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## A case-control study of domestic kitchen microbiology and sporadic *Salmonella* infection

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### SUMMARY

The microbiology of domestic kitchens in the homes of subjects who had suffered sporadic *Salmonella* infection (cases) was compared with control domestic kitchens. Case and control dishcloths and refrigerator swabs were examined for the presence of *Salmonella* spp., total Enterobacteriaceae counts and total aerobic colony counts. *Salmonella* spp. were isolated from both case and control dishcloths and refrigerators but there were no significant differences between the two groups. Colony counts were similar in case and control dishcloths and refrigerator swabs. There was no relationship between the total counts and presence of *Salmonella*. There was no evidence that cases of *Salmonella* infection were more likely to have kitchens which were contaminated with these bacteria or have higher bacterial counts than controls. Total bacterial counts were poor indicators of *Salmonella* contamination of the domestic kitchen environment. Further factors which could not be identified by a study of this design may increase risk of *Salmonella* food poisoning. These factors may include individual susceptibility of the patient. Alternatively, sporadic cases of *Salmonella* food poisoning may arise from food prepared outside the home.

### INTRODUCTION

Cross contamination is an important contributory factor in outbreaks of foodborne disease originating from both commercial and domestic kitchens [1, 2] but its role in sporadic cases of food infection is less clear [3]. Microbiological studies indicate that there are significant opportunities for cross contamination in the domestic kitchen; labelled microbial strains

or zoonotic pathogens have been found on hands, utensils, cloths, equipment and surfaces after inoculated raw poultry meat and eggs [4, 5] and naturally contaminated chicken carcasses [6] were handled. *Salmonella* can survive for at least 24 h on kitchen surfaces providing an ongoing source of contamination [5]. One feature of domestic kitchens, the dishcloth, has been shown to be an ideal environment for bacterial multiplication and heavily contaminated dishcloths have been sampled in commercial and domestic kitchens [7, 8]. However, epidemiological evidence linking the microbiological status of domestic kitchens with the likelihood of a case of food poisoning arising from food prepared in that kitchen has yet to be established.

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We conducted a case-control study of sporadic *Salmonella* infection to investigate domestic kitchen risk factors. Data on food-handling risk factors have been published. We found that food purchasing and food hygiene practices did not differ between case and control households [3]. Here we compare the microbiological environment of the case and control domestic kitchens.

## MATERIALS AND METHODS

### Case definition

A person with microbiologically confirmed *Salmonella* (any serovar) infection who was not part of a recognized general outbreak, aged over 1 year and who had not travelled abroad in the 7 days prior to onset, identified in the South East Wales area (population = 1.3 million) between July 1997 and December 1998.

### Case selection

We calculated that 153 cases and controls would be required to detect an odds ratio of 2 with 95% confidence and 80% power where 30% of controls were exposed. We set a target for each local authority by calculating the expected number of cases based on the previous 4 years' experience. Collaborating local authorities were asked to provide a random sample of cases from their registers, using a supplied randomly generated list of numbers. We personally contacted every local authority every 1–2 weeks to monitor recruitment. However, occurrence of outbreaks and periods of staff shortages meant that collaborators found it difficult to always follow this regime. In order to check whether there was bias in the recruitment of cases we compared study cases with total cases of *Salmonella* infection reported to the PHLS in Wales (now the National Public Health Service Communicable Disease Surveillance Centre, Cardiff), over the study period by age, sex, location and date of onset.

### Data collection

Following informed signed consent, household details and information on kitchen cleaning, food handling and dishcloth hygiene were recorded on a standard questionnaire. The kitchen dishcloth and the lower internal surface of the refrigerator were

microbiologically analysed. Two cotton-tipped swabs, the first pre-moistened in maximum recovery diluent (MRD) (Oxoid Ltd, Basingstoke, Hants., UK; CM733) containing 0.05% sodium thiosulphate (10268, BDH, Merck Eurolab, Poole, Dorset, UK), were used consecutively to sample a 50 cm<sup>2</sup> area of the lower internal surface of each refrigerator. The swabs were placed in 10 ml of MRD. The dishcloth in use in the kitchen at the time of the visit was also collected and sealed in a sterile plastic bag. Samples were transported to the PHLS Food Microbiology Research Unit (FMRU) overnight at temperatures under 4 °C in cool boxes. Transport temperatures were recorded regularly using Testostor 175 (Testo Ltd, Alton, Staffs., UK) data loggers.

