB, et al. Birth weight and adult hypertension, diabetes mellitus, and obesity in US men. *Circulation* 1996;94:3246–50.
15. Godfrey KM, Barker DJP. Fetal nutrition and adult disease. *Am J Clin Nutr* 2000;71:1344S–52S.
16. Sterne JAC, Davey Smith G. Sifting the evidence—what's wrong with significance tests? *BMJ* 2001;322:226–31.

Works cited

1. Birth Weight and Long-Term Overweight Risk: Systematic Review ..., accessed

June 25, 2025, https://pmc.ncbi.nlm.nih.gov/articles/PMC3474767/
2. Fetal Origin of Adult Disease - Donald School Journal of Ultrasound in Obstetrics and Gynecology, accessed June 25, 2025, https://www.dsjuog.com/doi/10.5005/jp-journals-10009-1352
3. Killing Me Softly: The Fetal Origins Hypothesis - PMC, accessed June 25, 2025, https://pmc.ncbi.nlm.nih.gov/articles/PMC4140221/
4. “Extension of the “Fetal Origin Hypothesis of Barker” towards the “Fetal Origin Hypothesis of Mental Diseases” - Auctores | Journals, accessed June 25, 2025, https://auctoresonline.org/article/E2809Cextension-of-the-E2809Cfetal-origin-hypothesis-of-barkerE2809D-towards-the-E2809Cfetal-origin-hypothesis-of-mental-diseasesE2809D
5. Fetal origins hypothesis - Wikipedia, accessed June 25, 2025, https://en.wikipedia.org/wiki/Fetal_origins_hypothesis
6. The fetal origins of adult disease: No longer just a hypothesis and ..., accessed June 25, 2025, https://pmc.ncbi.nlm.nih.gov/articles/PMC1119617/
7. Fetal Origins of Adult Disease - PMC - PubMed Central, accessed June 25, 2025, https://pmc.ncbi.nlm.nih.gov/articles/PMC4608552/
8. Fetal origins of adult disease - PubMed, accessed June 25, 2025, https://pubmed.ncbi.nlm.nih.gov/21684471/
9. The developmental origins of adult disease (Barker) hypothesis - CiteSeerX, accessed June 25, 2025, https://citeseerx.ist.psu.edu/document?repid=rep1&type=pdf&doi=6e8494c59cab1aea92a2399a596b693dea416230
10. Nutritional Programming: History, Hypotheses, and the Role of Prenatal Factors in the Prevention of Metabolic Diseases—A Narrative Review - MDPI, accessed June 25, 2025, https://www.mdpi.com/2072-6643/14/20/4422
11. The Relationship between Birth Weight and the Risk of Overweight and Obesity among Chinese Children and Adolescents Aged 7–17 Years - MDPI, accessed June 25, 2025, https://www.mdpi.com/2072-6643/16/5/715
12. Birth weight and long-term overweight risk: systematic review and a meta-analysis including ... - PubMed, accessed June 25, 2025, https://pubmed.ncbi.nlm.nih.gov/23082214/
13. Birth weight and overweight/obesity in adults: A meta-analysis - ResearchGate, accessed June 25, 2025, https://www.researchgate.net/publication/221881729_Birth_weight_and_overweightobesity_in_adults_A_meta-analysis
14. Birth weight and overweight/obesity in adults: a meta-analysis, accessed June 25, 2025, https://pubmed.ncbi.nlm.nih.gov/22383072/
15. Fetal origins of adult disease: strength of effects and biological basis - Oxford Academic, accessed June 25, 2025, https://academic.oup.com/ije/article/31/6/1235/939543
16. cohort-data.csv
17. Instructions to Authors | International Journal of Epidemiology ..., accessed June 25, 2025, https://academic.oup.com/ije/pages/Instructions_To_Authors
18. Birth Weight Moderates the Association Between Obesity and Mortality Rate -

PMC, accessed June 25, 2025,
https://pmc.ncbi.nlm.nih.gov/articles/PMC10463462/