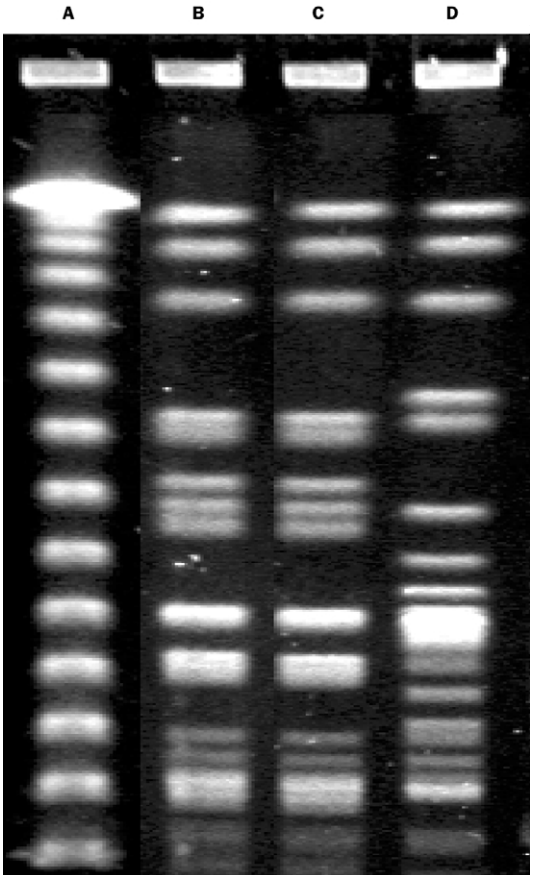

Isolate genotyping by SpeI macrorestriction digestion and pulsed field gel electrophoresis

Lane A, size markers; lane B, CF sputum isolate AU1107; lane C, onion field soil isolate HI2424; lane D, unrelated genomovar III soil isolate included for contrast.

digestion with *Spe*I and pulsed field gel electrophoresis (PFGE) showed slight variations in banding profile among the onion field and CF PHDC isolates, some—eg, cystic fibrosis isolate AU1107 and onion field isolate HI2424, were identical (figure). Finally, PHDC clonal isolates from both cystic fibrosis patients and soil sources macerated onion tissue and contained an identical *pehA* gene (encoding a pectic hydrolase), phytopathogenic features infrequently found among genomovar III CF-related strains. (Additional methodological details are at http://go.to/cepacia).

Our finding in soil samples of a *B cepacia* genomovar III clone that causes extensive infection in patients with cystic fibrosis indicates that human pathogenic strains are not necessarily distinct from environmental strains, an issue central to the debate over commercial use of *B cepacia* complex species in agriculture and bioremediation. This finding also has important implications for infection control in patients with cystic fibrosis, which presently focuses on preventing interpatient spread of *B cepacia* complex. Acquisition of *B cepacia* complex from the natural environment might explain why current control measures have not eliminated the incidence of this infection in patients with cystic fibrosis. Further study is needed to define more specifically the preferred natural habitats of each *B cepacia* complex species. The risk posed by soil-borne strains and the optimal measures to mitigate this risk can then be determined.

Contributors

C F Gonzalez did the initial identification and characterisation of the soil isolates and the *pehA* gene analyses. T Coenye did the *fliC* RFLP analyses. T Spilker did the speciation, genotyping, and cluster analyses. J J LiPuma did the idenfication and genotyping analyses and wrote the paper. All investigators helped to write and revise the report.

Conflict of interest statement

None declared.

Acknowledgments

This work was supported by grants from the Cystic Fibrosis Foundation. The sponsors of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report.

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Breastfeeding and lowering the risk of childhood obesity

Julie Armstrong, John J Reilly, and the Child Health Information Team

Breastfeeding might confer protection against obesity later in life, but the evidence is inconclusive. We tested the hypothesis that breastfeeding is associated with a reduced risk of obesity in a population-based sample of 32 200 Scottish children studied at age 39–42 months in 1998 and 1999. Obesity was defined as body-mass index (BMI) at the 95th and 98th percentiles or higher. The prevalence of obesity was significantly lower in breastfed children, and the association persisted after adjustment for socioeconomic status, birthweight, and sex. The adjusted odds ratio for obesity (BMI ⩾98th percentile) was 0·70 (95% CI 0·61–0·80). Our results suggest that breastfeeding is associated with a reduction in childhood obesity risk.

Lancet 2002; **359:** 2003–04

Few strategies for prevention of childhood obesity are evidence based.[1] Breastfeeding is one strategy that has many benefits, in addition to a possible protective effect on risk of obesity later in life. There are several probable mechanisms by which breastfeeding might protect against obesity, but evidence is inconclusive: most previous studies were limited by sample size and failure to control for confounding variables.[2] In one study,[3] the confounding effect of socioeconomic status was not adequately considered. The results of two other studies[1] were also inconclusive, in part because of limited sample size. Our aim was to test the hypothesis that breastfeeding is associated with a reduced risk of obesity in young children in a population-based, contemporary sample.