All samples were examined in the laboratory blind to the case/control status. A total of 250 ml of buffered peptone water (BPW; CM509 Oxoid Ltd) was added to each dishcloth. These samples were then homogenized in a Stomacher (Lab-blender 400, Seward Ltd, London, UK) for 1 min to ensure the release of bacteria into the liquid phase. The swabs in diluent broth were mixed for 1 min using a Vortex mixer (Jencans Miximate, Leighton Buzzard, Beds., UK). One ml of each of these was removed, diluted as appropriate in MRD, and used to obtain aerobic colony counts (using plate count agar incubated at 30 °C for 72 h) and counts of Enterobacteriaceae [using Violet Red bile glucose agar (Oxoid Ltd) incubated at 37 °C for 20–24 h].

The dishcloth and swab samples were also examined for the presence of *Salmonella*. Six ml of the swab diluent was used to inoculate 250 ml of BPW. With the dishcloth samples 200 ml of homogenate was examined for *Salmonella*.

For *Salmonella* spp. isolation, swab diluent and dishcloth homogenate were incubated overnight at 37 °C. Then 0.1 ml of these cultures was inoculated into 10 ml Rappaport–Vassiliadis soya peptone broth (RVS; CM866, Oxoid Ltd) and incubated at 41 ± 0.5 °C for 24 h. The RVS broths were streaked onto xylose, lysine, deoxycholate agar (XLD; CM469, Oxoid Ltd) and incubated at 37 °C for 18–24 h. *Salmonella*-like colonies were confirmed using standard biochemical and serological techniques. All *Salmonella* isolates were sent to the PHLS Laboratory of Enteric Pathogens (LEP) for definitive identification and phage typing.

Household demographic details and food-handling practices were recorded on standard questionnaires.

## Controls

Control households, from the same electoral ward as the case were selected by applying random numbers to the electoral register. Persons eligible to vote in the United Kingdom are legally obliged to be on the electoral register which is considered a comprehensive list of households in a given area. Only first entries for an address were included to avoid over-representation of large households. Controls were approached initially by letter and non-responders were contacted by telephone or by a personal visit. Three attempts were made to contact the first control before a further control was selected. As for cases, observations were made in the domestic kitchen. Microbiological samples and information on food-handling practices and household details were obtained in the same way as for cases. The control series was validated by comparison with census data for the study area by age of household members and household size.

When *Salmonella* was isolated from a refrigerator or dishcloth the household was informed of the result, provided with advice on kitchen hygiene and given the opportunity to have further samples submitted if they wished.

## Data analysis

Data were recorded in a Microsoft Excel spreadsheet and analysed using Epi-Info Version 6 (CDC, USA/WHO Geneva, Switzerland) and SPSS for Windows (SPSS Inc., Chicago, IL, USA).

Our first hypothesis was that salmonella were more likely to be found in case compared to control kitchens. We compared the proportion of case kitchens with salmonella to that of control kitchens by calculating odds ratios (OR) with exact 95% confidence intervals (CI) and Mantel–Haenszel  $\chi^2$  test for significance. We adjusted the odds ratio for confounders by including appropriate terms in a logistic regression model. Data were analysed unmatched to preserve all records of *Salmonella*-contaminated dishcloths.

To investigate whether the salmonella in case dishcloths was likely to be associated with the food poisoning episode or indicative of continuing unhygienic practices (i.e. fresh contamination) we compared the strains of *Salmonella* isolated from the dishcloths with the clinical isolates.

Our second hypothesis was that case kitchens were microbiologically dirtier than control kitchens. Total

aerobic colony (TAC) and Enterobacteriaceae counts for case dishcloths and refrigerator were compared to those of controls using the Mann–Whitney test for non-parametric data.

Our third hypothesis was that *Salmonella* was likely to be found in microbiologically dirty dishcloths. TAC and Enterobacteriaceae counts for *Salmonella*-positive dishcloths (in cases and control combined) were compared to those of negative dishcloths using the Mann–Whitney test for non-parametric data.

## RESULTS

### Summary data on cases and controls

All the 137 cases and 99 of 129 (77%) controls approached agreed to participate in the study.

*Salmonella* serogroups were available for 119 of the 137 isolates from the cases. These included, *S. Enteritidis* (99) including 43 phage type (PT) 4, PT6 (18), PT6a (6), PT21b (9), PT34a (3), PT5a (2) untyped (11) and one each of PT 13a, 14b, 1a, 21, 24, 29 and 34a. Fifteen of the 119 serotyped isolates were *S. Typhimurium* of which 10 were definitive type (DT) 104, two were untyped and one of DT 104b, 12 and 49 respectively. The remaining serogroups were *S. Hadar*, *S. Infantis*, *S. Oyonnax*, *S. Potsdam* and *S. Virchow*.

