

Early life risk factors for obesity in childhood: cohort study

John J Reilly, Julie Armstrong, Ahmad R Dorosty, Pauline M Emmett, A Ness, I Rogers, Colin Steer, Andrea Sherriff for the Avon Longitudinal Study of Parents and Children Study Team

Abstract

Objective To identify risk factors in early life (up to 3 years of age) for obesity in children in the United Kingdom.

Design Prospective cohort study.

Setting Avon longitudinal study of parents and children, United Kingdom.

Participants 8234 children in cohort aged 7 years and a subsample of 909 children (children in focus) with data on additional early growth related risk factors for obesity.

Main outcome measures Obesity at age 7 years, defined as a body mass index \geq 95th centile relative to reference data for the UK population in 1990.

Results Eight of 25 putative risk factors were associated with a risk of obesity in the final models: parental obesity (both parents: adjusted odds ratio, 10.44, 95% confidence interval 5.11 to 21.32), very early (by 43 months) body mass index or adiposity rebound (15.00, 5.32 to 42.30), more than eight hours spent watching television per week at age 3 years (1.55, 1.13 to 2.12), catch-up growth (2.60, 1.09 to 6.16), standard deviation score for weight at age 8 months (3.13, 1.43 to 6.85) and 18 months (2.65, 1.25 to 5.59); weight gain in first year (1.06, 1.02 to 1.10 per 100 g increase); birth weight, per 100 g (1.05, 1.03 to 1.07); and short ($<$ 10.5 hours) sleep duration at age 3 years (1.45, 1.10 to 1.89).

Conclusion Eight factors in early life are associated with an increased risk of obesity in childhood.

Introduction

An epidemic of childhood obesity has occurred in recent years, beginning in the late 1980s in the United Kingdom.¹ It is of concern because of adverse consequences in the short term and long term.^{1,2} The identification of risk factors is the key to prevention.³ Evidence on risk factors for childhood obesity is limited at present,^{4,5} although awareness is increasing for the importance of the environment in early life. Almost all recognised risk factors are potential rather than confirmed. A systematic review found that most previous studies on risk factors for obesity were unable to adequately account for confounding variables (particularly socioeconomic status),⁵ were mainly cross sectional, were underpowered,^{4,5} and failed to investigate the effect of several potential risk factors simultaneously.^{4,5}

We identified and quantified risk factors for obesity at age 7 years in children who were participating in the Avon longitudinal study of parents and children (ALSPAC). The study concerns a large contemporary cohort in which confounding variables are being considered and potential risk factors are being tested simultaneously. For the present study, we took into account only risk factors supported by a priori hypotheses.

Methods

The Avon longitudinal study of parents and children is a longitudinal birth cohort study of the determinants of development, health, and disease during childhood and beyond. This study is described in detail elsewhere.⁶ Briefly, 14 541 pregnant women with an expected date of delivery between April 1991 and December 1992 were enrolled; 13 971 of their children formed the original cohort. About 85-90% of eligible mothers took part in the study. Parents gave informed written consent for their children to participate. Data have been collected from questionnaires completed by the parents, medical records, and biological samples. We randomly selected a subsample of children from the last six months of recruitment (children in focus group), aged from 4 months to 5 years, and invited their parents to bring them in for regular physical examinations. From age 7 years onwards these examinations were extended to the whole cohort.

Anthropometric variables

We measured height to 0.1 cm using the Harpenden stadiometer (Holtain; Crymych, Wales). Weight was measured to 0.1 kg. From these values we calculated the body mass index (weight (kg)/(height (m)²). Body mass indices were converted to standard deviation scores relative to UK reference data in 1990.⁷

Definition of obesity

We defined obesity as a body mass index equal to or greater than the 95th centile, equivalent to a standard deviation score of 1.64 or more. This definition has high specificity and moderate sensitivity for identifying the children with highest body fat percentage within the British population.^{8,9} Obesity defined in this way is also biologically meaningful as it identifies those children who are most likely to experience comorbidity, such as persistence of obesity, presence and clustering of cardiovascular risk factors, and psychological problems.²

Potential risk factors

We chose putative risk factors on the basis of previously reported associations with obesity, or plausible prior hypotheses. We cross checked the results of our literature search against a systematic review.⁵ Overall, we identified 31 potential risk factors. Measures for 21 of these risk factors were available for the entire cohort of the Avon longitudinal study of parents and children. A further four potential risk factors relating to growth in infancy and early childhood were available for the children in focus subsample (table 1).

