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A community outbreak of food-borne small round-structured virus gastroenteritis caused by a contaminated water supply

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SUMMARY

In August 1994, 30 of 135 (23%) bakery plant employees and over 100 people from South Wales and Bristol in the United Kingdom, were affected by an outbreak of gastroenteritis. Epidemiological studies of employees and three community clusters found illness in employees to be associated with drinking cold water at the bakery (relative risk 3·3, 95%, CI 1·6–7·0), and in community cases with eating custard slices (relative risk 19·8, 95%, CI 2·9–135·1) from a variety of stores supplied by one particular bakery. Small round-structured viruses (SRSV) were identified in stool specimens from 4 employees and 7 community cases. Analysis of the polymerase and capsid regions of the SRSV genome by reverse transcription-polymerase chain reaction (RT-PCR) demonstrated viruses of both genogroups (1 and 2) each with several different nucleotide sequences. The heterogeneity of the viruses identified in the outbreak suggests that dried custard mix may have been inadvertently reconstituted with contaminated water. The incident shows how secondary food contamination can cause wide-scale community gastroenteritis outbreaks, and demonstrates the ability of molecular techniques to support classical epidemiological methods in outbreak investigations.

INTRODUCTION

The small round-structured virus (SRSV or Norwalk-like virus) is the most common cause of epidemic food- and water-borne viral gastroenteritis [1]. Several large outbreaks due to contaminated drinking water have been described [2–4]. Food-borne outbreaks fall into two main categories according to the source and mode of contamination. They arise either when food contaminated with virus arrives in the kitchen, or when food is contaminated in the kitchen by an infected food handler [5]. Primary contamination is almost invariably associated with molluscan shellfish such as oysters and can cause wide-scale outbreaks [6–8]. By contrast, a range of foods may be secondarily contaminated by food handlers, but outbreaks tend to be well circumscribed, and community outbreaks are uncommon [9]. We describe one of the largest food-borne community outbreaks of this kind identified in the United Kingdom.

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THE OUTBREAK

On 10 August 1994, the personnel department of a large bakery in South Wales notified the local Environmental Health Department (EHD) that 6 of its 135 staff were ill with vomiting and diarrhoea. The premises were inspected and affected employees advised to stay off work for 48 h after symptoms had

resolved. On 15 August, the EHD was informed by two neighbouring EHD of 16 community cases of gastroenteritis all of whom had eaten cakes from outlets of the bakery's retail chain. We undertook an investigation in order to describe the outbreak, identify cases, examine risk factors for illness and determine the cause.

METHODS

Epidemiological investigation

A retrospective cohort study of all 135 bakery employees who had been at work between 1 and 18 August 1994 was conducted. Structured questionnaires were used to obtain details of any illness, shift-work pattern, work responsibilities, canteen use and foods consumed. A case was defined as any employee with either diarrhoea (3 or more loose stools in 24 h) or vomiting during the above period.

Case ascertainment to identify community cases was carried out by alerting all EHDs in South Wales via EPINET (an electronic mail network used for epidemiological surveillance), local general practitioners by telephone, and the general public by means of a news release. Stool specimens recently submitted to the local laboratory were also reviewed and a survey of all bakery outlets in the retail chain undertaken to identify any customers or staff with recent gastrointestinal illness. A case was defined as an individual with either diarrhoea or vomiting and a date of onset between 12 and 14 August, and a cluster as two or more related cases. Cohort studies were carried out on three clusters of community cases. These had occurred among groups of office or factory workers who had purchased a cake selection from different outlets of the bakery's retail chain. Subjects were interviewed using a structured questionnaire to ascertain details of illness and food histories.

We calculated risks with 95% confidence intervals (CI) to measure association of illness with individual risk factors and used the χ^2 test with Yates' correction or Fisher's exact test to derive P values. Multiple logistic regression was performed using GLIM statistical software [10] to fit a sequence of models to assess the relationships between illness and risk factors, taken separately and together, to allow for confounding.

