

PAPER

Obesity from cradle to grave

J Eriksson^{1*}, T Forsén¹, C Osmond² and D Barker²

¹Department of Epidemiology and Health Promotion, National Public Health Institute, Diabetes and Genetic Epidemiology Unit, Mannerheimintie, Helsinki, Finland; and ²MRC Environmental Epidemiology Unit, University of Southampton, Southampton General Hospital, Southampton, UK

BACKGROUND: Obesity is known to track from early life into adult life.

OBJECTIVE: To examine the relation of obesity in adult life to growth and living conditions during childhood.

DESIGN: Birth cohort study.

PARTICIPANTS: A total of 4515 people (2135 men and 2380 women) who were born at Helsinki University Central Hospital between 1934 and 1944, who attended child welfare clinics and were still resident in Finland in the year 2000.

MEASUREMENTS: Incidence of obesity based upon lifetime maximum body mass index (BMI) ascertained from a postal questionnaire and defined as a BMI ≥ 30 kg/m². The main explanatory measurements were size at birth, childhood growth, and socioeconomic status in childhood and in adult life.

RESULTS: The cumulative incidence of obesity was 33.8% in men and 32.4% in women. The incidence rose with increasing body size at birth. From birth the mean weight and BMI of people who later became obese exceeded the average and remained above average at a statistically significant level at all ages from 6 months to 12 y. Childhood BMI was a stronger predictor of adult obesity than body size at birth. A higher maternal BMI in pregnancy was associated with a more rapid childhood growth and an increased risk of becoming obese in adult life. Higher socioeconomic status and better educational attainment were associated with a lower prevalence of obesity. There was no association between the duration of breastfeeding and later obesity.

CONCLUSIONS: These results emphasize the importance of early life factors in the pathogenesis of adult obesity.

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Keywords: birth weight; body mass index; childhood growth; breastfeeding; socioeconomic status

Introduction

Obesity is known to track from childhood into adult life.^{1,2} In a study of people born in Helsinki, we have shown that those who had a high body mass index (BMI) from 7 y onwards were at increased risk of becoming obese in adult life.³ However, in this cohort—born 1924–1933—there were no data on growth between birth and 7 y. Therefore, it was not possible to determine at what age rapid weight gain began. This could be important since weight gain during certain periods of childhood is especially important.^{4,5} After the age of 2 y, the body fatness of young children, as measured by BMI, falls to a minimum around 6 y of age before rising again, the so-called adiposity

rebound. An early adiposity rebound predicts obesity in later life.

We report here on associations between early growth and obesity in a cohort of 4515 men and women born in Helsinki during 1934–1944, for whom serial measurements of height and weight from birth to the age of 12 y were recorded in obstetric, child welfare clinic (0–7 y) and school health records (7–12 y). In this cohort, the existence of child welfare clinic records allows us to examine the effects of weight gain during infancy and early childhood, in relation to adult obesity.

Methods

We studied a sample of 8760 men and women who were born at Helsinki University Central Hospital during 1934–1944, who attended child welfare clinics in the city of Helsinki and who were still resident in Finland in 1971. The majority (77%) of the subjects also went to school

*Correspondence: Dr J Eriksson, National Public Health Institute, Department of Epidemiology and Health Promotion, Mannerheimintie 166, FIN-00300, Helsinki, Finland.

E-mail: johan.eriksson@ktl.fi

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in Helsinki. Details of the birth records, child welfare clinic records and school health records have been previously described.⁶ The birth records include data on the newborn babies including birth weight, birth length, placental weight and head circumference. They also include information about the occupation of the parents, and data on the mother, for example, age, height and date of the last menstrual period as well as body weight measured on admission in labor. The child welfare clinic records include serial measurements of height and weight between birth and 7 y of age. These records also include information on living conditions in childhood, socioeconomic factors and duration of breastfeeding. The school records include measurements of height and weight recorded at periodic medical examinations from age 7 y onwards, as well as information on socioeconomic factors during school years.

