PAPERS AND ORIGINALS

Nature, nurture, and childhood overweight

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Summary and conclusions

The relative importance of dietary and familial factors in determining weight in early infancy were studied in 203 5-year-old children. Their age at weaning, energy intake in infancy and at 5 years, and maternal percentage expected weight were studied in relation to their percentage expected weight. Neither the estimated energy intake in infancy nor the intake at 5 years correlated significantly with their percentage expected weight at 5 years. Overweight 5-year-olds had not been weaned earlier than normal-weight 5-year-olds. There was a significant correlation between the percentage expected weights of the mothers and those of their children at 5 years of age, although the children of overweight mothers did not have higher energy intakes than the children of underweight mothers.

A familial, perhaps genetically determined, tendency to overweight seems to be more important in determining whether a child will be overweight at 5 years old than early weaning and overfeeding in infancy.

Introduction

In a previous study[1] we showed that most well-nourished and overweight infants did not become overweight children. Furthermore, some normal-weight children became overweight or obese in early childhood. The high energy intake that may result from bottle feeding and early weaning has been suggested as a cause of rapid weight gain in British infants[2] and even of overweight in young children.[3] Yet familial trends in weight also occur.[4] We used data collected in our earlier study to compare and contrast the relative importance of some dietary and familial factors in determining body size in early childhood.

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Patients and methods

The children studied are described in the earlier paper.[1] All had been seen in 1969-70 in a study of feeding practices and weight in infancy.[5] Two hundred and three (100 boys and 103 girls) of these infants were then reviewed between the ages of 4·3 and 6·4 years. Details of height, weight, and 24-hour dietary intake for each child on the day of infancy survey were available. The review at about 5 years included measurement of the heights and weights of the children and estimations of 24-hour food intake. The children's mothers were also weighed and measured.

ANTHROPOMETRY

Weight and height of children and mothers—All weights were recorded to 100 g on the same set of bathroom-type Salter scales checked with standard weights before each use. Mothers were weighed without shoes and outdoor clothing. Children were weighed in vest and pants without shoes. Heights were recorded to 1 mm on a Holtain portable stadiometer with the head supported so that the external auditory meatus and external canthus of the eye were in the same plane.

Interpretation of anthropometric data—The percentage expected weight for age of the children was assessed as (actual weight of the child/50th centile weight at that age when the child's height was on the 50th centile of reference standards[6]) × 100. For mothers, percentage expected weight was (actual weight/mean weight for height and age of a British reference population[7]) × 100. Both children and mothers were classed as underweight if their expected weight was less than 90%, normal weight if it was 90-110%, and overweight if it was over 110%.

DIETARY DATA

Energy intake in infancy—This was estimated by 24-hour dietary recall when the child was seen in the first year.[5]

Energy intake in childhood—Each mother was asked to work back over the previous 24 hours describing everything the child had eaten or drunk. Household utensils, food packages, and details of recipes were used to determine the quantity of food given to the child. Energy intake was then calculated from food tables and manufacturers' information.[8-10]

Age at weaning and duration of breast feeding—Age at weaning was interpreted as the age in weeks when the child was first given food other than milk. Data on this and on the duration of breast feeding were obtainable from the infancy survey.

Results

Eighty of the 203 children reviewed had been overweight as infants and 28 of these had weighed more than 120% of expected weight. At 5 years only 27 children were overweight and five of these weighed more than 120% of expected weight. These results have been discussed in detail elsewhere.[1]

DIETARY DATA

Compared with recommended energy allowances for age,[11] intakes were high in the first six, and particularly the first three, months of life, though at review intakes were similar to those recommended for age (table I). There was no significant correlation between the percentage expected weights of the 5-year-old children and energy intakes at either age. Similarly, the energy intakes of individuals expressed as percentages of the recommended allowance for age did not correlate significantly at either age.