Environmental investigation

The bakery plant was thoroughly inspected, particularly the food production areas, water system, toilet and washing facilities, and the staff canteen. Details of food production were obtained and food hygiene policies and procedures reviewed. In-depth interviews of eight key workers were undertaken.

Laboratory investigation

Stool samples were available for 38 ill bakery employees and 27 community cases. Samples were cultured for bacteria (including *Salmonella*, *Shigella* and *Campylobacter* species) and faecal smears examined by microscopy for oocysts or parasites of *Giardia* and *Cryptosporidium* species. Stool samples from 7 bakery employees and 17 community cases (obtained within 48 h of onset of symptoms) were prepared as 10–20% emulsions in Eagle's minimal essential medium and examined for viruses by solid-phase immune electron microscopy (SPIEM). SRSV were captured onto formvar/carbon grids coated with anti-human IgG and human convalescent serum by placing the grid onto a small drop of faecal emulsion for 2 h at room temperature. The grid was then washed with water (5 s), stained using 1·5% phosphotungstic acid (3 s), dried and examined using a Philips EM420 electron microscope ($\times 65000$).

Samples were also analysed by reverse transcription-polymerase chain reaction (RT-PCR). Viral RNA was extracted from faecal emulsions by RNA enrichment [11] followed by concentration and purification [12]. One hundred μl of emulsion was extracted in 500 μl of Tri-reagent (Sigma-Aldrich Company Ltd, UK) by vortex mixing for 10 min, followed by addition of 100 μl of chloroform and vortexing for a further 10 min and finally, centrifugation (13000 g) for 15 min. The upper phase was transferred to a fresh tube containing 1 ml L6 buffer [12] and 40 μl of silica slurry and the sample processed as described previously [12]. The purified RNA was eluted with 50 μl of sterile distilled water.

For cDNA synthesis, 36·5 μl of the RNA solution was made up to a 50 μl reaction containing 50 mM Tris-HCl (pH 8·3), 75 mM KCl, 10 mM DTT, 3 mM $MgCl_2$, 0·5 mM dNTPs, 1 pg pdN_6 primers (Pharmacia) and 100 units MMLV reverse transcriptase (Gibco-BRL). The reaction was incubated at 37 °C for 1 h. Two μl of cDNA was used to seed each 20 μl PCR reaction containing 10 mM Tris-HCl (pH 9·0), 3 mM $MgCl_2$, 50 mM KCl, 0·1% Triton X-100, 0·2 mM dNTPs and 0·1 μg of either Group 1 or Group 2 SRSV primers [13]. PCR cycling conditions

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were 35 cycles at 94 °C for 20 s (3 min cycle 1), 50 °C for 20 s, 72 °C for 5 s on a Perkin–Elmer 9600. For the capsid primers, the extension phase at 72 °C was increased to 10 s. Fifteen μl of PCR product was mixed with 2 μl of loading dye (50% glycerol in TBE running buffer containing 0·05% bromophenol blue) and loaded onto a TBE-2% agarose gel. SRSV positive specimens were identified by the presence of a 155 bp amplicon for the polymerase primers or a 223 bp amplicon for the capsid primers.

For DNA sequencing, 2 μl of cDNA from PCR positive specimens were amplified in 100 μl preparative reactions by simply scaling up the above PCR reactions. Amplicons were purified by PEG precipitation [14] to remove primer dimers and unincorporated nucleotides, resuspended in water, and chloroform extracted to remove residual traces of PEG. DNA sequencing was carried out using the Dye Deoxy method on an ABI377 automated DNA sequencer (Applied Biosystems). DNA sequence from both strands was obtained by using the same primers as used to produce the amplicon. For polymerase gene amplicons, the DNA product was initially purified by excising the band from TBE agarose gel using a sterile disposable scalpel. The excised band was placed into a screw top microfuge tube containing 1 ml of sterile water and incubated at 100 °C for 10 min to melt the agarose. Preparative PCR was carried out as above using 2 μl of the gel purified product to seed the reaction. Consensus DNA sequences were determined by comparing the sequence of both DNA strands and ambiguities resolved by manual inspection of the electropherograms. Sequences were aligned and phylogenetic trees generated using the programmes CLUSTAL W [15], RETREE [16] and TREEVIEW [17].

