A hospital outbreak of salmonella food poisoning due to inadequate deep-fat frying

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SUMMARY

In an outbreak of plasmid-free *Salmonella enteritidis* phage type 4 (PT4) food poisoning at a hospital for mentally handicapped people in July 1990, 101 residents and 8 staff were affected and a cohort study implicated beef rissoles cooked by deep-fat frying as the vehicle of infection (relative risk 2.92, 95% confidence interval 1.73–4.93, $P \leq 0.001$). Replication of the cooking process demonstrated that the rissoles achieved core temperatures of only 48–60 °C despite external temperatures of 91–95 °C and an oil temperature of 142–154 °C. No residual food was available for microbiological testing but plasmid-containing *S. enteritidis* PT 4 was isolated in shell eggs from the hospital kitchen.

INTRODUCTION

In the U.K. there are about 25–30 outbreaks of salmonella infection in hospital each year [1]. Most of these involve fewer than 10 patients or staff and are usually due to person-to-person spread; but food poisoning accounts for a disproportionate number of larger outbreaks [2]. Hospital outbreaks are of especial concern both because of the increased susceptibility of hospital patients and because they can seriously disrupt health services. In hospitals for the care of the psychiatrically ill or mentally handicapped such outbreaks are particularly hazardous, and secondary spread can cause severe management problems [3, 4].

Hospital outbreaks are often poorly investigated. No information was available on the source of infection in 107/248 (46%) reported hospital outbreaks of salmonella in England and Wales between 1978–87 [1]. Fifty-seven were attributed to food poisoning but in only half of these was the specific food vehicle identified. In addition to microbiological

investigation of hospital outbreaks, clinical and epidemiological data need to be collected to identify vehicles of infection [2, 5]. We describe how the results of epidemiological investigation allowed us to focus on specific cooking procedures and hence pinpoint the cause of the outbreak.

METHODS

In July 1990, an outbreak of salmonella occurred at a 300-bed hospital for the care of people with a mental handicap. The hospital consists of an older core of Victorian buildings, together with a group of newer wards about 20 years old. The hospital kitchen is modern, spacious and well-equipped and provides meals to all wards on site and to the staff canteen. At the time of the outbreak there were 15 wards in use, each housing between 7 and 27 residents grouped together on the basis of age, sex, mental and physical ability. The total hospital population was 293 (including 20 residents who were away on holiday).

On the morning of 12 July, 13 residents on 4 different wards were reported to be ill with acute gastro-enteritis later confirmed to be due to salmonella infection. Infection control measures were introduced and preliminary investigation commenced. An Outbreak Control Team was convened the following day, and it was decided to close the hospital to admissions and screen all residents and staff. Altogether, 101 residents and 8 staff had salmonella infection including 1 resident who died of salmonella septicaemia. Investigations centred on food prepared by the hospital kitchen.

Epidemiological investigation

A case-control study of the first 44 ill residents was conducted. Cases were defined as residents with diarrhoea or with fever plus any other gastrointestinal symptom with a date of onset between 11 and 13 July. The next resident listed on the ward's alphabetic roll was selected as a control. A brief questionnaire to ascertain personal and clinical details, time of onset of illness and food consumption history (based on the menu ordered) was completed by nursing staff on behalf of each resident.

A cohort study of all hospital residents present during the week beginning 8 July was subsequently conducted. Nursing staff completed a questionnaire giving details of food eaten by each resident for supper on 10 July. Cases were defined as residents with microbiologically confirmed salmonella infection.

Data were analysed using Epi Info, Version 5 [6]. Food preference tables were constructed and probabilities calculated using χ^2 with Yates' correction, or Fisher's exact test for expected values less than 5. For the cohort study, relative risks were calculated for dichotomous variables with Greenland Robins 95% confidence intervals (CI) and the correlation coefficient used for continuous variables.

Microbiological investigation

Faecal specimens from all residents and ill staff at the hospital and from all catering staff were cultured. Confirmatory serotyping, phage typing and plasmid analysis were carried out by the Public Health Laboratory Service Division of Enteric Pathogens.

There were no residual cooked food samples but samples of ingredients used for the implicated food-

stuff, including eggs and bread crumbs, were examined. The eggs tested were from a different batch but from the same distributor. Eggs were processed in pooled batches of 120 or 180 and cultured for salmonellae on selective media – brilliant green agar and xylose lysine desoxycholate agar or desoxycholate citrate agar – after pre-enrichment in double strength buffered peptone water and enrichment in Rappaport 10 medium. Foods other than eggs were enriched in single strength selenite broth, and sub-cultured to brilliant green and desoxycholate citrate selective agars. Water samples from the hospital's water supply were examined for coliforms and *Escherichia coli* by standard methods as well as for salmonellae using pre-enrichment, enrichment and selective stages with double strength buffered peptone water, Rappaport 25 broth and brilliant green agar.