Statistical analyses

We carried out a multivariable analysis for the prevalence of obesity in three stages using multivariable binary logistic

Table 1 Potential risk factors and confounders, method and time of data collection, and factor level of analysis

Variables	Method and time of data collection	Factor level of analysis
Potential confounders:		
Maternal social class (SES)	Self report (derived from maternal occupation) at 32 weeks' gestation	I (highest), II, IINM, IIINM, IV, V (lowest)
Maternal education	Self report at 32 weeks' gestation	CSE or no qualification (lowest), vocational, O level, A level, degree (highest)
Energy intake of child	Food frequency questionnaire at age 30 months	Megajoules per day, continuous
Intrauterine and perinatal factors:		
Birth weight	Measured in delivery room	Continuous (unit 100 g)
Sex	Medical records	Male or female
Maternal parity	Self report questionnaire at 18 weeks' gestation	Number of previous pregnancies resulting in live birth or stillbirth: 0, 1, ≥ 2
Maternal smoking during pregnancy (28-32 weeks' gestation)	Self report questionnaire at 32 weeks' gestation	Daily: none, 1-9, 10-19, ≥ 20
Season of birth	Date of birth on medical records	Jan-Mar, Apr-Jun, Jul-Sept, Oct-Dec
Gestational Age	Derived from last menstrual period	Weeks: <37, 37-42, >42
No of fetuses	Medical records	1, 2 (twins)
Infant feeding:		
Breast feeding	Child feeding questionnaire at six months post partum	Exclusive breast feeding at two months; stopped or non-exclusive breast feeding; never breast fed
Age at introduction of complementary feeding	Child feeding questionnaire at six months post partum	Time (months): <1, 1-2, 2-3, 3-4, 4-6, not yet introduced
Family characteristics:		
Parents body mass index pre-pregnancy	Maternal self report questionnaire during pregnancy	Body mass indices: both parents <30; father only, >30; mother only, >30; both parents >30
Number of siblings of child at 18 months	Child based maternal self report questionnaire at 18 months post partum	Number of siblings younger and older: 0, 1, 2, ≥ 3
Ethnicity of child	Maternal self report questionnaire at 32 weeks' gestation	White, non-white
Age of mother at delivery	Enrolment cards, clinical records, notification of delivery	Years: <20, 20-24, ≥ 25
Childhood lifestyle:		
Time spent watching television per week (at 38 months)	Questionnaire	Hours: ≤ 4 , 4.1-8, >8
Time in car on weekdays per day (at 38 months)	Questionnaire	Not at all, <1 hour, ≥ 1 hour
Time in car on weekend days per day (at 38 months)	Questionnaire	Not at all; <1 hour; ≥ 1 hour
Duration of night time sleep (at 38 months)	Questionnaire	Quartiles of sleep duration (hours): <10.5, 10.5-11.4, 11.5-11.9, ≥ 12)
Dietary patterns	Food frequency questionnaire at 38 months	Factor loadings for food categories from principal components analysis scores in quartiles: junk, healthy, traditional, fussy or snack*
Risk factors in children in focus subsample:		
Weight standard deviation score at eight months	Measurement as part of children in focus study	In quartiles at eight months
Weight standard deviation score at 18 months	Measurement as part of children in focus study	In quartiles at 18 months
Rapid catch up growth 0-2 years	Weight gain of >0.67 standard deviation scores in first two years	Catch down, no change, catch up
Adiposity rebound	Change in body mass index up to 60 months	Very early (by 43 months), early (by 61 months), later (after 61 months)
Weight gain in first 12 months	Calculated from variable weight at 12 months minus birth weight	Continuous: weight gain between birth and 12 months (units 100 g)

*Definitions taken from North and Emmett.¹⁰ Factor loading score (highest first): food group 1, Junk type—fizzy drinks, sweets and confectionery, chocolate, chips (fries), fried foods, sausages, burgers, crisps, takeaway meals, pot noodles, cook in sauces, bread, pizza, biscuits, and flavoured milks; food group 2, healthy type—pulses, vegetarian foods, rice, pasta, salad, fruit juice, fruit, water, eggs, cheese, and fish; food group 3, traditional type—meat and vegetables as meals, meat, poultry, root vegetables, potatoes (not fries), green vegetables, and legumes (peas, sweetcorn); food group 4, fussy or snack type—puddings, cakes and buns, biscuits, squash, crisps, cheese, and fruit.