A unique personal identifier has been assigned to every Finnish citizen at the latest in 1971. We used this number to trace people belonging to the cohort. All 7047 people belonging to the original cohort who were still alive and resident in Finland in the year 2000 were sent a questionnaire, in order to obtain information about their current height and weight and their maximum body weight during adult life, excluding pregnancy weights in women. We used maximum lifetime BMI to assess the cumulative incidence of obesity, using $\text{BMI} \geq 30 \text{ kg/m}^2$ as the cut-off point.

A total of 4515 (2135 men and 2380 women) individuals provided adequate data on weight and height, and were included in the present study. Each individual had on average 18 (s.d.: 10) measurements of height and weight between birth and 12 y of age; nine (s.d.: 7) measurements from birth to 1 y, three (s.d.: 4) measurements from 1 to 6 y and six (s.d.: 4) measurements from 6 to 12 y. For example, at age 1 y of age, 4508 individuals had measurements of height and weight. The corresponding numbers at ages 3, 5, 7, 9 and 11 y were 4011, 3623, 3516, 3493 and 3318 individuals, respectively.

Using the fathers' occupation, which was recorded on the child welfare clinic records, we grouped the fathers of the men and women according to a social classification used by Statistics Finland. Overall 61.3% of the fathers were laborers and 20.1% were classified as lower middle class. Together these constitute the lower social class as opposed to the upper social class to which 11.8% of the fathers belonged. The social class of 6.8% of the cohort was unclassified/missing.

Through Statistics Finland, we obtained data on level of education achieved, recorded in the 1970 Census. Based upon a classification used by Statistics Finland and based upon the International Standard Classification of education, we grouped the people into three levels of education: high (upper secondary and tertiary), middle (lower secondary) and low—based upon an original nine group classification scale.

Statistical analyses

Tests for trends were based on multivariate logistic regression using continuous variables, adjusted for year of birth and gender. We converted each height, weight and BMI measurement for each child to a Z-score using the method of Royston.⁷ High degree polynomials (sixth order) were applied to fit the means and absolute residuals of the observed data set, treated as it was cross sectional, sometimes after transformation to adjust for any non-normality in the residuals. This enables each observation to be represented as a Z-score, using the exact age of measurement.

We interpolated between successive Z-scores with a piecewise linear function and obtained a Z-score at each birthday from age 0 to 12 y. These Z-scores were back-transformed to estimate height, weight and BMI at these ages. The onset of adiposity rebound was assessed by estimating BMI at each birthday from age 1 to 12 y. The timing of the adiposity rebound was that age between 1 and 12 y when BMI was the smallest.

Results

Table 1 shows the maternal, neonatal and childhood characteristics of the 2135 men and 2380 women in the cohort. BMIs in childhood at all ages assessed were mostly within normal range according to today's criteria. The cumulative lifetime incidence of obesity was 33.1% in the whole cohort, 33.8% among men and 32.4% among women. The association between current BMI and maximum lifetime BMI was strong ($r = 0.90$; $P < 0.0001$).

Size at birth

The cumulative incidence of obesity increased with increasing birth weight ($P = 0.01$, sexes combined) and ponderal index (birth weight/length³, $P = 0.001$) (Table 2). The positive relation between birth weight and adult obesity was statistically significant only among men. There were no trends with length at birth ($P = 0.60$), head circumference ($P = 0.45$) or gestational age ($P = 0.50$).

Growth in childhood

The subjects who became obese in adult life had above-average body weights and BMIs at all ages from birth to 12 y (all $P < 0.001$). They also had above-average heights at each age from 1 y onward. Table 3 shows that childhood BMI predicted adult obesity, as well at 6 months of age as at 11 y of age. The growth of the subjects who later became obese is shown in Figure 1, expressed as Z-scores. The Z-score for the whole cohort is set at zero, and a child maintaining a steady position as large or small in relation to other children would follow a horizontal path in the figure. Children who later became obese, however, had accelerated gain in weight and BMI, compared to the rest of the cohort.

