Asthma and infant diet

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SUMMARY The relationship of milk diet and solid feeding practices during the first 4 months of life to rates of early childhood asthma was studied prospectively in a birth cohort of 1110 children. The results of the analysis showed no significant association between rates of asthma and breast feeding or solid feeding practices. This was true for children both of asthmatic and non-asthmatic parentage. It is concluded that there is no evidence to indicate that early breast feeding had any detectable effect on the risk of subsequent asthma in this birth cohort. However, the possibility still remains that breast feeding may have a prophylactic effect for children from highly atopic families.

There have been many studies of the role of breast feeding in the prevention of childhood atopic disease.1–18 Despite strong claims4–14 that breast feeding prevents childhood atopy the evidence is equivocal with many reports finding no benefit.9–13 At least two factors may explain the differing results of these studies.

Firstly, some investigators have overlooked the role of early solid feeding in the development of childhood atopy and have contrasted a group of exclusively breast-fed children receiving no solid food with bottle-fed controls who may or may not have been given solid food.2–5 This design inextricably confounds the benefits of breast feeding with the benefits of early avoidance of other food allergens. Fergusson et al.13 suggested that allergen avoidance might be more important than breast-feeding per se in the reduction of childhood eczema. The study showed a consistent trend for rates of childhood eczema to age 3 years to vary with early solid feeding practices, but no apparent benefit from breast feeding. It was also found possible to produce an artefactual association between exclusive breast feeding and reduced rates of eczema by confounding the effects of early solid feeding with those of breast feeding.

A second factor which may be concerned is the mediating influence of parental atopy. It may be that the benefits of breast feeding exist or are most pronounced for children of atopic parentage, so that studies which fail to take account of parental atopy may obscure a correlation between breast-feeding practices and rates of atopic disease in childhood.

This paper reports the results of a 4-year prospective study of the relationship between parental asthma, early milk diet, solid feeding practices, and rates of asthma in a birth cohort of New Zealand children.

Method

The data were collected during the first six stages of the Christchurch Child Development Study in which a cohort of 1265 infants born in the Christchurch (New Zealand) urban region during the period 15 April to 5 August 1977 was studied at birth, at 4 months, and at 1, 2, 3, and 4 years. At each stage information was collected on the child’s health, social background, and other factors using a structured interview lasting an hour with the child’s mother. This was supplemented by information from hospital records, general practitioner notes, and a diary of the child’s medical attendances kept by the child’s mother. The method of data collection has been described in previous papers.11 18 The following measures were chosen for analysis:

1. Childhood asthma: Had the child had at least 2 medical attendances for wheeze which had been diagnosed by a medical practitioner as either asthma or wheezy bronchitis? Wheezy bronchitis was included in the definition of asthma on the basis of Williams and Mcnicol’s conclusions15 that the two conditions are indistinguishable (of the 77 children classified as asthmatic only 10 were classified as asthmatic on the basis of diagnoses of wheezy bronchitis). In all cases the diagnosis of asthma or wheezy bronchitis was made subsequent to one year. Two diagnoses were used to classify the child as it is well known that the diagnosis of asthma or wheezy bronchitis during early childhood is uncertain.
Information on medical attendance for asthma or wheezy bronchitis was collected annually as part of an interview with the child's mother. In just under two-thirds (61%) of cases this information came from a diary record held by the child's mother in which she recorded her child's history of medical attendance during the year. In the remaining one-third of cases maternal recall was used but if this was at all suspect, direct contacts were made with the family doctor or hospital to obtain confirmation. (2) The child's milk diet during the first 4 months of life: this was based on information collected from (a) maternity unit notes about the child's feeding history shortly after birth; (b) a diary record kept by the child's mother on the child's feeding history during the first 4 months (85% of mothers kept such diaries); (c) questioning of the mother about the child's feeding history to find out if at any time he or she had been given cows' or formula milk.

On the basis of this information, the early milk feeding history of the sample was classified into 2 groups: (a) children who had been either totally bottle fed or partially bottle fed; (b) children whose early milk diet had been only breast milk and who had received no bottled milk by age 4 months.

(3) History of solid feeding: this was collected in a similar way to the information on early milk diet (that is by a combination of a maternal diary record and direct questioning). For the purposes of this analysis the sample was classified into 2 groups: (a) those children who had received no solid food by age 4 months; (b) those children who had been introduced to at least one solid food by age 4 months.

(4) Parental asthma: Was there a history of asthma (past or present) in either or both the child's natural parents? (Consideration was given also to the role of parental eczema in the aetiology of childhood asthma but detailed analysis showed that once parental asthma was taken into account parental eczema had no association with childhood asthma. A report on these relationships has been prepared and is available from the authors).

The analysis is based on a sample of 1110 children for whom complete information on parental asthma and childhood medical history was available. This sample represented 88% of the initial cohort of 1265 children.

Results

Table 1 shows the sample subdivided according to a history of parental asthma, the introduction of solid food into the child's diet by age 4 months, and the child's milk diet during the first 4 months. For each combination of parental asthma, solid feeding, and milk diet the rates of asthma (per 100 children) are shown.