Five sets of water samples and environmental surface swabs were taken at the bakery for microbiological examination during August–October. In addition, microbiological results were available from routine water and environmental samples collected by the bakery on 10 and 12 August and tested at a private laboratory. Virological investigations were not carried out on these samples.

RESULTS

Epidemiological investigation

Questionnaires were completed by 133/135 bakery employees of whom 30 (23%) fulfilled the case definition. The first case occurred on 1 August, with an epidemic peak between 9 and 12 August (Fig. 1). Symptoms included diarrhoea (80%), abdominal pain (80%), vomiting (70%), fever (43%) and headache (43%). Median duration of illness was 48 h (range 14–168 h) and no one required hospitalization. Attack rates were similar in both sexes (25% male *vs.* 17% female) and both work shifts (22% day shift *vs.* 25% night shift). There was no relationship between risk of illness and work undertaken in different locations within the bakery, nor with eating bakery products. However, employees were more likely to be ill if they drank cold water either from the drinking fountain (relative risk 3·0; 95% CI 1·5–5·9) of from any other source in the bakery (relative risk 3·3; 95% CI 1·6–7·0) during the week of the outbreak (Table 1). Employees from both day and night shifts ate breakfast in the bakery canteen, and eating breakfast on 8 August (relative risk 19·2; 95% CI 2·5–148·0) or 9 August (relative risk 17·2; 95% CI 2·2–137·9) was associated with becoming ill within 16–48 h of the meal. No illness was reported among canteen staff themselves in the week leading up to 10 August.

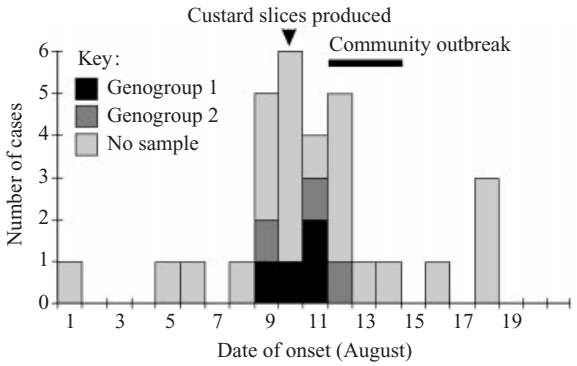

Fig. 1. Epidemic curve of cases among bakery employees ($n = 30$) showing SRSV genogroup identifications among cases and relationship to community gastroenteritis outbreak in South Wales, 1994.

Illness was associated with eating any one of a wide range of cooked foods for breakfast on 8 or 9 August including sausages, fried eggs, bacon, toast, beans and tomatoes, as well as with drinking tea or coffee (Table 1). Using multiple logistic regression to control for drinking water and for the range of foods eaten, only consumption of tomatoes (adjusted odds ratio 6·4; 95% CI 1·0–40·4) and water from the drinking fountain (adjusted odds ratio 4·0; 95% CI 1·7–9·6) on 8 August were associated with illness. Regression models also suggested a significant association between illness and drinking tea or coffee on 9 August. However, only a sub-set of cases were eligible or had

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Table 1. *Activity-specific attack rates for bakery employee cohort* ($n = 133$)