Table 1 Maternal, neonatal and childhood characteristics of 2135 men and 2380 women born at Helsinki University Central Hospital during 1934–1944

Characteristics	Mean	s.d.	Range
Men			
Maternal height (cm)	160	6	136–195
Maternal BMI (kg/m ²)	26.4	2.9	13.4–43.4
Birth weight (g)	3457	497	1600–5170
Length at birth (cm)	50.6	2.1	41–59
Ponderal index (kg/m ³)	26.5	2.3	16.8–50.3
Height (cm) at age 1 y	76	3	64–87
Height (cm) at age 5 y	107	4	94–125
Height (cm) at age 11 y	142	6	123–167
Weight (kg) at age 1 y	10.4	1.5	5.7–17.0
Weight (kg) at age 5 y	16.3	2.2	10.7–29.0
Weight (kg) at age 11 y	35.4	5.3	23.1–68.0
BMI (kg/m ²) at age 1 y	17.7	1.6	11.7–23.0
BMI (kg/m ²) at age 5 y	14.7	1.2	11.2–20.5
BMI (kg/m ²) at age 11 y	17.2	1.7	13.1–26.0
Women			
Maternal height (cm)	160	6	142–178
Maternal BMI (kg/m ²)	26.3	2.8	18.4–42.7
Birth weight (g)	3331	460	1450–5400
Length at birth (cm)	49.9	1.8	41.0–56.0
Ponderal index (kg/m ³)	26.7	2.2	18.4–34.4
Height (cm) at age 1 y	74	3	59–90
Height (cm) at age 5 y	107	4	90–128
Height (cm) at age 11 y	143	6	119–166
Weight (kg) at age 1 y	9.7	1.4	4.4–15.2
Weight (kg) at age 5 y	16.1	2.2	9.6–28.3
Weight (kg) at age 11 y	35.9	6.3	22.8–75.4
BMI (kg/m ²) at age 1 y	17.3	1.6	12.1–27.1
BMI (kg/m ²) at age 5 y	14.7	1.3	10.8–20.0
BMI (kg/m ²) at age 11 y	17.4	2.1	12.6–30.7

Table 2 Cumulative incidence and 95% confidence intervals (in parentheses) of obesity according to birth weight and ponderal index in men and women

	Men	Women
Birth weight (g)		
–2500	31.3 (20.9–41.6)	32.9 (22.7–43.1)
–3000	31.3 (25.7–36.8)	31.4 (27.2–35.6)
–3500	31.8 (28.7–35.0)	32.3 (29.4–35.2)
–4000	35.4 (31.8–38.9)	31.6 (28.1–35.2)
>4000	39.5 (33.5–45.6)	39.5 (31.6–47.3)
P for trend	0.022	0.219
Ponderal index at birth (kg/m³)		
–25	30.5 (26.5–34.5)	29.3 (25.4–33.2)
–26	37.8 (32.7–43.0)	31.2 (26.6–35.8)
–27	31.6 (27.0–36.2)	31.1 (26.7–35.6)
–28	31.0 (26.2–35.9)	34.2 (29.5–38.9)
>28	37.8 (33.5–42.0)	35.2 (31.5–39.0)
P-values	0.04	0.008

Among those in the top 10th of BMI at age 1, 3, 5, 7, 9 and 11 y, the incidence of obesity in adult life was 41, 47, 48, 56, 61 and 64%, respectively.

Table 3 Cumulative incidence and 95% confidence intervals (in parentheses) of obesity according to BMI at 6 months and 11 y of age in men and women

BMI (kg/m ²)	Men	Women
6 months		
<16.3	28.6 (24.1–33.1)	27.5 (23.8–31.3)
–16.8	24.2 (19.6–28.8)	31.1 (26.8–35.5)
–17.3	36.3 (31.6–41.0)	33.7 (29.5–37.9)
–18.0	32.2 (28.2–36.3)	34.5 (30.4–38.7)
>18.0	44.1 (40.0–48.5)	36.8 (32.0–41.7)
P-values	<0.0001	0.001
11 y		
–15.8	16.2 (11.9–20.4)	19.1 (15.2–23.1)
–16.6	23.2 (18.8–27.6)	23.9 (19.2–28.6)
–17.4	31.3 (26.4–36.2)	33.5 (28.3–38.8)
–18.5	40.2 (35.0–45.4)	28.9 (23.7–34.0)
>18.5	63.6 (57.5–69.6)	51.8 (47.0–56.7)
P-values	<0.001	<0.001

The average age at adiposity rebound was 5.8 y (s.d.: 1.0), similar in boys and girls. Table 4 shows that children who had an adiposity rebound at an early age had a higher incidence of obesity in later life.

