National outbreak of *Salmonella senftenberg* associated with infant food

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SUMMARY

Eight cases of *Salmonella senftenberg* infection in infants were identified in the first half of 1995 in England, five were indistinguishable *S. senftenberg* strains. A case-control study showed an association between illness and consumption of one brand of baby cereal (*P* = 0.03). The cereal manufacturer reported isolating *S. senftenberg* in June 1994 from an undistributed cereal batch. Outbreak strains and the cereal strain were all plasmid-free in contrast to other human isolates of *S. senftenberg* in the same period. Changes in the production process were implemented to prevent further contamination.

Surveillance centres should strengthen the detection and investigation of outbreaks of gastrointestinal infection in susceptible groups, especially young children. In this outbreak, the study of only five cases led to identification of the vehicle of infection. Even when few cases are reported, epidemiological investigation in conjunction with molecular typing may lead to public health action which prevents continuing or future outbreaks.

INTRODUCTION

In July 1995, a case of *Salmonella senftenberg* infection in a 10-month-old infant was reported to the PHLS Communicable Disease Surveillance Centre (CDSC); *S. senftenberg* had also been isolated from a tin of open infant formula milk (Brand A) in the same household. Twenty isolates of *S. senftenberg* had been identified in England and Wales during the first half of 1995 by the Laboratory of Enteric Pathogens (LEP), Central Public Health Laboratory. Seven of the 20 were from infants compared with none of 115 isolates of this serotype in 1993 and 1994 (*P* < 0.01) in England and Wales.

Most cases of infection with *S. senftenberg* in the UK have acquired their infection abroad as this serotype is common in India [1, 2], the Far East and Africa. This serotype has been isolated in England and Wales from coconut products (L. Ward, unpublished data) and animal feed [3, 4]. Nosocomial outbreaks have been documented both in the UK [5] and abroad [1, 2].

In view of the increased incidence of infection by this serotype among infants and possible contamination of a baby food, a national outbreak investigation was set up by CDSC.

METHODS

For the preliminary investigation, a case was defined as an infant with a gastrointestinal illness associated with the isolation of *S. senftenberg* from a faecal sample from 1 January 1995 and no recent history of
Table 1. Case-control study analysis for cereals

<table>
<thead>
<tr>
<th></th>
<th>All cereals</th>
<th>Baby cereals</th>
<th>Brand cereal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cases (n = 5)</td>
<td>5</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>Controls (n = 12)</td>
<td>11</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Fishers exact P-value</td>
<td>1.0</td>
<td>0.05</td>
<td>0.03</td>
</tr>
<tr>
<td>Odds Ratio</td>
<td>—</td>
<td>20.0</td>
<td>OR = undefined</td>
</tr>
<tr>
<td>95% Confidence Interval</td>
<td>—</td>
<td>0.96–1079</td>
<td>* Estimate of lower limit 4.78</td>
</tr>
</tbody>
</table>

* Profile likelihood technique on GLIM.

Parents of cases were contacted and interviewed by telephone using a semi-structured ‘trawl’ questionnaire in order to generate hypotheses as to possible vehicles of infection.

A case-control study to test the hypothesis that there was an association between illness due to \( S.\ senftenberg \) and consumption of any baby food was undertaken. A case was defined as an infant with a gastrointestinal illness associated with isolation of \( S.\ senftenberg \) from a faecal sample reported between 1 January and 31 July 1995. Secondary and travel associated cases were excluded as well as cases shown not to be infected with the outbreak strain on molecular typing. Parents of cases were asked to nominate up to five families with infants aged 5–11 months and living in the same area to act as controls.

Parents of cases and controls were interviewed by telephone. Parents of cases were asked about food consumption in the 3 days preceding illness, and controls the 3 days prior to the interview. Questions were asked about a wide range of baby and infant foods. The data was entered and analysed unmatched on an EPI-INFO 6 database [7] and a statistical package GLIM [8].

Isolates of salmonella were submitted to LEP for identification and confirmation as \( S.\ senftenberg \). Plasmid profiles and chromosomal macrorestriction fingerprinting using pulsed-field gel electrophoresis (PFGE) were performed on all \( S.\ senftenberg \) human isolates received between 1 January and 31 July 1995, and on the strain isolated from the cereal.

The Department of Health contacted the manufacturers of the infant formula milk powder (Brand A) from the first reported case and subsequently the manufacturers of the identified baby cereal (Company B) and one of their suppliers (Company C) and enquired into the manufacturing processes. Public Health Laboratories tested samples of Company B’s baby cereal from retail outlets across the south and east of England.

RESULTS

Eight infants with \( S.\ senftenberg \) infection were identified from laboratory reports within the study period. No increase in infant cases of \( S.\ senftenberg \) was identified in other European countries by Salm-Net.

Although the initial hypothesis was that illness was due to salmonella contamination of a particular infant milk powder, the cases were all fed on different milk powders. Salmonella was not isolated from any milk samples taken from previously unopened tins (Brand A) in retail outlets and from the manufacturer. No association with any type of baby food was identified from the ‘trawl’ questionnaires.