Environmental investigation

Kitchen facilities were inspected, hygiene practices reviewed and details of menus for the relevant period obtained. All catering staff involved in preparing, cooking or serving the suspect food were interviewed. Preparation methods for meals served immediately prior to the outbreak were ascertained. The entire process for preparing the implicated food vehicle was replicated including cooking experiments. Refrigeration, cooking, serving and food distribution temperatures were checked.

RESULTS

Epidemiological

Questionnaires were returned for all 44 cases and 44 controls from 9 of the hospital's 15 wards. Mean age of cases was 45 years (range 24–78 years) and of controls 43 years (range 19–73 years) and 28 (64%) cases were male compared with 34 (77%) controls. Of the 44 cases, 41 (93%) had diarrhoea, 27 (61%) had fever, 12 (27%) had vomiting and 6 (14%) had abdominal pain. Peak time of onset of illness was during the morning of 12 July with the earliest cases occurring at 04.00 h on 11 July. The main food associations are shown in Table 1. Analysis of food histories showed no clear association between illness and consumption of a particular food(s). The highest odds ratios were associated with each of the main menu items for supper on 9 July (sausage), lunch on

Table 1. Association between illness and foods eaten by cases and controls

Food	Cases (n = 44)		Controls (n = 44)		Odds ratio (95% CI*)	P value
	Ate	Not ate	Ate	Not ate		
9 July supper						
Sausage	38	6	31	13	2.63 (0.82–9.46)	0.12†
Chicken salad	3	41	1	43	3.11 (0.24–169)	0.62‡
10 July breakfast						
Bacon	39	5	38	6	1.23 (0.29–5.55)	1.00†
10 July lunch						
Lamb and kidney pie	39	5	34	10	2.27 (0.63–9.35)	0.26†
Minced lamb	3	41	6	38	0.47 (0.07–2.37)	0.48‡
10 July supper						
Beef rissole	39	5	33	11	2.57 (0.73–10.45)	0.17†
Sandwiches	1	43	1	42	0.98 (0.03–37.17)	1.00‡
Egg savoury	2	42	6	37	0.30 (0.03–1.80)	0.16‡

* 95% confidence interval.

† χ^2 test with Yates' correction.

‡ Fisher's exact test (two tailed).

Fig. 1. Epidemic curve for 57 symptomatic cases of salmonella food poisoning.

10 July (lamb and kidney pie) and supper on 10 July (beef rissole). Subsequent investigation focused on the 10 July supper since this was the meal implicated by the epidemic curve.

The cohort study yielded completed questionnaires for all 273 residents present at the time of the outbreak. The final epidemic curve was consistent with a point-source outbreak with a peak on the morning of 12 July (Fig. 1). The overall attack rate was 37% (101/273) with considerable variation between wards (Table 2). On the one ward which did not have any beef rissoles there were no cases. There was a close correlation between beef rissole consumption by ward and ward attack rates ($r = 0.88$, 95% CI 0.66–0.96). Overall, only consumption of beef

rissole was significantly associated with an increased risk of infection (relative risk 2.92, 95% CI 1.73–4.93, $P \ll 0.001$) (Table 3).

Microbiological

Salmonella enteritidis PT4 was isolated from 101 residents and 8 staff (including 3 catering staff), although only 57/101 (56%) residents had symptoms. The organism isolated from cases was plasmid-free, but a *Salmonella enteritidis* PT4 isolated from pooled egg samples had a single 38 MDa plasmid. Salmonellae were not isolated from other foodstuffs, and no *E. coli* or salmonellae were cultured from the water samples.

Table 2. *Ward-specific attack rates for salmonella infection and numbers of rissoles consumed per ward*

Ward	Total residents	Salmonella positive (% attack rate)	Ate beef rissole (%)
A	18	11 (61)	12 (67)
B	19	10 (53)	17 (77)
C	22	11 (50)	22 (100)
D	10	5 (50)	10 (100)
E	24	12 (50)	24 (100)
F	23	11 (48)	16 (70)
G	7	3 (43)	6 (86)
H	26	11 (42)	20 (77)
I	20	7 (35)	20 (100)
J	24	8 (33)	18 (75)
K	27	9 (33)	15 (56)
L	13	2 (15)	5 (38)
M	16	0 (0)	5 (31)
N	24	0 (0)	0 (0)
All wards	273	100 (37)	190 (70)
On holiday	20	1 (5)	0 (0)
Total	293	101 (34)	190 (65)

Correlation coefficient $r = 0.88$, 95% CI 0.66–0.96.