regression models. Firstly, owing to the strong association between maternal education (as a proxy for socioeconomic status) and childhood obesity, we assessed whether the effect of potential risk factors was confounded by the mother's education. Secondly, in an effort to reduce collinearity and to minimise the likelihood of producing misleading estimates for the variables (data not shown), we analysed putative risk factors for childhood obesity (the effects of which were found to be independent of maternal education) simultaneously within each of the four risk factor groups (intrauterine and perinatal factors; infant feeding and complementary feeding (weaning) practice; family characteristics and demography; and lifestyle in early childhood). Finally, risk factors that were independently significant ($P < 0.10$) at the within group stage were then entered into a final model in which we analysed all variables simultaneously. In the final model we further adjusted the variables for sex (we had a priori reasons for believing that sex might mediate some

of the effects of some risk factors, although in practice its inclusion or exclusion made little difference to the odds ratios in the final model); maternal education (to control or adjust for socioeconomic status); and, for the food group variables,¹⁰ the child's estimated energy intake at age 3 years. We used χ^2 tests for linear trend for ordered categorical variables and Fisher's exact test in contingency tables when the expected frequency in any cell was less than 5.

To assess the effect of the four growth related risk factors (measured in the children in focus subsample only) on obesity, we used multivariable binary logistic regression models, while controlling for all other statistically significant risk factors obtained from the analysis of the whole cohort. Owing to the correlation between these growth related risk factors, we independently assessed their effect on obesity. We produced five separate models for the four risk factors. One risk factor, size in early life, was measured at age 8 and 18 months.

Results

In total, 8234 children attended the clinic at age 7. Measures for height and weight were available for 7758 children (3934 boys and 3824 girls; 55.5% of the original 13 971 children) median age 7.6 years (range 6.9–8.5 years). The prevalence of obesity did not differ significantly between the sexes (9.2% for boys (n = 362) and 8.1% for girls (n = 309; P = 0.08)). Overall, 5493 children (70.8% of those with measures for height and weight who attended at age 7, 39.3% of the original cohort) had complete data for the multivariable analyses.

Risk factors in entire cohort

Intrauterine and perinatal factors

Increasing birth weight was independently and linearly associated with increasing prevalence of obesity at age 7 (table 2). Obesity at age 7 was also significantly associated with maternal smoking between 28 and 32 weeks' gestation, with some indication of a dose response (χ^2 test for linear trend 27.17).

Infant feeding and weaning practice

The apparent protective effect of exclusive breastfeeding on obesity at age 7 observed in the univariable analysis remained when breastfeeding was considered together with the other infant feeding and weaning practice variable (adjusted odds ratio 0.70, 95% confidence interval 0.54 to 0.91), but had disappeared in the final model (table 2). In the final model, timing of introduction of complementary feeding was not significantly related to the risk of obesity at age 7.

Family characteristics and demographics

When only one parent was obese, the risk of obesity at age 7 was increased. The risk was higher when both parents were obese (adjusted odds ratio 10.44, 5.11 to 21.32; table 2).

Lifestyle in early childhood

Sleep Sleep duration in children aged 30 months was independently associated with prevalence of obesity at age 7 (table 2). Children in the lowest two quarters of sleep duration (< 10.5 hours and 10.5–10.9 hours) were more likely to be obese at age 7 than children in the highest quarter (> 12 hours; χ^2 test for linear trend 17.8).

Sedentary behaviour The odds ratio for obesity increased linearly as the number of hours of television viewing increased (χ^2 test for linear trend 26.7). For children reported to watch television for 4–8 hours per week at age 3 the adjusted odds ratio for obesity at age 7 was 1.37 (1.02 to 1.83). For those reported to watch more than eight hours per week the adjusted odds ratio was 1.55 (1.13 to 2.12).

Dietary patterns We found no conclusive evidence of an association between dietary patterns at age 3 and risk of obesity at age 7. A junk food type dietary pattern at age 3 was significantly associated with risk of obesity at age 7, although the association only just reached significance at the 10% level in the final model (table 2).

Risk factors in children in focus subsample

The prevalence of obesity at 7 years in the children in focus subsample was not significantly different from that in the entire cohort (8.7%; 79/909). Children in the highest quarter for weight (standard deviation scores) at age 8 months and 18 months were more likely to be obese at age 7 than children in the lower quarters (table 3). Early adiposity or body mass index rebound, catch-up growth between birth and two years, and high rates of

weight gain in the first 12 months were also independently associated with obesity at age 7 (table 3).

Discussion

We found that eight of 25 putative early life risk factors for obesity in childhood were significantly related to risk of obesity. Our study supports the hypothesis that the environment in early life can determine risk of later obesity, and suggests several influences in early life that might be suitable targets for future obesity prevention interventions. Our study has advantages over previous similar ones because of its contemporary nature, large sample size, longitudinal design, and the use of multivariable analysis. The most critical observers of causes for obesity^{4 11} believe that evidence suggests a causal role for sedentary behaviour, consumption of sugar sweetened drinks, and, possibly, formula feeding during infancy. Risk factors for obesity in childhood were, however, uncertain, and evidence based targets for preventive strategies were lacking.^{4 5} We found evidence that the list of potential risk factors for childhood obesity and targets for preventive interventions should be extended.