TABLE I—*Mean (±SD) energy intakes in infants and 5-year-old children compared with DHSS recommended dietary allowances for age*[11]

Age (months)	DHSS Recommended allowance (MJ/day)	Boys		Girls	
		No	Intake (MJ/day)	No	Intake (MJ/day)
<3	2·3	28	3·3±1·0	25	3·0±0·6
3-5	3·2	36	3·8±1·0	38	3·5±1·1
6-8	3·8	25	3·5±1·1	22	3·6±0·6
9-11	4·2	11	4·4±1·2	18	4·0±1·2
4·3-6·4 years	7·1*	90†	7·6±1·6	99†	7·2±1·2

*Mean of recommended allowance of 6·7 MJ/day for children aged 3-5 years and 7·5 MJ/day for children aged 5-7 years.
†Dietary data incomplete for 10 boys and four girls at 5-year review. Energy intakes for these children were excluded.
Conversion: SI to traditional units—Energy: 1 MJ/day ≈ 239 kcal/day.

Weaning and breast feeding—The mean age at weaning was 6·4 weeks, and the correlation between age at weaning and percentage expected weight at 5 years was not significant. Sixty-five (32%) of the children reviewed had been breast fed. Twenty of these children were still being wholly breast fed and had not been weaned at 6 weeks' age. In infancy two of these had been obese (more than 120% expected weight), five overweight, 12 normal, and one underweight. At 5 years the same group consisted of no obese, one overweight, 17 normal, and two underweight children.

WEIGHTS OF CHILDREN AND MOTHERS

The correlation between the expected weights of the mothers and those of the infants was not significant ($r = -0·02$), but there was a highly significant correlation between the percentage expected weights of the mothers and those of their 5-year-old children ($r = 0·29$; $P < 0·001$) (table II).

TABLE II—*Weight distribution of children at infancy and 5 years according to maternal percentage expected weight*

Child's weight	Mother's weight		
	Overweight (n = 58)	Normal weight (n = 111)	Underweight (n = 34)
Infancy:			
Overweight (n = 80)	22	48	10
Normal weight (n = 120)	35	62	23
Underweight (n = 3)	1	1	1
5 years:			
Overweight (n = 27)	17	9	1
Normal weight (n = 168)	41	98	29
Underweight (n = 8)	0	4	4

Although significantly heavier, the children of overweight mothers did not have higher energy intakes than the children of underweight mothers (table III).

TABLE III—*Mean (±SD) percentage expected weight and energy intake of children of overweight and underweight mothers*

	Overweight mother			Underweight mother		
	No of children†	Percentage expected weight of child	Energy intake	No of children†	Percentage expected weight of child	Energy intake
Boys	22	106±7*	7·6±1·6	11	97±9*	7·6±1·5
Girls	30	103±10**	7·3±1·4	20	98±6**	7·1±1·2

†Energy intakes of six children of overweight mothers and three children of underweight mothers excluded as dietary data was incomplete.
Significantly different: *$P < 0·01$; **$P < 0·05$.

Discussion

Rapid weight gain in young infants has often been attributed to excessive energy intake, bottle feeding, and early weaning. If an excessive intake leads to rapid weight gain in infancy, it might also lead to failure in the development of appetite control and thus to both continued high intake and an overweight child. In our study many of the children had 24-hour intakes much greater than recommended values for age in infancy, yet their percentage expected weights five years later showed no significant correlation with their intakes in infancy. Furthermore, those children who had had high energy intakes in infancy did not necessarily have high intakes at the 5-year review. Thus overfeeding in infancy and early weaning had not programmed the children for either overweight or a large appetite later.

Since most obese and overweight infants become normal-weight 5-year-olds,[1] perhaps not surprisingly the pattern of feeding in infancy had no significant relation to percentage expected weight at 5 years. Other studies have also failed to relate energy intakes of infants or older children to their percentage weight[12] or their fatness.[13 14] But if energy intake in infancy or at review was not significantly related to weight at 5 years, what were the determinants of overweight in these children?