Five infants were included in the case-control study. Three were excluded from the analysis; one had a multidrug-resistant strain, one had an isolate with a different plasmid profile and one case was not available for interview. Twelve controls in total were nominated by parents of cases during the study and their parents interviewed. The age range of the 5 cases and 12 controls was 5–12 months. They lived across the south and east of England (infant population of the regions containing the cases, 331968 [9]).

The main difference demonstrated between cases and controls in single variable analysis related to the consumption of baby cereals. Four out of 5 cases had

Travel abroad, who were not in contact with a case of diarrhoea within 24–72 h of onset of illness. Laboratory directors in England and Wales were requested to send salmonellas with antigenic structure \( O = 1, 3, 19 \) and \( H = \) gst to LEP. Consultants in Communicable Disease Control and Chief Environmental Health Officers were informed about the investigation. Salm-Net collaborators in Europe (a Europe-wide surveillance network for salmonella [6]) were also contacted for details of any additional cases.

Parents of cases were contacted and interviewed by telephone using a semi-structured ‘trawl’ questionnaire in order to generate hypotheses as to possible vehicles of infection.

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The main difference demonstrated between cases and controls in single variable analysis related to the consumption of baby cereals. Four out of 5 cases had
enjoyed one or more brands of baby cereal compared to 2 out of 12 controls (P = 0.05, OR = 2.0, 95% CI 0.96–1079). One particular cereal brand produced by Company B had been eaten by 3 out of 5 cases and none of 12 controls (P = 0.03, OR = undefined, estimate of lower limit 95% CI 4–78). Overall, 4 out of 5 cases had consumed one or more Company B products compared with none of the controls. No other differences in food consumption between cases and controls were statistically significant.

Enquiries to Company B revealed that a batch of bulk cereal supplied to them by Company C in the early summer of 1994 had not arrived in good condition. S. senftenberg had been isolated from a sample of this batch. None of this product was distributed. The bulk cereal had contained the ‘cleaning remains’ from the milling machinery at Company C; these could have contaminated the cereal as other products on the suppliers premises went through the same machines and were not necessarily previously heat treated. The Hazard Analysis Critical Control Points (HACCP) system in place was evaluated and highlighted this hazard. Changes implemented included changing to dedicated machinery for the baby cereal. Salmonella was not isolated from any subsequent samples taken by Company B.

Isolates from the five cases were plasmid-free, in distinction to all other isolates of S. senftenberg received at LEP between 1 January and 31 July 1995. When studied by PFGE, all five isolates had an identical pulsed-field profile (PFP) which was indistinguishable from that of the strain from cereal isolated by the manufacturer in 1994.

No salmonella was identified in 114 samples of the baby cereal from retail outlets.

DISCUSSION

Although the number of cases was small, this case-control study demonstrated a statistically significant association between infection with S. senftenberg and the consumption of one particular brand of baby cereal. A positive association was found without a prior hypothesis for a particular food as the vehicle of infection. A subsequent report from the manufacturer that the same organism had been isolated from one batch of cereal supplied some months before the first infant cases provided strong support for a causal link.

Previous outbreaks of salmonellosis related to infant food stuffs have involved larger numbers of cases and mainly implicated formula milk [10, 11]. There have been no previously reported outbreaks of S. senftenberg associated with an infant food product.

This outbreak has several features of interest. It would not have been detected without the comprehensive local investigation of a single case or without the detailed typing which identified a relatively uncommon serotype with a characteristic plasmid and pulsed-field profile. Even after identification of the outbreak, a successful conclusion seemed unlikely. First, the small number of cases meant that the statistical power of the study was low. Secondly, no hypothesis had emerged after administration of the ‘trawl’ questionnaire. Thirdly, recall by parents of their children’s food history, especially months after illness, was likely to be unreliable. Eating a baby cereal was an important marker in this instance. Fourthly, after confirming the statistical association between illness and the baby cereal, samples of the cereal from retail shelves were not found to be contaminated. The identification of the probable vehicle of infection was only made possible by the manufacturer’s voluntary disclosure. Manufacturers are not obliged to report the isolation of salmonella in food stuffs for human consumption to the local authority but they have an obligation to provide safe food [12].

Deficiencies in the HACCP system in the factory of the cereal supplier were identified. In applying a HACCP system all aspects of a manufacturing process should be considered and all possible sources of contamination taken into account.

Surveillance centres should strengthen the detection and investigation of gastro-intestinal infections in vulnerable groups such as young children. Even with few cases identified, epidemiological investigation in conjunction with detailed microbiological typing may identify the vehicle of infection and result in public health measures that prevent subsequent contamination of food products on a much larger scale [13].

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sampling in the PHLS network and of Mr Stephen Rooke and Dr Lena Robinson, Department of Health, who made enquiries of the companies involved, keeping PHLS informed.

REFERENCES