Activity	Exposed			Not exposed			Relative risk (95% CI)	*P* value
	Case*	Total	(%)	Case*	Total	(%)		
Drank water on any day 8–12 August	22	58	38	8	73	11	3·3 (1·6–7·0)	0·0009
Drank water from fountain on any day 8–12 August	20	53	38	10	80	13	3·0 (1·5–5·9)	0·001
Ate breakfast in canteen on 8 August	8	38	21	1	91	1	19·2 (2·5–148·0)	< 0·0001
Sausage	5	13	38	4	116	4	11·1 (3·4–36·4)	0·005
Fried egg	4	10	40	5	119	4	9·5 (3·0–29·9)	0·002
Bacon	3	8	37	6	121	5	7·6 (2·3–24·8)	0·01
Toast	5	33	15	4	96	4	3·6 (1·0–12·7)	0·05
Beans	4	14	29	5	115	4	6·6 (2·0–21·6)	0·008
Tomato	3	6	50	6	123	5	10·2 (3·3–31·3)	0·004
Tea	2	20	10	7	109	6	1·6 (0·3–7·0)	0·63
Coffee	5	30	17	4	99	4	4·1 (1·2–14·4)	0·03
Ate breakfast in canteen on 9 August	6	32	19	1	92	1	17·2 (2·2–137·9)	0·001
Sausage	5	11	45	2	111	2	25·2 (5·5–115·1)	0·0004
Fried egg	4	9	44	2	112	2	24·9 (5·2–117·9)	0·002
Bacon	2	7	28	5	116	4	6·6 (1·5–28·3)	0·05
Toast	5	25	20	2	93	2	7·7 (1·6–37·9)	0·009
Beans	2	10	20	5	111	4	4·4 (1·0–20·0)	0·10
Tomato	3	5	60	4	117	3	17·5 (5·3–58·3)	0·001
Tea	3	18	17	4	106	4	4·4 (1·1–18·1)	0·06
Coffee	6	30	20	1	94	1	18·8 (2·4–150·0)	0·0008

* For breakfast eating, only includes cases who became ill between 16 and 48 h of the meal.

Table 2. *Food-specific attack rates for community cohort* ($n = 42$)

Food	Exposed			Not exposed			Relative risk (95% CI)	*P* value
	Case	Total	(%)	Case	Total	(%)		
Any bakery product	19	37	51	0	5	0	—	0·05*
Any bakery savoury product	0	2	0	19	40	48	—	0·49*
Cream cakes	0	11	0	19	31	61	—	0·0003*
Custard slices	18	20	90	1	22	5	19·8 (2·9–135·1)	< 0·0001†
Other cakes	2	7	29	17	18	49	0·6 (0·2–2·0)	0·002*
Seafood	3	5	60	16	37	43	1·4 (0·6–3·1)	0·64

* Fisher's exact test (2-tailed).
† χ^2 test with Yates' correction.

sufficient data for these analyses and the models were unstable, despite the removal of non-significant variables. Consequently, they could not be reliably interpreted.

One hundred and four community cases from six districts across South Wales and the West Country were identified. The largest single cluster was nine cases. Median age of cases was 40 years (range 11–77 years) and 58% were women. All but four of the cases had bought custard slices on 11 August from 17 of the bakery's 42 retail outlets and all became ill between 12 and 14 August. Median incubation period was 32 h (range 10–71 h).

All 42 persons in the three community groups were interviewed and 19 met the case definition. The main symptoms were diarrhoea (89%), abdominal pain (79%), vomiting (74%), headache (63%), and fever (58%). Eating a custard slice produced by the bakery was significantly associated with illness (relative risk 19·8; 95% CI 2·9–135·1) (Table 2). Eighteen of 20 persons who reported eating a custard slice developed gastroenteritis, a 90% attack rate.