We investigated a cohort of Scottish children born in 1995 or 1996, who were routinely reviewed by their health visitor at age 6–8 weeks as part of the Child Health Surveillance Programme. More than 98% of Scottish children attend these health reviews. At this review, the health visitor records whether the baby has been breastfed, fed on formula, or both. In our cohort, 25% of infants were fed only breastmilk, 7% were fed breastmilk and formula, and 68% were fed only formula. Children recorded as exclusively breastfed or exclusively fed on formula were included in the analysis, resulting in a total of 52 394 eligible children. This information was linked to the Child Health

	Number	Obesity		Severe obesity	
		%	Adjusted odds ratio (95% CI)	%	Adjusted odds ratio (95% CI)
Sex					
Female*	15 957	8·1	1·00	4·2	1·00
Male	16 243	9·0	1·03 (0·95–1·11)	4·4	0·96 (0·86–1·08)
Deprivation					
1* (low)	1966	8·0	1·00	3·9	1·00
2	3736	7·9	0·98 (0·80–1·20)	3·8	0·95 (0·71–1·26)
3	6789	8·1	0·98 (0·82–1·18)	3·7	0·90 (0·69–1·17)
4	8140	9·0	1·09 (0·91–1·31)	4·4	1·08 (0·84–1·40)
5	5486	9·2	1·12 (0·93–1·36)	5·2	1·27 (0·98–1·65)
6	4017	8·6	1·02 (0·83–1·25)	4·3	1·03 (0·78–1·37)
7 (high)	2066	8·0	0·99 (0·78–1·25)	4·4	1·11 (0·81–1·53)
Birthweight (g)					
500–2499*	2019	4·7	1·00	2·4	1·00
2500–2999	5105	6·3	1·40 (1·11–1·77)	3·1	1·37 (0·98–1·89)
3000–3499	11 465	7·4	1·68 (1·35–2·09)	3·7	1·67 (1·23–2·25)
3500–3999	9750	9·4	2·22 (1·79–2·76)	4·5	2·08 (1·54–2·82)
⩾4000	3861	14·8	3·77 (3·01–4·72)	7·9	3·86 (2·83–5·26)
Feeding at 6–8 weeks					
Formula*	23449	9·1	1·00	4·6	1·00
Breast	8751	7·2	0·72 (0·65–0·79)	3·4	0·70 (0·61–0·80)
All	32 200	8·5	..	4·3	..

Crude prevalences (%) and adjusted odds ratio (AOR) and 95% CIs were calculated with binary logistic regression and all risk factors entered simultaneously. Definition of obesity is ⩾95th BMI percentile and severe obesity ⩾98th BMI percentile from UK 1990 reference data. *Reference categories for calculation of odds ratios.

Prevalence of obesity and severe obesity at 39–42 months by risk factor

Surveillance Programme routine health check at 39–42 months of age. We excluded 19 874 children because they did not have this check, or because data such as sex, age, socioeconomic status, birthweight, height, or weight were missing. We also excluded 320 children who had body-mass index (BMI) SD scores of greater than 4 or less than −4, because these BMIs were likely to have been incorrectly measured or recorded. After exclusions, sample size was 32 200, which was 62% of the original cohort.

Obesity was defined by expression of each BMI as an SD score relative to UK 1990 reference data. The UK 1990 reference represents the normalised distribution of BMI for age and sex of British children in 1990, and any BMI can be expressed relative to the mean in the form of an SD score (number of SDs above or below the reference mean).[4] We defined obesity as a BMI SD score of 1·64 or greater (95th percentile). This definition is widely accepted; it has high specificity and moderately high sensitivity for children with the highest amount of body-fat, as calculated by direct methods such as body density measurement. Obesity defined in this way tends to persist and is associated with short-term and long-term morbidity.[4] We also defined obesity as BMI on the 98th percentile or higher (BMI SD score 2·06), since that provided a definition of extreme obesity that could be useful in assessment of associations between breastfeeding and obesity later in life.[1] The corresponding BMI values used as definitions were 18·4 kg/m^2 (95th percentile) and 19·0 kg/m^2 (98th percentile) at 39 months; and 18·3 kg/m^2 and 19·0 kg/m^2 at 42 months. BMI was calculated from health visitor measurements of standing height (to 0·1 cm) and weight (to 0·1 kg) at the 39–42-month health check. We quantified socioeconomic status for each family using a geographically defined index, the Carstairs deprivation category.[5] We used logistic regression to test for an association between obesity and breastfeeding, adjusted for sex, birthweight, and socioeconomic status.

Prevalence of obesity greatly exceeded expected frequencies for the reference population of 5% and 2%, respectively (table). Unadjusted prevalence of obesity (based on 95th percentile) was significantly lower among breastfed children compared with formula-fed children, with an odds ratio of 0·78 (95% CI 0·70–0.85) for BMI ⩾95th percentile or higher, and 0·73 (95% CI 0·64–0·83) for BMI ⩾98th percentile or higher. After adjustment for the potentially confounding effects of socioeconomic status, sex, and birthweight, breastfeeding remained significantly associated with a reduced risk of obesity (table). Birthweight was also significantly and positively associated with obesity at age 39–42 months. There were no significant interactions between breastfeeding and socioeconomic status (p=0·48) or birthweight (p=0·14).

In our large study, we have addressed the question of relations between breastfeeding and obesity later in life. The principal strength of our study was its power: even apparently large cohort studies that addressed this question have been underpowered.[1] Some limitations should be noted. The ability to adjust for confounders was limited to those variables obtained as part of the Child Health Surveillance Programme. However, we were able to adjust for socioeconomic status, which was likely to be the most important confounder.[5] The inability to adequately control for social factors has increased the risk of bias arising in previous work. Our findings suggest that breastfeeding is associated with a modest reduction in childhood obesity risk. They also suggest that the reduction in risk is present in early childhood, which is unexpected on the basis of evidence from animals.[1] Breastfeeding is therefore potentially useful for population-based strategies aimed at obesity prevention, particularly with the other benefits that breastfeeding provides.

Contributors

Both authors designed the study and wrote the report. Julie Armstrong and the Child Health Information Team did data collection and analyses.

Conflict of interest statement

None declared.

Acknowledgments

This study was funded by the Scottish Executive Chief Scientists Office. The sponsors of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report.

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