Potential risk factors not supported by present study

Many putative risk factors for obesity in our study were not independently associated with the risk of obesity in childhood: sex, parity, season of birth, gestational age, number of fetuses, timing of introduction of complementary feeding, number of siblings, ethnicity, maternal age, and time spent in the car.

Inconclusive potential risk factors

We did not observe an independent protective effect of exclusive breast feeding on obesity in our final model, despite strong univariable associations, and in contrast to our previous studies.¹² The change in direction and magnitude of the odds ratio between univariable and multivariable models in the present study, combined with recent evidence of an interaction between breast feeding and maternal smoking during pregnancy,¹³ led us to test for an interaction of this type. Breast feeding in women who did not smoke during pregnancy (but not in women who smoked during pregnancy) was significantly associated with a reduced risk of obesity at age 7 years. Evidence from both human studies and animal models show that maternal smoking during pregnancy may increase the risk of obesity later in life, and that this may operate by programming the regulation of appetite.^{14 15}

Our measures of dietary patterns were generally not significantly associated with the risk of obesity in the final model, but associations of this kind may be worthy of further investigation.

Potential risk factors supported by present study

In the entire cohort, birth weight, parental obesity, sleep duration, and television viewing remained independently associated with the risk of obesity in the final model. A further four factors were significant in the children in focus subsample: size in early life (standard deviation scores for weight at age 8 months and 18 months), weight gain in infancy, catch-up growth, and early adiposity or body mass index rebound (before 43 months¹⁶).

Parental obesity may increase the risk of obesity through genetic mechanisms or by shared familial characteristics in the environment such as food preferences.¹⁷ Duration of night time sleep may alter later risk of obesity through growth hormone secretion, or because sleep reduces the child's exposure to factors in the environment that promote obesity, such as food intake in the evening. Alternatively, duration of night time sleep may be a marker for some other variable such as level of physical

Table 2 Associations between 21 risk factors and obesity at age 7 years in cohort of Avon longitudinal study of parents and children. Values are percentages (numbers) unless stated otherwise