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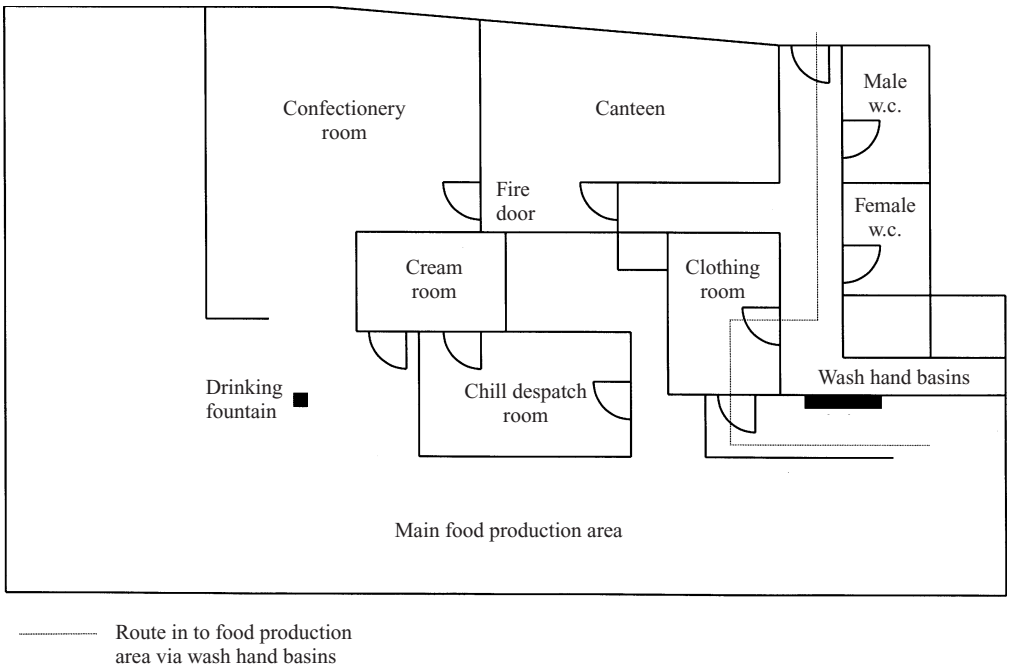

Fig. 2. Floor plan of bakery plant associated with gastroenteritis outbreak in South Wales, 1994 showing relationship of canteen and toilet facilities to food production areas.

Environmental investigation

The bakery plant is of modern design (Fig. 2) and responsible for supplying 42 retail bakeries. Custard slices were made daily in the cream room using a cold custard mix that required no cooking. The custard mix was prepared by day staff from reconstituted custard powder (using water from the public water supply drawn in the cream room) which was mechanically mixed, scooped onto pastry sheets with a metal scoop and levelled with a palette knife before applying a second pastry layer. The slices were kept chilled and finished by night staff who applied pre-prepared fondant icing and cut the slices in the confectionery room. The finished product was placed in trays, taken to the chill despatch room and loaded onto delivery vans. Between 2600 and 3000 custard slices were distributed daily.

In-depth interviews of bakery employees established that Worker 1, shortly after arrival at work on 10 August (the date the suspect batch was produced), developed sudden, explosive diarrhoea and vomiting. He vomited in the toilet bowl, flushed the toilet, washed his hands, reported the incident to his manager and was sent home on sick leave. Worker 2, who was involved in preparation of custard slices on the same day, admitted applying the cold custard mix to the pastry by hand rather than using the designated palette knife. He became ill at home on the following evening, 11 August, at 8.00 p.m. and went on sick leave. Both workers had eaten breakfast in the staff canteen on 8 and 9 August. Staff interviews also revealed a number of unhygienic practices at the bakery. These included unprotected food handling, use of a fire exit between the staff canteen and bakery production area at night (by-passing the hand washing area) (Fig. 2), poor temperature control of water for hand washing and malfunctioning soap dispensers in the toilets.

Laboratory investigation

One community case was positive for *Salmonella mbandaka* and samples from two bakery employees yielded *Campylobacter* sp. and enterotoxogenic *Staphylococcus aureus*, respectively. SRSVs were identified by SPIEM in samples from 1 bakery employee and 3 community cases. However, SRSV RNA was detected by RT-PCR in 6 employees and 8 community cases using capsid primers, and in 7 employees and 12 community cases using polymerase primers. RT-PCR is more sensitive than EM in detecting SRSV and, in our experience, the polymerase primers have a better detection rate than the capsid primers. However, the polymerase primers

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Table 3. *Detection of SRSV in stool samples from bakery employees and community cases by solid-phase immune electron microscopy (SPIEM) and reverse transcription-polymerase chain reaction (RT-PCR) for polymerase and capsid primers*