We assessed the combined effects of ponderal index at birth and BMI at age 11 y on the odds ratios for adult obesity (Table 5). Among people who were in the highest fifth of ponderal index at birth and BMI at 11 y, the risk of adult obesity was more than six times that among those whose ponderal index and BMI were in the lowest fifth. In a simultaneous logistic regression with ponderal index at birth and BMI at 11 y included, only BMI at age 11 y predicted adult obesity ($P < 0.0001$).

Maternal characteristics

Both maternal weight and maternal BMI in pregnancy were strongly and positively related to childhood BMI at each age studied ($P < 0.0001$, applies to each of the comparisons). Maternal BMI in pregnancy was strongly and positively related to incidence of obesity in adult life (Table 6). Maternal height was not related to obesity in the offspring ($P = 0.83$). In a simultaneous regression, both maternal BMI ($P = 0.006$) and BMI at age 11 y ($P < 0.001$) were associated with adult obesity.

Socioeconomic status

The incidence of obesity in adult life was 35.0% in children born into homes of laborers, 30.5% in children born into lower-middle-class homes and 26.4% in children in upper-middle-class homes (Table 7). Adult obesity was also commoner in people who had lower educational achievement: 37.2% in people with low educational achievement, 32.9% in those with moderate achievement and 25.1% in those with high achievement.

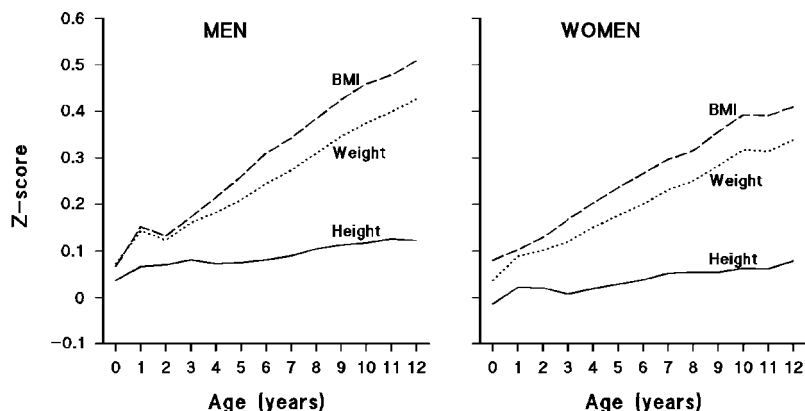


Figure 1 The growth of the men and women who later became obese is shown expressed as Z-scores. The Z-score for the whole cohort is set at zero and a child maintaining a steady position as large or small in relation to other children would follow a horizontal path. Children who later became obese, however, had accelerated gain in weight and BMI.

Table 4 Cumulative incidence of obesity according to age at adiposity rebound in men and women

Age at adiposity rebound (y)	Men	Women
-4	48.8%	46.7%
5	39.0%	37.0%
6	30.2%	30.3%
7	32.2%	25.3%
8-	32.3%	21.6%
P-values	10.007	<0.001

Table 5 Age-adjusted odds ratios for adult obesity grouped by ponderal index at birth and BMI at 11 y in men, women and sexes combined