Risk factors	Prevalence of childhood obesity (n=7758)	Unadjusted (univariable) odds ratio (95% CI)	P value	Final model adjusted odds ratio (95% CI)† (n=5493)	P value
Intrauterine and perinatal factors					
Birth weight, continuous* (100 g units)	8.4 (605/7224)	1.05 (1.03 to 1.07)	<0.001	1.05 (1.03 to 1.07)	<0.001
Missing data	12.3 (66/534)	—	—	—	—
Sex*					
Male	9.2 (362/3934)	1.00	0.079	1.00	0.890
Female	8.1 (309/3824)	0.87 (0.74 to 1.02)		0.99 (0.81 to 1.21)	
Missing data	—	—	—	—	—
Parity*					
0	8.3 (265/3205)	1.00	0.854	Did not enter	—
1	8.4 (215/2557)	1.02 (0.84 to 1.23)			
≥2	7.9 (103/1308)	0.95 (0.75 to 1.20)			
Missing data	12.8 (88/688)	—	—	—	—
Maternal smoking during pregnancy per day*					
None	7.5 (443/5889)	1.00	<0.001	1.00	<0.010
1-9	11.5 (50/434)	1.60 (1.17 to 2.18)		1.76 (1.21 to 2.52)	
10-19	11.3 (48/423)	1.57 (1.15 to 2.16)		1.59 (1.08 to 2.34)	
≥20	13.8 (19/138)	1.96 (1.20 to 3.22)		1.80 (1.01 to 3.39)	
Missing data	12.7 (111/874)	—	—	—	—
Season of birth*					
Jan-Mar	8.7 (106/1219)	1.00	0.884	Did not enter	—
Apr-Jun	8.0 (167/2080)	0.92 (0.71 to 1.18)			
Jul-Sep	8.2 (160/1949)	0.94 (0.73 to 1.21)			
Oct-Dec	8.6 (177/2065)	0.98 (0.77 to 1.27)			
Missing data	13.7 (61/445)	—	—	—	—
Gestational age (weeks)					
<37	8.4 (31/412)	1.00	0.436	Did not enter	—
37-42	8.4 (574/6864)	1.12 (0.87 to 1.45)			
>42	13.5 (5/37)	1.75 (0.68 to 4.50)			
Missing data	13.7 (61/445)	—	—	—	—
No of fetuses					
1	8.7 (655/7551)	1.00	0.635	Did not enter	—
2	7.7 (16/207)	0.88 (0.53 to 1.48)			
Infant feeding					
Breast feeding*:					
Exclusive at 2 months	6.9 (152/2211)	0.64 (0.50 to 0.82)	0.002	1.22 (0.87 to 1.71)	0.464
Stopped or non-exclusive	8.5 (281/3287)	0.81 (0.64 to 1.01)		1.08 (0.80 to 1.45)	
Never	10.4 (120/1153)	1.00		1.00	
Missing data	10.7 (118/1107)	—	—	—	—
Introduction of solids (months)*:					
<1	9.6 (17/177)	1.44 (0.85 to 2.45)	0.003	0.88 (0.42 to 1.87)	0.296
1 or 2	10.6 (12/113)	1.61 (0.83 to 3.01)		1.08 (0.50 to 2.32)	
2 or 3	11.6 (77/664)	1.78 (1.33 to 2.39)		1.48 (1.01 to 2.16)	
3 or 4	8.3 (320/3868)	1.22 (0.99 to 1.51)		1.08 (0.83 to 1.39)	
4-6	6.9 (134/1953)	1.00		1.00	
Missing data	11.3 (111/983)	—	—	—	—
Family characteristics					
Parental obesity (body mass index)*:					
Both parents (<30)	6.2 (264/4253)	1.00	<0.001	1.00	<0.001
Father (>30)	16.2 (49/302)	2.93 (2.10 to 4.07)		2.54 (1.72 to 3.75)	
Mother (>30)	23.6 (46/195)	4.66 (3.28 to 6.64)		4.25 (2.86 to 6.32)	
Both parents obese	43.8 (21/48)	11.75 (6.55 to 21.06)		10.44 (5.11 to 21.32)	
Missing data	9.8 (291/2960)	1.65 (1.38 to 1.96)		1.33 (1.06 to 1.67)	
No of siblings*:					
None	8.8 (251/2858)	1.31 (0.81 to 2.13)	0.304	Did not enter	—
1	7.5 (196/2604)	1.11 (0.69 to 1.81)			
2	8.5 (80/944)	1.26 (0.75 to 2.12)			
≥3 (I)	6.8 (19/278)	1.00			
Missing data	11.6 (125/1074)	—	—	—	—
Ethnicity of child:					
White	8.0 (535/6647)	1.00	0.156	Did not enter	—
Non-white	10.5 (29/277)	1.34 (0.90 to 1.98)			
Missing data	12.8 (107/834)	—	—	—	—