		Polymerase primers		Capsid primers	
Identity	SPIEM	Group 1	Group 2	Group 1	Group 2
Baker A	+	+	−	+	−
Baker B	−	+	−	−	−
Baker C	−	+	−	+	−
Baker D	−	+	+	−	+
Baker E	−	+	+	+	−
Baker F	−	+	+	−	+
Baker G	−	+	+	−	+
Bristol A	−	+	+	+	−
Bristol B	−	+	−	+	−
Bristol C	+	−	−	−	−
Bristol D	+	+	+	−	−
Cardiff A	−	+	+	−	+
Cardiff B	−	+	+	−	−
Cardiff C	−	+	−	+	+
Cardiff D	−	+	+	−	−
Cardiff E	−	+	+	+	+
Swansea A	−	+	+	−	+
Swansea B	+	−	−	−	+
Swansea C	−	−	−	−	−
Swansea D	−	−	−	−	−
Swansea E	−	−	−	−	−
Swansea F	−	+	+	−	−
Swansea G	−	+	+	−	−
Swansea H	−	+	+	+	+

are less discriminatory, and consequently, genotype information is not always resolved (Table 3).

Nucleotide sequences from the polymerase region were obtained for 4 employees and 7 community cases (Fig. 3). Two employees had identical group 1 virus sequences and 2 other employees had extremely similar group 2 sequences (99% identity). Three community cases had group 1-like sequences which were distinct from each other and from the sequences identified in bakery employees. The sequence of the polymerase amplicon from the virus in the stool of one consumer (Bristol A) proved particularly difficult to resolve. It possessed similarities to sequences identified in both employees and community cases and may indicate a mixed infection of genogroup 1 viruses that were co-amplified by the polymerase primers. One group 2 sequence from a community case (Swansea F) was identical to that identified in an employee (Baker F) and had 99% identity with a sequence from Baker G. Three other community cases with a different genogroup 2 virus had identical (Bristol D and Cardiff E) or similar (Swansea A; 99% identity) nucleotide sequences.

The possibility that community cases carried mixed infection was investigated by sequencing capsid amplicons (Fig. 4). This confirmed that three community cases were carrying both group 1 and group 2 viruses. Two cases (Cardiff C and Swansea H) had group 1 viruses with similar sequences (96·8% identity), but the third case (Cardiff E) carried a completely different sequence type (69·5% identity). All three cases had group 2 viruses which were closely similar, two being identical (Cardiff E and Swansea H) and the third (Cardiff C) showing 98·8% identity. A further two amplicons were sequenced for group 1 virus from an employee (Baker A) and a community base (Bristol A) and were found to be identical to each other, but distinct from the sequence types carried by the community cases with mixed infections (Fig. 4).

A sample of water obtained from the drinking

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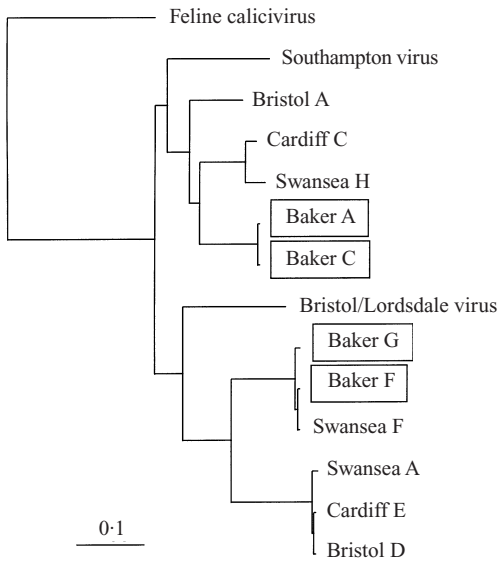

Fig. 3. Dendrogram showing genetic relationships of polymerase region amplicons of SRSV identified in the South Wales outbreak, together with equivalent regions of Southampton virus (genogroup 1), Bristol/Lordsdale virus (genogroup 2) and feline calicivirus for comparison. Letters denote individual community or bakery employee cases. The length of the abscissa to the connecting node is proportional to the genetic distance between sequences. The scale represents nucleotide substitutions per site.