	Ponderal index (kg/m ³)				
	<15.8	-16.6	-17.4	-18.5	>18.5
BMI at 11 y (kg/m²)					
Men					
<25	1.0	1.5	2.2	2.3	6.1
-26	0.9	1.3	2.7	3.5	8.5
-27	0.4	1.0	1.9	2.7	11.7
-28	0.7	1.5	1.9	2.8	4.1
>28	1.2	1.3	1.9	3.5	9.7
Women					
<25	1.0	1.3	1.8	1.1	5.2
-26	0.7	0.8	3.1	1.9	3.1
-27	1.0	1.9	1.5	1.3	3.8
-28	0.7	1.0	1.9	1.6	4.6
>28	1.4	1.2	2.3	2.0	5.0
All					
<25	1.0	1.4	1.9	1.7	5.7
-26	0.8	1.0	2.8	2.5	4.8
-27	0.7	1.5	1.6	1.9	4.8
-28	0.7	1.2	1.8	2.1	4.5
>28	1.3	1.2	2.1	2.6	6.4

Table 6 Cumulative incidence and 95% confidence intervals (in parentheses) of obesity according to maternal BMI in late pregnancy in men and women

Maternal BMI (kg/m ²)	Men	Women
-24	28.5 (23.6–33.3)	27.0 (22.6–31.3)
-25.5	31.1 (26.6–35.6)	30.7 (26.5–35.0)
-27	30.9 (26.5–35.4)	30.4 (26.1–34.6)
-28.5	36.5 (31.3–41.8)	34.8 (29.9–39.6)
>28.5	45.5 (40.2–50.7)	42.0 (37.1–46.9)
P for trend	<0.0001	<0.0001

Table 7 Cumulative incidence and 95% confidence intervals (in parentheses) of obesity according to childhood social class and educational achievements in adult life in men and women

	Men	Women
Social class in childhood		
Upper class	24.9 (19.9–29.9)	28.2 (22.5–33.8)
Lower middle	33.5 (29.0–38.0)	27.9 (23.9–31.9)
Laborer	35.5 (32.9–38.1)	34.6 (32.2–37.0)
P-values	0.001	0.005
Educational achievements		
Low	39.2 (35.6–42.9)	35.9 (33.0–38.8)
Middle	33.6 (30.4–36.7)	32.3 (29.1–35.5)
High	26.0 (22.1–29.8)	24.0 (19.9–28.1)
P-values	<0.001	<0.001

Breastfeeding

For 84% of the subjects, the method of infant feeding was recorded. A total of 99.6% of them were breastfed. The duration of breastfeeding was not associated with later obesity. The cumulative incidence of obesity among the 16% who were breastfed for less than 2 months was 33.8% and among the 26% who were breastfed for more than 8 months it was 35.9%.

Discussion

In a study of 4515 children, we have shown that the average BMI of the children who later became obese rose progressively in relation to other children from birth to 12 y (Figure 1). Few, if any, of the children growing up in Finland around the time of the Second World War were obese, by the standards of today. Our findings, therefore, cannot be interpreted as showing that childhood obesity leads to adult obesity. Rather, it is accelerated weight gain in childhood that seems to establish obesity in later life.

We used BMI above 30 kg/m² as a measure of obesity, as recommended by the WHO.⁸ Although BMI is widely used as the standard obesity assessment, it is only one indicator of obesity. For any given BMI, there is a large variation in percentage body fat and in body fat distribution. Part of the prediction of adult body mass from childhood body mass could result from persistence of muscularity rather than persistence of fatness. We have previously shown that large body size at birth is correlated with higher lean body mass in adult life.⁹ Although, a larger body size at birth is associated with a higher BMI in adult life the association has been suggested to be inverse between body size at birth and abdominal obesity.^{10,11} In the present study, we did not have any measure of abdominal obesity and could therefore not focus upon this issue.

Our study cohort consisted of men and women who were born at Helsinki University Central Hospital and who attended child welfare clinics and who were alive in the year 2000 and responded to our questionnaire. The men and women were therefore not representative of all men and women in the city. Our analysis, however, was based upon internal comparisons within the cohort, and the association between early growth and obesity seems unlikely to be because of bias. The present study was also restricted to children who had attended child welfare clinics. Attendance could be related to socioeconomic circumstances. Furthermore, the subjects in our cohort grew up during the Second World War; however, the war did not have a major impact on growth with only minor influences on body weight.