<table>
<thead>
<tr>
<th>Solid food diet (0–4 months)</th>
<th>Milk diet (0–4 months)</th>
<th>Parental asthma</th>
<th>Rates of asthma per 100 children aged 0–4 years (number with asthma/number studied) by parental asthma, infant milk, and solid food diet 0–4 months</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Neither parent</td>
<td>One or both parents</td>
</tr>
<tr>
<td>------------------------------</td>
<td>-----------------------</td>
<td>-----------------</td>
<td>--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>No solids</td>
<td>Bottles or complement-</td>
<td>9.4% (3/32)</td>
<td>5.4% (10/186)</td>
</tr>
<tr>
<td>fed</td>
<td>4.5% (7/154)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Milk only</td>
<td>13.0% (3/23)</td>
<td>7.4% (9/122)</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>10.9% (6/55)</td>
<td>6.2% (19/308)</td>
<td></td>
</tr>
<tr>
<td>Bottle or complement-fed</td>
<td>10.5% (14/133)</td>
<td>6.9% (50/725)</td>
<td></td>
</tr>
<tr>
<td>Solids</td>
<td>Breast milk only</td>
<td>21.4% (3/14)</td>
<td>10.4% (8/77)</td>
</tr>
<tr>
<td></td>
<td>7.9% (5/63)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>11.6% (17/147)</td>
<td>7.2% (58/802)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>6.3% (41/655)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>5.9% (54/908)</td>
<td>6.9% (77/1110)</td>
<td></td>
</tr>
</tbody>
</table>

Conclusions that can be made from Table 1.

(1) Parental asthma was associated with increased risks of childhood asthma; children who had at least one asthmatic parent had rates of asthma that were nearly twice those of children with no asthmatic parent (11.4% (23/202) compared with 5.9% (54/908)).

(2) There was no evidence to suggest that breast feeding had any influence on risks of asthma; indeed, breast-fed children had slightly higher rates of asthma than bottle- or complement-fed children (8.5% (17/199) compared with 6.6% (60/911)).

(3) There was no evidence to suggest an association between early solid feeding practices and childhood asthma: children who had been introduced to solid food during the first 4 months had a rate of 7.2% (58/802) compared with a rate of 6.2% (19/308) for children who had not been given solid food.

(4) Within the series of children of asthmatic parentage, children who were given an exclusive breast-milk diet with no solid food did not appear to have any less asthma (13% (3/23)) than other children (11.2% (20/179)).

These conclusions were confirmed by fitting an interactive logistic model18 to the data in Table 1. The results (Table 2) show that the only factor significantly related to variations in rates of child asthma was parental asthma (χ² = 6.70; df = 1; P<0.01) and that neither solid food nor breast feeding made a significant contribution.

It may also be seen from Table 1 that there was no correlation between asthma in the child's family
and breast feeding practices: of the 202 children of asthmatic parentage 37 (18.3%) received a breast milk diet compared with 162 (17.8%) of the 908 children of non-asthmatic parents.

Discussion

Early breast-feeding was not associated with a reduction in rates of asthma in this birth cohort of children studied to 4 years. This was true for children of both asthmatic and non-asthmatic parentage. Unlike previous studies of this cohort\(^1\)\(^\text{12}\)\(^\text{13}\) which found that exposure to an early diverse solid diet increased risks of childhood eczema, there was no evidence to suggest that early solid feeding was related to early childhood asthma. This is consistent with the hypothesis\(^17\) that while children with eczema are sensitive to dietary allergens, children with asthma are sensitive to inhaled allergens. If this were the case one would expect to see early solid feeding practices implicated in eczema but not in asthma.

This is the third study of this cohort which has failed to find an association between breast-feeding practices and a reduction of atopic disease in early childhood. At the same time it would be incorrect to conclude that these findings for a general population of children imply that benefits for breast feeding cannot be demonstrated in highly selected samples of children. Examination of reports on breast feeding and the reduction of atopic disease shows an interesting association between methods of sample selection and the likelihood of reporting a positive benefit for breast feeding. Of the eight studies reporting positive benefits, five\(^6\)-\(^8\)\(^10\) were based on samples of children chosen for a clear history of parental atopy, one study\(^6\) was based on a general population sample in which children of atopic parentage were thought to be over-represented, and only two\(^1\)\(^7\) were based on population samples. In contrast, of the six studies (including the present study) which have found no benefits, five\(^9\)-\(^11\)\(^13\) have been based on population samples and one\(^12\) on a case control design. This correlation between methods of sample selection and reports of benefits for breast feeding strongly suggests that a correlation between breast feeding and reduced risks of atopic disease may exist only for children with a clear and perhaps severe family history of atopy. It is possible that these children are not represented in sufficient numbers in population samples for significant correlations to be detected. If this were the case one might expect to see relatively large population studies showing no benefit for breast feeding whereas studies of highly selected samples may produce evidence of a benefit. This situation is analogous to the demonstration that placing all children on a diet suitable for children with phenylketonuria has negligible effects on the overall rate of mental retardation while at the same time if children with phenylketonuria are placed on this diet, striking benefits may be demonstrated.

If the preceding conjecture is correct the important issue seems to be not so much whether breast feeding prevents childhood atopy but rather the conditions under which benefits for breast feeding can be shown. It is clear from this study that the simple presence of asthma in one or both of the child's parents is not sufficient to produce conditions which are favourable to demonstrating a correlation between breast feeding and a reduction in childhood asthma in a fairly large sample of children. What is required is further research which discriminates (on the basis of the severity, duration, and extent of atopy in the child's family) the small minority of children who may benefit from breast feeding from the rest of the population who appear to show no reduction in the risk of atopy as the result of breast feeding.

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References

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1983 12–16 April York University
1984 10–14 April York University
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