The mean delay between onset and interview in cases was 19.4 days (range 4–58 days, mode 14 days, median 16 days) and the period between the case and control interviews averaged 35 days (mode 7 days, median 27 days).

Recruited cases were similar to total cases in age, onset and sex distribution but a higher proportion came from the Cardiff, Vale of Glamorgan and Monmouth area. However, when data from these areas were analysed separately this did not affect the results. Control households were similar to all households in the area in distribution by age of occupants and household size.

Thirty-five per cent of cases had eaten only meals prepared in the domestic kitchen in the 72 h prior to onset of symptoms.

### Hypothesis 1: *Salmonella* are more likely to be isolated from case kitchens than control kitchens

Dishcloths were obtained from 125 case and 81 control households. *Salmonella* spp. were isolated from 12 out of 125 (10%) case dishcloths compared to four out of 81 (5%) controls (OR 2.0, exact

Table 1. Details of *Salmonella* isolates from cases, case dishcloths and control dishcloths

Case age and sex	Clinical isolate serovar and phage type	Cloth isolate serovar and phage type
42F	<i>S. Enteritidis</i> PT6	<i>S. Enteritidis</i> PT6
43F	<i>S. Enteritidis</i> PT6	<i>S. Enteritidis</i> PT6
33F	<i>S. Enteritidis</i> PT4	<i>S. Enteritidis</i> PT4
M	<i>S. Enteritidis</i> PT21b	<i>S. Enteritidis</i> PT6
F8	<i>S. Enteritidis</i> PT4	<i>S. Enteritidis</i> PT4
F	<i>S. Enteritidis</i> PT21b	<i>S. Typhimurium</i> DT104
F	<i>S. Enteritidis</i> PT4	†
F57	<i>S. Enteritidis</i> PT4	<i>S. Enteritidis</i> PT4
F56*	<i>S. Enteritidis</i> PT8	<i>S. Enteritidis</i> PT6
F5	<i>S. Enteritidis</i> PT6a	<i>S. Enteritidis</i> PT6a
M15	<i>S. Enteritidis</i> PT6	<i>S. Enteritidis</i> PT6a
M37	<i>S. Typhimurium</i> PT104b	†
Controls‡		
1	n.a.	<i>S. Enteritidis</i> PT4
2	n.a.	<i>S. Enteritidis</i> PT4
3	n.a.	<i>S. Enteritidis</i> PT6
4	n.a.	†

\* *S. Enteritidis* also isolated from a refrigerator swab.

† Serotype and phage type results unavailable.

‡ On no occasions was *Salmonella* isolated from cloths from both a case and his/her matched control.

CI 0.6–7.8, Mantel–Haenszel  $\chi^2$   $P=0.3$ ). Several potential confounding factors were examined. There were no statistically significant differences between case households with positive and negative dishcloths in delay between onset and sampling (means 17 days vs. 19 days), proportion of meals eaten in domestic kitchen (77% vs. 78%) and time of day of sampling.

Case households were more likely to have younger main food handlers ( $P<0.0001$ ) and to have been interviewed during the third quarter of the year (July, August, September) ( $P=0.004$ ). After adjusting for these confounders by logistic regression the presence of salmonella in the dishcloth remained insignificant (adjusted OR 2.2,  $P=0.6$ ).

All four controls with positive cloths had handled chicken in the previous weeks and three had handled eggs. One reported that they bleached the dishcloth every day; two said that they bleached the dishcloth weekly and one did not bleach but changed the cloth every 3 days.

In six of the 12 case households with a *Salmonella*-positive dishcloth, the dishcloth strain was the same phage type as the clinical isolate, in four a different strain was isolated and two were not serotyped or

phage typed (Table 1). In 48 case households the infected person was the main food handler and five of these submitted a *Salmonella*-positive dishcloth compared to seven of the 73 households where the main food handler had not suffered food poisoning (5/48 vs. 7/73,  $P=0.8$ ). In 106 case households the infected person was an adult and 10 of these submitted a *Salmonella*-positive dishcloth compared to two of the 19 households where the infected person was a child (10/106 vs. 2/19,  $P=0.7$ ).