In the present study, BMI was based upon self-reported measures of height and weight. In our previous study, the correlation between BMI measured in the same subjects by questionnaire and in the clinic was 0.95.¹² The cumulative lifetime incidence of obesity in our study was similar among males and females, 33.8 and 32.4%, respectively. The finding is consistent with the known prevalence of obesity in many Western societies.^{13,14} Obviously, the use of maximal lifetime BMI instead of current BMI makes the overall prevalence of obesity higher.

Birth weight is a crude indicator of prenatal growth, and we found that ponderal index at birth predicted obesity more strongly. This association with body size at birth points to a role of the early environment in the development of adult obesity, consistent with previous studies.^{15–18} We also found that an early adiposity rebound was associated with obesity in adult life. People who had an adiposity rebound

after the age of 8 y had a 40% lower incidence of obesity in comparison with those who had a rebound before 5 y of age. This association has been described before.^{4,5} The mean age at adiposity rebound in our study was 5.8 y—similar to that in Western countries today. Interestingly, it has been shown that adiposity at both 1 y of age and at adult age is linked with age at adiposity rebound. It has been proposed that an early rebound is associated with a lower adiposity at 1 y of age and a higher adiposity at adult age; conversely, a late rebound corresponded to higher adiposity at 1 y and a lower adiposity at adult age. In other words, fatter infants with an early adiposity rebound have an increased risk of obesity in adult life while lean infants whose rebound is late are more likely to stay lean. One possibility is that an early adiposity rebound is associated with life-long setting of hormones such as insulin and insulin-like growth factors that facilitate the deposition of fat, while genetic factors are proposed to mediate the timing of the rebound.^{4,5}

There is considerable evidence that impaired fetal growth and rapid childhood weight gain is associated with an increased risk of type II diabetes and cardiovascular disease.^{6,12,19–24} However, although obesity is closely associated with these disorders, adult obesity seems to be associated with a large body size at birth rather than a small body size at birth. Obviously, this relation has received great attention. Increased muscularity, as nicely shown by Kahn and co-workers, may partly explain the cardiovascular benefits in later life associated with a higher birth weight.²⁵ We have previously shown that in an older Finnish birth cohort there was a positive correlation between body size at birth and lean body mass in adult life.⁹ Therefore, we propose that those individuals with a larger body size at birth have a bigger muscle mass potentially protecting them from metabolic disorders in adult life. We suggest that individuals with a high adult BMI and who have been small at birth would benefit more from weight loss metabolically than individuals who were born heavier, because of the above-mentioned facts.

We found an inverse association between social class in childhood and the incidence of obesity in adult life. This association between socioeconomic status and obesity has been shown before and is thought to result from people from lower social classes consuming more energy-dense and fat food.^{26–28} There was also an inverse association between educational attainment and obesity. Lower educational attainment was also associated with higher incidence of obesity. This association is probably because of similar factors in food choice and food consumption as described above. A large proportion of our study cohort came from lower social classes: 61% of the fathers were laborers. We know that at this time in Helsinki around 60% of the men were laborers. Therefore, the social class distribution of our sample may be similar to that of the city as a whole.

Recent studies have suggested that breastfeeding is linked to development of obesity in later life, that is, breastfeeding may help prevent childhood overweight.^{29,30} Our data do

not confirm this, but such an association might differ between populations and at different times.

Genetic factors are of major importance in the pathogenesis of obesity.^{31–34} The search for major obesity genes has not been successful and this is mostly because of the fact that the common form of obesity must be considered a polygenic disorder with important environmental components. We have no data on paternal body size but information on maternal body size in pregnancy. A high maternal BMI in pregnancy was associated with an increased risk of becoming obese among the offspring. The combination of a high maternal BMI and higher BMI in childhood increased the risk for obesity compared with either factor alone. Associations between mothers' weights and BMIs and those of their children have been found before.³ It is not known whether this indicates genetic influences or the effects of better nutrition during lactation or better availability of food after weaning. Given the social conditions of the time, few of the mothers would have been obese, as currently defined.

The present study shows that obesity in adult life is heralded at birth. Our findings suggest that one of the strategies to reduce obesity should be avoidance of accelerated weight gain in children—also among those whose BMI is within the normal range.

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