Risk factors	Prevalence of childhood obesity (n=7758)	Unadjusted (univariable) odds ratio (95% CI)	P value	Final model adjusted odds ratio (95% CI)† (n=5493)	P value
Age of mother at delivery:					
<20	7.4 (10/135)	0.91 (0.47 to 1.73)	0.204	Did not enter	—
20-24	9.8 (98/998)	1.23 (0.98 to 1.55)			
≥25	8.1 (502/6180)	1.00			
Missing data	13.7 (61/445)	—	—	—	—
Lifestyle factors in childhood					
Time spent watching television per week (hours)*:					
≤4	5.2 (75/1450)	1.00	<0.001	1.00	<0.010
4.1-8	8.3 (252/3026)	1.66 (1.28 to 2.17)		1.37 (1.02 to 1.83)	
>8	10.3 (201/1950)	2.10 (1.60 to 2.77)		1.55 (1.13 to 2.12)	
Missing data	10.7 (143/1332)	—	—	—	—
Time in car per day					
Weekdays (hours):					
None	6.9 (37/533)	1.00	0.34	Did not enter	—
<1	8.1 (426/5227)	1.19 (0.84 to 1.69)			
≥1	9.2 (67/725)	1.37 (0.90 to 2.07)			
Missing data	11.1 (141/1273)	—	—	—	—
Weekend (hours):					
None	12.3 (26/211)	1.00	0.090	Did not enter	—
<1	8.0 (346/4332)	0.62 (0.40 to 0.95)			
≥1	8.2 (157/1907)	0.64 (0.41 to 0.99)			
Missing data	10.9 (142/1308)	—	—	—	—
Duration of night time sleep (hours)*					
First quarter (<10.5)	10.3 (171/1660)	1.57 (1.23 to 1.99)	<0.001	1.45 (1.10 to 1.89)	<0.010
Second quarter (10.5-10.9)	8.8 (135/1539)	1.31 (1.02 to 1.69)		1.35 (1.02 to 1.79)	
Third quarter (11-11.9)	6.4 (87/1351)	0.94 (0.71 to 1.25)		1.04 (0.76 to 1.42)	
Fourth quarter (>12)	6.8 (128/1876)	1.00		1.00	
Missing data	11.3 (150/1322)	—	—	—	—
Dietary patterns					
Food group 1 (junk)*:					
First quarter	5.5 (89/1607)	1.00	<0.001	1.00	0.083
Second quarter	7.9 (127/1606)	1.46 (1.11 to 1.94)		1.43 (1.00 to 1.85)	
Third quarter	10.1 (162/1607)	1.91 (1.46 to 2.50)		1.48 (1.08 to 2.02)	
Fourth quarter	9.2 (148/1607)	1.73 (1.32 to 2.27)		1.24 (0.87 to 1.76)	
Missing data	10.9 (145/1331)	—	—	—	—
Food group 2 (healthy)*:					
First quarter	9.2 (148/1607)	1.00	0.061	1.00	0.769
Second quarter	8.5 (136/1609)	0.91 (0.71 to 1.16)		1.01 (0.76 to 1.34)	
Third quarter	8.4 (135/1607)	0.90 (0.71 to 1.15)		1.02 (0.75 to 1.37)	
Fourth quarter	6.7 (107/1605)	0.70 (0.54 to 0.91)		0.87 (0.61 to 1.22)	
Missing data	10.9 (145/1331)	—	—	—	—
Food group 3 (traditional)*:					
First quarter	7.6 (122/1608)	1.00	0.088	1.00	0.386
Second quarter	8.3 (133/1612)	1.10 (0.85 to 1.42)		1.13 (0.84 to 1.53)	
Third quarter	7.3 (117/1607)	0.96 (0.74 to 1.25)		1.01 (0.74 to 1.38)	
Fourth quarter	9.6 (154/1601)	1.30 (1.01 to 1.66)		1.26 (0.92 to 1.72)	
Missing data	10.9 (145/1331)	—	—	—	—
Food group 4 (fussy)*:					
First quarter	9.9 (159/1606)	1.00	0.005	1.00	0.635
Second quarter	8.7 (139/1606)	0.86 (0.68 to 1.10)		0.95 (0.71 to 1.27)	
Third quarter	7.6 (122/1609)	0.75 (0.58 to 0.96)		0.92 (0.67 to 1.27)	
Fourth quarter	6.6 (106/1607)	0.64 (0.50 to 0.83)		0.79 (0.55 to 1.14)	
Missing data	10.9 (145/1331)	—	—	—	—

*Effect of variable on obesity at age 7 years is independent of maternal education.

†Odds ratio (95% CI) also adjusted for maternal education, energy intake at age 3 years (food groups), and sex. Variables significant (P<0.01) at within group analysis entered into final model (data from within group analyses not shown).

activity—that is, children who are more physically active may sleep longer at night. Television viewing may confer risk through a reduction in energy expenditure because watching television is associated with dietary intake, or because large amounts of time spent sedentary may contribute to impairment of the regulation of energy balance by uncoupling food intake from energy expenditure.^{18 19}

The precise mechanisms by which the early life growth variables studied in the children in focus subsample might increase the risk of obesity are generally unclear. They are, however, consistent with the increasing body of evidence that the early life environment is an important determinant of risk of obesity in later life.^{3 4 12 20 21}

Table 3 Associations between four risk factors and obesity at age 7 years in children in focus subsample. Values are numbers (percentages) unless stated otherwise

Potential risk factor	Prevalence of childhood obesity (n=909)	Unadjusted (univariate) odds ratio (95% CI)	P value	Final model adjusted odds ratio (95% CI)*	P value
Standard deviation score for weight					
Age 8 months†:					
All others	6.1 (42/685)	1.00	<0.001	1.00	0.004
Highest quarter	16.5 (37/224)	3.03 (1.89 to 4.85)		3.13 (1.43 to 6.85)	
Age 18 months†:					
All others	5.9 (37/625)	1.00	<0.001	1.00	0.011
Highest quarter	18.9 (39/206)	3.71 (2.29 to 6.00)		2.65 (1.25 to 5.59)	
Missing data	3.8 (3/78)	—	—	—	—
Catch-up growth†					
Catch down	3.8 (8/212)	0.46 (0.2 to 1.03)	<0.001	0.18 (0.04 to 0.75)	0.002
No change	7.5 (27/359)	1.00		1.00	
Catch-up	15.8 (33/209)	2.21 (1.30 to 3.8)		2.60 (1.09 to 6.16)	
Missing data	8.5 (11/129)	—	—	—	—
Adiposity rebound†					
Later (after 61 months)	4.6 (25/547)	1.00	<0.001	1.00	<0.001
Early (by 61 months)	12.0 (19/158)	2.85 (1.53 to 5.33)		2.01 (0.81 to 5.20)	
Very early (by 43 months)	36.5 (19/52)	12.02 (6.01 to 24.03)		15.00 (5.32 to 42.30)	
Missing data	10.5 (16/152)	—	—	—	—
Weight gain in first 12 months† (per 100 g increase)					
Data present	8.6 (74/857)	1.07 (1.05 to 1.10)	<0.001	1.06 (1.02 to 1.10)	0.003
Missing data	9.6 (5/52)	—	—	—	—

*Effect of variable on obesity is independent of maternal educational attainment.