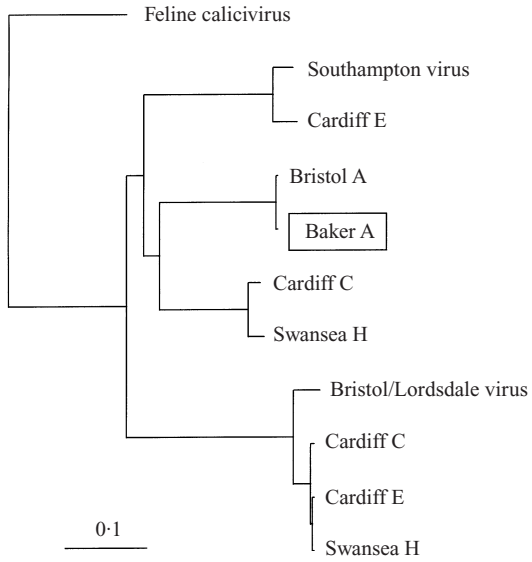

Fig. 4. Dendogram showing genetic relationships of capsid region amplicons of SRSV identified in the South Wales outbreak, together with equivalent regions of Southampton virus (genogroup 1), Bristol/Lordsdale virus (genogroup 2) and feline calicivirus for comparison. Letters denote individual community or bakery employee cases. The length of the abscissa to the connecting node is proportional to the genetic distance between sequences. The scale represents nucleotide substitutions per site.

fountain in the food production area on 10 August grew coliforms > 1200/100 ml, *Escherichia coli* > 1200/100 ml and faecal streptococci 160/100 ml. Unfortunately, this water sample was not available for virological analysis. Environmental swabs from numerous food preparation areas and sinks also taken on 10 August were all negative. Water samples taken on 12 August from the same drinking fountain, the mains supply, a chilled outlet and the cream room supply (where the custard slices were produced) grew no coliforms or faecal pathogens. Further samples taken between August and October from the mains water supply and from various water outlets and food preparation areas at the bakery were all negative.

DISCUSSION

This is one of the largest community outbreaks of food-borne viral gastroenteritis due to secondary food contamination to be reported in the United Kingdom. It followed an apparent point source outbreak of gastroenteritis at a large bakery plant that peaked on 10 August. The epidemic curve, clinical features, and detection of SRSV in stool confirm this outbreak as viral gastroenteritis. Epidemiological studies identified custard slices as the vehicle of transmission, and detection of SRSV of identical nucleotide sequence in samples from bakery employees and community cases imply a common source outbreak. The identification of multiple SRSV strains in both bakery employees and community cases, and of mixed infections in community cases, strongly supports the view that the source of the outbreak was contamination of the bakery water supply. Mixed infections have only previously been reported in outbreaks associated with the consumption of shellfish [18] or from bathing in sewage contaminated recreational water [19].

The hypothesis that the bakery outbreak was most probably caused by contaminated water supply is supported by epidemiological evidence of an association between illness among bakery employees and drinking cold water at the bakery; microbiological evidence of heavy faecal contamination of the drinking fountain on 10 August; and by illness due to multiple SRSV strains. Alternatively, the outbreak might have been caused by a vomiting incident in the bakery canteen with secondary aerosol spread, but no such event was reported. Nor can the outbreak be explained by use of a contaminated toilet since both sexes were similarly affected in spite of the existence of separate male and female toilets. Finally,

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neither of these explanations can explain illness due to multiple SRSV strains.