There was a significant weight relationship between mothers and their 5-year-olds. Overweight 5-year-old children were more likely to have overweight mothers than those of normal weight. Such similarity, although not seen in infancy, was not unexpected and might have been due to overeating within the family. Yet if this had been the case the mean energy intake of children of overweight mothers should have been higher than that of the children of underweight mothers. This was not so. Although significantly heavier than other children, the children of overweight mothers had a mean 24-hour energy intake almost exactly the same as the children of underweight mothers.

The genetic make-up of the children seems to have been more important than their dietary environment in determining their percentage expected weight at 5 years. Such a conclusion is in keeping with twin studies[15] and with the findings of the Ten State Nutrition Survey.[16] Yet the way in which the genetic make-up could influence the effect of similar diet in different individuals remains largely undetermined.

Griffiths and Payne[17] showed that normal-weight children with one obese parent had both lower mean energy intake and lower mean energy output than normal-weight children with two normal-weight parents. In their study an intake adequate for the children of normal parents would presumably have created positive energy balance and eventual overweight in the children with one obese parent. The effect of the over-adequacy for some families of an average intake could be obscured in an adverse environment. In contrast, the good health and social environment of the families in our study may have allowed free expression—in both parents and children—of genetic tendencies to overweight.

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Role of Chlamydia trachomatis and HLA-B27 in sexually acquired reactive arthritis

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Summary and conclusions

Inflammatory arthritis, tendinitis, and fasciitis after non-specific urethritis ("sexually acquired reactive arthritis" (SARA)) was studied prospectively in 531 men with non-specific urethritis, with particular reference to the frequency of isolation of Chlamydia trachomatis and the presence of HLA-B27. Satisfactory cultures were obtained from the urethral swabs from 384 patients; and HLA typing was performed on 482, of whom 30 (6%) were HLA-B27-positive. Arthritis developed in 16 patients, and five of the 14 (36%) with satisfactory cultures were positive for C trachomatis; 135 of the patients without arthritis were also positive for C trachomatis, an identical proportion. Seven of the 15 patients (40%) with arthritis who were HLA-typed were HLA-B27-positive.

Six of the 30 patients with HLA-B27 developed peripheral arthritis in contrast to only nine of the 452 patients lacking the antigen, suggesting a tenfold increased susceptibility. C trachomatis, however, was no more prevalent in cultures from HLA-B27-positive men than from the others. Thus carriage of C trachomatis is unlikely to be influenced by HLA-B27.

C trachomatis may be an important pathogen in some cases of SARA but does not appear to be an exclusive trigger factor for this condition.

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Introduction

Since Sir Benjamin Brodie's observation in 1818[1] of arthritis and inflammatory disorders of the eye after sexually acquired urethritis, increasing epidemiological evidence has suggested that non-specific urethritis and this form of arthritis result from infections. A clinically similar acute arthritis with conjunctivitis and urethritis after gastrointestinal infection was described by Fiessinger and Leroy[2] and by Reiter,[3] and enteric infections with *Shigella*,[1 5] *Salmonella*,[6] and *Yersinia*[7] are now known to cause Reiter's syndrome.

With improved culture techniques,[9] 30-50% of men with non-specific urethritis have *Chlamydia trachomatis* isolated from the urethra[10-12]—even more in selected groups of patients.[13-15] Various strands of evidence have led to the conclusion that *C trachomatis* is at least one of the causative agents of non-specific urethritis.[16-18] It has also been isolated in Reiter's syndrome,[19-22] though the numbers of patients have been too small to establish a causal relationship. Moreover, raised titres of antibodies to Chlamydia have been found,[22 25] and a high proportion of patients with Reiter's syndrome and ankylosing spondylitis affecting peripheral joints have been reported[23 24] to show evidence of lymphocyte sensitisation to chlamydial antigens.

Reiter's syndrome and postenteric reactive arthritis occur especially in individuals possessing the HLA-B27 antigen.[8 26-32] In our study of arthritis and the role of *C trachomatis* in men with new episodes of non-specific urethritis we therefore carried out HLA typing to assess the prevalence of HLA-B27. We refer to arthritis after a proved or putative infection of the genital tract as sexually acquired reactive arthritis (SARA), the