†Each variable was offered separately to a model (five models in total) containing birth weight, maternal smoking, parental obesity, hours of sleep at age 30 months, time spent watching television at age 30 months, food groups 1–4, maternal education, sex, and daily energy intake (MJ). Total number in multivariable models: 533, 511, 487, 486, and 522.

Limitations of the study

Cohort studies are inherently limited to identifying associations rather than confirming causality. We were unable to analyse several potential risk factors—notably physical activity and energy expenditure, parental control over feeding in childhood,²² and maternal diabetes during pregnancy.²³ The use of definitions of obesity based on body mass index is acceptable as an outcome measure, but no definition of obesity is ideal at present.^{7–9}

The Avon longitudinal study of parents and children cohort is broadly representative of the UK population,⁶ although ethnic minority groups are slightly under-represented. We cannot rule out the possibility that we underestimated the effect of some risk factors that are more prevalent in these groups. When we considered multivariable effects in our models, the sample size was reduced. The degree of attenuation of effect sizes towards the final models was, however, generally small, and univariable

odds ratios were usually similar to the multivariable odds ratios, with the exception of breast feeding.

Implications

Intrauterine life, infancy, and the preschool period (around the time of the adiposity or body mass index rebound) have all been considered as possible critical periods during which the long term regulation of energy balance may be programmed.²⁴ Our study provides evidence of the role of the early life environment in the later risk of obesity. Prevention strategies for childhood obesity to date have usually been unsuccessful and typically focus on change in lifestyle during childhood or adolescence. Future interventions might focus on environmental changes targeted at relatively short periods in early life, attempting to modify factors in utero, in infancy, or in early childhood, which are independently related to later risk of obesity.

We thank the participants of the Avon longitudinal study of parents and children. The study team comprises interviewers, computer technicians, laboratory technicians, clerical workers, research scientists, volunteers, and managers who continue to make the study possible. The Avon longitudinal study of parents and children is part of the WHO initiated European longitudinal study of pregnancy and childhood.

Contributors: JJR, AS, JA, and PME obtained funding. JJR, ARD, AS, JA, and AN were responsible for the concept and design of the study. PME, IR, and CS collected the data. JJR, AS, and JA drafted the manuscript. AS, CS, and AN provided statistical expertise. All the authors were responsible for the analysis and interpretation of data and for critical revision of the manuscript. The funding bodies had no role in the decision to publish or the content of this article.

Funding: This secondary analysis was funded by the Scottish Executive Health Department. The Avon longitudinal study of parents and children is funded by the Medical Research Council, Wellcome Trust, and various UK government departments, the US National Institutes of Health, a variety of medical research charities and commercial companies. ARD was funded by the Iranian Ministry of Health and Medical Education.

Competing interests: None declared.

Ethical approval: Law and ethics committee of the Avon longitudinal study of parents and children and the local research ethics committees.

1 Reilly JJ, Dorosty AR. Epidemic of obesity in UK children. *Lancet* 1999;354:1874–5.

What is already known on this topic

Obesity is common in children and adolescents and its prevalence is still increasing

Risk factors for childhood obesity are not well established

Existing prevention strategies, focused on late childhood and adolescence, are largely unsuccessful

What this study adds

The early life environment can determine later risk of obesity

Eight factors in early life were independently associated with obesity risk at age 7

Eight evidence based targets for future population based obesity prevention interventions have been identified