The community outbreak was associated with eating custard slices. These were most probably contaminated by the use of contaminated water to reconstitute the cold custard mix. This hypothesis is supported by the presence of mixed infection due to both SRSV genogroups and several genotypes in community cases. Classically, food-borne spread of SRSV follows direct contamination by a food handler. This is what occurred in one previously reported bakery-associated outbreak where an ill employee was found to have contaminated cakes by submerging his arms in buttercream frosting [20]. Although the employee who prepared the custard slices reported illness, his date of onset was 11 August, the day after the implicated batch was produced. The employee may have incorrectly reported his illness onset date, but we found no evidence to contradict his account. Alternatively, the custard slices might have been contaminated by pre-symptomatic excretion of virus [21, 22] or by direct virus transfer [23], as have been suggested in previous outbreaks. However, none of these explanations account for the virological findings.

Previous molecular studies of SRSV outbreaks have been able to show identical nucleotide sequences in all cases in point source food-borne outbreaks [24] or in index and secondary cases in outbreaks involving spread from person-to-person [25, 26]. Sequence heterogeneity among SRSV is such that identical sequences in outbreaks strongly support a common source of infection. Co-infection with both genogroup 1 and 2 has occasionally been reported [18, 19], but only as a consequence of consumption of contaminated shellfish (which are filter feeders able to concentrate a mixture of viruses in their mid gut gland) [18] or from bathing in contaminated recreational water [19].

In this outbreak, molecular biology identified both SRSV genogroups, with several genotypes in each group, among both bakery employees and community cases. Some of the sequences identified in bakery employees were identical to some of those found in community cases but there were some sequence types unique to bakery employees and some unique to community cases (although not every community case was studied at the molecular level). The absence of mixed infections in the few bakery employees that were tested by PCR may either reflect prior immunity to some virus strains or may be due to infection by secondary spread from workmates (3 of 4 employees in whom genogroup identification was successful all had late illness onset dates). No faecal sample was available from the bakery employee who could have contaminated the custard slices.

By contrast, three community cases had mixed genogroup infections. Altogether, six different genotypes (3 group 1 and 3 group 2) were identified among community cases from the polymerase region (assuming even small variations in sequence are significant), and six genotypes (4 group 1 and 2 group 2) from the capsid region. This suggests that the custard slices may have been contaminated by at least seven SRSV sequence variants. Some of the sequence variations are small (around 1%) and may be a consequence of Taq polymerase-induced differences. However, even if only gross variations are considered as significant there remains evidence of infection of community cases by at least 4 or 5 distinct sequence types. Additionally, the community cases with mixed infections had both genogroup 1 and 2 in their stools.

How did the water supply become contaminated? At the time of the outbreak, no obvious explanation for possible contamination of the bakery water supply was found. Subsequently, during investigation of an outbreak of gastroenteritis among employees of another premises on the same industrial estate, an unauthorized connection between river water and the mains supply without isolating valve control was identified at a nearby factory. It is possible that this connection may have allowed intermittent reverse flow contamination of mains water supply when mains water pressure was unusually low. A similar situation has been described due to reverse flow between a farm's water irrigation system and the drinking water supply that caused gastrointestinal illness in a local rural community [27]. Unfortunately, none of the contemporary bakery water samples were available for virological analysis.

The batch of custard slices that caused the community outbreak was prepared by the night shift on 10 August and distributed to 42 retail bakeries early the following morning. Over 2600 custard slices were sold from the contaminated batch, so as many as 2300 cases may have occurred (assuming a uniform 90% attack rate). However, only 104 cases were reported and only 17 of 42 retail outlets implicated. A variable attack rate due to variable contamination of custard slices is unlikely if the source was contaminated water. Extensive undernotification of cases may therefore have occurred.

This outbreak demonstrates the value of molecular

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techniques in the investigation of SRSV outbreaks. It also highlights the potential for wide-scale outbreaks of food-borne viral gastroenteritis even when the products and premises involved are generally regarded as low risk. As the scale of production and distribution of food products grows, the potential for large outbreaks which cross both district and national boundaries also increases. Several international food-borne outbreaks have been reported in recent years [28]. This outbreak illustrates not only the hazards of secondary viral contamination of food but also serves to emphasize the importance of good cross-boundary communication to ensure effective outbreak management and control.

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