Environmental

All three salmonella positive catering staff had some link with the beef rissoles. One helped prepare them and had tasted a spoonful of the mixture prior to cooking. Another had been asked to discard the first batch of rissoles from the fryer because they were overcooked and had consumed one in the process. The third was a member of dining room staff who had handled left-over rissoles when clearing plates after supper.

The beef rissoles were prepared on 7 July, refrigerated and then deep-fried immediately prior to being served for supper on 10 July. They were made from pre-cooked minced meat, reconstituted dried potato, carrots, onions and seasoning, and the mixture bound with shell eggs. Mixing was done mechanically, but the mixture was then scooped out by hand and formed into around 240 3 oz rissoles which were placed on trays in a refrigerated larder until the following day. The rissoles were then passed through containers of flour, egg-wash and bread crumbs and returned to the refrigerator. Rissoles were deep-fried in batches of exactly 50.

The replication experiment was observed by one of the authors (PGH) and carried out by the same chef who had prepared the rissoles for the 10 July meal. Frying times varied between 5 and 7 min depending

on the oil temperature. Oil temperature fell from 157 to 144 °C after addition of the first batch of 50 rissoles and then gradually recovered. Rissoles were considered cooked when they floated to the surface of the oil, but achieved core temperatures of only 48–60 °C despite external temperatures of 91–95 °C and oil temperatures of 142–154 °C. Core temperatures were related to oil temperature and cooking time (first batch, 60 °C after 7 min at 144 °C; second batch, 48 °C after 6 min at 142 °C; third batch, 50 °C after 6 min at 147 °C; fourth batch, 50 °C after 5.5 min at 154 °C). After frying, each batch of rissoles was transferred to a bain-marie. Meals were plated up ward-by-ward in the same order as the rissoles had been fried and transferred to the ward by means of heated trolleys.

DISCUSSION

In the largest hospital outbreak of salmonella reported in the U.K., at Stanley Royd psychiatric hospital, over 400 people were affected and 19 people died [3]. This contrasts with the outbreak we describe in which salmonella infection was confirmed in 101 residents of whom 44 were asymptomatic, but only 5 required transfer for medical care, and only 1 died. The high proportion of asymptomatic infections may have been due to a low inoculum or may reflect variation in individual susceptibility [7]. It also posed infection control and clinical management problems similar to those experienced elsewhere [2, 4, 8], because of the severe mental and sometimes physical handicap of the hospital's residents. In order to achieve rapid elimination of salmonella carriage we decided on a strategy of using mass treatment with ciprofloxacin, the results of which are described in detail elsewhere [9].

Detailed epidemiological and environmental investigations of this outbreak implicated beef rissoles served for supper on 10 July as the vehicle of infection. Deficiencies in the preparation procedure included preparation too far in advance, poor temperature monitoring, and inadequate cooking. Raw shell egg was used as a binding agent for the rissoles and *S. enteritidis* PT4 was isolated from eggs, but the contaminated eggs belonged to a different batch from that used for the rissoles, and the egg isolates were a different plasmid type to the human isolates. Variability both between case isolates, and between case and food isolates, have been described in other egg-associated outbreaks [10, 11]. However, since no other rissole ingredients apart from breadcrumbs were

Table 3. *Food-specific attack rates from cohort study of all hospital residents (n = 273)*

Food	Eaten			Not eaten			Relative risk
	Case	Total	%	Case	Total	%	
Beef rissole	87	190	(46)	13	82	(16)	2.92†
Sandwiches	7	48	(15)	93	225	(41)	0.35*
Egg savoury	1	11	(9)	99	262	(38)	0.24
Other	5	24	(21)	95	249	(38)	0.55

* 95% confidence interval 0.17–0.71, $P < 0.001$.

† 95% confidence interval 1.73–4.93, $P \leq 0.0001$.

available for examination, these cannot be excluded as possible alternative sources of contamination of the rissoles.