- 2 Reilly JJ, Methven E, McDowell ZC, Hacking B, Alexander D, Stewart L, et al. Health consequences of obesity. *Arch Dis Child* 2003;88:748-52.
 - 3 Dietz WH. Birth weight, socioeconomic class, and adult adiposity among African Americans. *Am J Clin Nutr* 2000;72:335-6.
 - 4 Dietz WH. Breastfeeding may help prevent childhood overweight. *JAMA* 2001;285:2506-7.
 - 5 Parsons TJ, Power C, Summerbell CD. Childhood predictors of adult obesity: systematic review. *Int J Obes* 1999;23 (suppl 8):S1-107.
 - 6 Golding J, Pembrey M, Jones R, ALSPAC Study Team. ALSPAC—the Avon longitudinal study of parents and children I: study methodology. *Paediatr Perinat Epidemiol* 2001;15:74-87.
 - 7 Cole TJ, Freeman JV, Preece MA. Body mass index reference curves for the UK, 1990. *Arch Dis Child* 1995;73:25-9.
 - 8 Reilly J, Dorosty AR, Emmett PM, ALSPAC Study Team. Identification of the obese child: adequacy of the BMI for clinical practice and epidemiology. *Int J Obes* 2000;24:1623-7.
 - 9 Reilly JJ, Wilson ML, Summerbell CD, Wilson AC. Obesity: diagnosis, prevention, and treatment; evidence based answers to common questions. *Arch Dis Child* 2002;86:392-4.
 - 10 North K, Emmett P, ALSPAC Study Team. Multivariate analysis of diet among three-year-old children and associations with socio-demographic characteristics. *Eur J Clin Nutr* 2001;54:73-80.
 - 11 Whitaker RC. Obesity prevention in primary care: four behaviors to target. *Arch Pediatr Adolesc Med* 2003;157:725-7.
 - 12 Armstrong J, Reilly JJ. Child Health Information Team. Breastfeeding and lowering the risk of childhood obesity. *Lancet* 2002;359:2003-4.
 - 13 Bogen DL, Hanusa BH, Whitaker RC. The effect of breastfeeding with and without concurrent formula feeding on risk of obesity at 4 years of age. *Obes Res* 2004;12:1527-35.
 - 14 Von Kries R, Toschke AM, Koletzko B, Slikker W Jr. Maternal smoking during pregnancy and childhood obesity. *Am J Epidemiol* 2002;156:954-61.
 - 15 Grove KL, Sekhon HS, Brogan RS, Keller JA, Smith MS, Spindel ER. Chronic maternal nicotine exposure alters neuronal systems in the arcuate nucleus that regulate feeding behavior in the newborn rhesus macaque. *J Clin Endocrinol Metab* 2001;86:5420-6.
 - 16 Dorosty AR, Emmett PM, Reilly JJ, ALSPAC Study Team. Factors associated with early adiposity rebound. *Pediatrics* 2000;105:1115-8.
 - 17 Francis LA, Lee Y, Birch LL. Parental weight status and girls television viewing, snacking, and body mass indexes. *Obes Res* 2003;11:143-51.
 - 18 Gortmaker SL, Must A, Sobol AM, Petgerson K, Colditz GA, Dietz WH. Television viewing as a cause of increasing obesity among children in the United States 1986-1990. *Arch Pediatr Adolesc Med* 1996;150:356-62.
 - 19 Robinson TN. Reducing children's television viewing to prevent obesity: randomized controlled trial. *JAMA* 1999;282:1561-7.
 - 20 Stettler N, Zemel BS, Kumanyika S, Stallings VA. Infant weight gain and childhood overweight status in a multicenter cohort study. *Pediatrics* 2002;109:194-9.
 - 21 Ong KK, Ahmed ML, Emmett PM, Preece MA, Dunger DB. Association between post-natal catch-up growth and obesity in childhood: prospective cohort study. *BMJ* 2000;320:967-71.
 - 22 Birch LL, Fisher JO. Development of eating behaviors among children and adolescents. *Pediatrics* 1998;101:539-49.
 - 23 Dabelea D, Hanson RL, Lindsay RS, Pettitt DJ, Imperatore G, Gabir MM, et al. Intra-uterine exposure to diabetes conveys risks for type 2 diabetes and obesity: a study of discordant sibships. *Diabetes* 2000;49:2208-11.
 - 24 Dietz WH. Periods of risk in childhood for the development of adult obesity—what do we need to learn? *J Nutr* 1997;127(suppl 4):S1884-6.
(Accepted 19 April 2005)
- doi 10.1136/bmj.38470.670903.E0

University of Glasgow Division of Developmental Medicine, Yorkhill Hospitals, Glasgow G3 8SJ

John J Reilly *reader in paediatric energy metabolism*

Julie Armstrong *senior lecturer in nutrition*

Unit of Paediatric and Perinatal Epidemiology, Institute of Child Health, University of Bristol

Pauline M Emmett *senior research fellow in nutrition*

A Ness *senior lecturer in epidemiology*

I Rogers *research fellow in nutrition*

Colin Steer *research fellow in statistics*

Andrea Sherriff *research fellow in medical statistics*

School of Public Health, Tehran University of Medical Sciences, Islamic Republic of Iran

Ahmad R Dorosty *assistant professor*

Correspondence to: J J Reilly jjr2y@clinmed.gla.ac.uk