The initial case-control study had 90% power to identify a difference between groups at the 95% confidence level when 95% of cases were exposed, compared with 65% of controls. Misclassification due to asymptomatic infection of some controls probably contributed to the inconclusive result: 13 of the 44 controls were subsequently found to have salmonella infection. Asymptomatic salmonella infection is a recognized problem in hospital outbreaks and the need to exclude symptomless excretors from control groups is one justification for screening symptomless patients [5]. Furthermore, limited menu choice (most hospital residents ate the principal menu item at each meal) and difficulties in obtaining food histories (staff may have reported residents' food preferences rather than food eaten) may have led to measurement bias. Selecting controls from the same ward as cases also inadvertently led to overmatching. The hospital policy of grouping residents by ward according to age and mental ability meant that hospital ward acted as a confounding variable, since wards with elderly, disabled residents were more likely to order the 'soft food' option from the menu. This menu ordering pattern also accounted for the extreme variation in ward attack rates.

The cohort study was made easier by having a captive resident population, and the restricted daily menu limited recall errors by staff completing questionnaires. It is possible that the association suspected with beef rissoles may have biased the results, although these suspicions were not made public to hospital care staff until after the cohort study had been completed. The association between risk of illness and eating beef rissoles is further strengthened by the correlation between quantity of rissoles consumed per ward and ward attack rates.

The main contributory catering problem was inadequate cooking. Beef rissoles were judged to be cooked when they had turned a golden brown colour and risen to the surface of the oil in the deep-fat fryer. It was assumed that high oil temperature assured even cooking temperature throughout the food. The importance of measuring core temperature of food during and after cooking had not been appreciated, and routine monitoring was limited to recording temperatures of plated meals. This investigation demonstrates the value of replicating the cooking process as a means of establishing the cause of an outbreak.

The outbreak also illustrates the continuing hazard of salmonella food poisoning in hospitals. Large food-borne outbreaks are uncommon but can be prevented by good catering practice. Food should be prepared and cooked on the day it is to be eaten, adequately refrigerated and thoroughly heated. Department of Health guidance to the health service advises against use of raw shell eggs in food to be eaten without further cooking [12]. Replacing raw shell egg with pasteurised egg in all egg-containing recipes is recommended. We believe prompt investigation and rapid institution of control measures limited the extent of this outbreak, and that the use of ciprofloxacin ensured a speedier return to normal than would otherwise have been possible.

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REFERENCES

1. Joseph CA, Palmer SR. Outbreaks of salmonella infection in hospitals in England and Wales 1978-87. *BMJ* 1989; **298**: 1161-4.
2. Palmer SR, Rowe B. Investigation of outbreaks of salmonella in hospitals. *BMJ* 1983; **287**: 891-3.
3. Department of Health and Social Security. Report of the committee of inquiry into an outbreak of food poisoning at Stanley Royd Hospital. London: HMSO, 1986.
4. Meara J, Mayon-White R, Johnston H. Salmonellosis in a psychogeriatric ward: problems of infection control. *J Hosp Infect* 1988; **11**: 86-90.
5. Cruickshank JG. The investigation of salmonella outbreaks in hospitals. *J Hosp Infect* 1984; **5**: 241-3.
6. Dean AD, Dean JA, Burton AH, Dicker RC. Epi Info, Version 5: a word processing, database and statistics programme for epidemiology on micro-computers. USD Incorporated, Stone Mountain, Georgia, 1990.
7. Blaser MJ, Newman LS. A review of human salmonellosis. I. Infective dose. *Rev Infect Dis* 1982; **4**: 1096-106.
8. Lightfoot NF, Ahmad F, Cowden J. Management of institutional outbreaks of salmonella gastroenteritis. *J Antimicrob Chemother* 1990; **26** Suppl. F: 37-46.
9. Dyson C, Ribeiro CD, Westmoreland D. Large scale use of ciprofloxacin in the control of a salmonella outbreak in a hospital for the mentally handicapped. *J Hosp Infect* 1995; **29**: 287-96.
10. Lin FYC, Morris JG Jr, Trump D, et al. Investigation of an outbreak of *Salmonella enteritidis* gastroenteritis associated with consumption of eggs in a restaurant chain in Maryland. *Am J Epidemiol* 1988; **128**: 839-44.
11. Telzak EE, Budnick LD, Greenberg MSZ, et al. A nosocomial outbreak of *Salmonella enteritidis* due to the consumption of raw eggs. *N Engl J Med* 1990; **323**: 394-7.
12. Department of Health. Raw shell eggs. Department of Health, London, 1988. EL(88)P/136.