*Salmonella* was isolated from one out of 137 (0.7%) case refrigerators compared to three out of 96 (3%) control refrigerators (OR 0.7, exact CI 0.1–7.1, Fisher's exact  $P=0.98$ ). The case with the *Salmonella*-positive refrigerator swab also submitted a positive dishcloth but the refrigerator swab was *S. Enteritidis* PT4 and the dishcloth isolate was *S. Enteritidis* PT6. One of the controls who submitted a positive dishcloth also submitted a positive refrigerator swab with PT6 in both samples. One of these three controls had handled chicken in the previous 7 days and had stored it in the refrigerator.

### Hypothesis 2: Higher colony counts are recorded in case kitchens compared to control kitchens

Most dishcloths were heavily contaminated and TAC counts frequently exceeded  $10^{10}$  colony forming units (c.f.u.) per cloth. The counts of Enterobacteriaceae were also generally high, although some cloths were found to have undetectable levels ( $<1250$  c.f.u. per cloth). Bacterial counts from refrigerators were generally lower; 93 of 137 case swabs and 52 of 96 control swabs had counts of  $\leq 5$  c.f.u.

The bacterial counts of dishcloths from case kitchens were not significantly different from those in control homes and swab counts from case refrigerators were not significantly different from that recorded in controls (Table 2).

### Hypothesis 3: Dishcloths and refrigerators with higher colony counts are more likely to be contaminated with *Salmonella*

The Enterobacteriaceae counts (Fig.) (Mann–Whitney  $P=0.78$ ) and TAC counts (Mann–Whitney  $P=0.40$ ) were similar for *Salmonella*-positive and *Salmonella*-negative dishcloths.

Three of the four contaminated refrigerators swabs had Enterobacteriaceae counts of  $<5$  c.f.u., the remaining count was  $3.0 \times 10^6$ . Two of the four

Table 2. Comparison of dishcloth and refrigerator swab *Enterobacteriaceae* counts and total aerobic colony counts from cases and controls

Variable	Case/ Control	Obs	Minimum (c.f.u.)	Maximum (c.f.u.)	Median	Mann–Whitney <i>P</i> value
<b>Dishcloths</b>						
Enterobacteriaceae counts	Case	125	<1250	$1.5 \times 10^{11}$	$1.2 \times 10^7$	0.48
	Control	81	<1250	$8.1 \times 10^{10}$	$4.6 \times 10^7$	
Aerobic colony count	Case	125	<1250	$6.3 \times 10^{11}$	$3.15 \times 10^9$	0.88
	Control	81	<1250	$5.0 \times 10^{11}$	$1.40 \times 10^9$	
<b>Refrigerator swabs</b>						
Enterobacteriaceae counts	Case	125	<5	$2.8 \times 10^5$	5	0.47
	Control	81	<5	$3.2 \times 10^6$	5	
Aerobic colony count	Case	125	<5	$1.6 \times 10^7$	1150	0.14
	Control	81	<5	$5.5 \times 10^6$	2200	

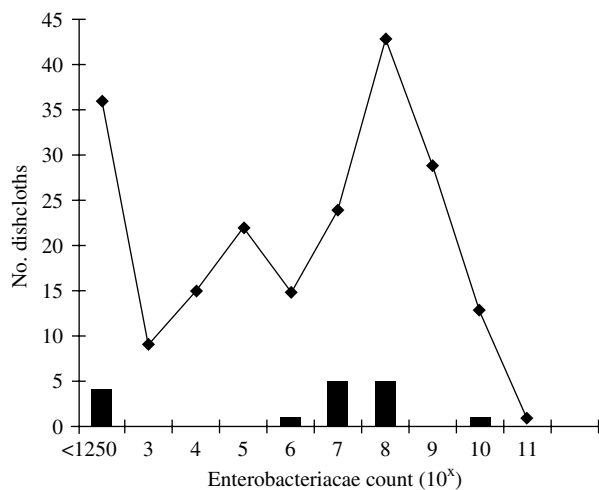


Fig. Dishcloth Enterobacteriaceae counts (—◆—) and *Salmonella* isolations (■).

contaminated swabs had TAC counts of <5, one was 200 and one was  $3.0 \times 10^6$  c.f.u.

## DISCUSSION

The sources of individual sporadic *Salmonella* infections are rarely identified. Food samples are usually not available and the food history of one individual is often impossible to interpret. It is widely assumed that a significant proportion of these cases are acquired from food prepared in the domestic kitchen although food eaten outside the home and non-food sources cannot be ruled out [1–3].

Experimental studies suggest that the conditions necessary for foodborne transmission to occur can be found in domestic kitchens. We know that recognized

sources of *Salmonella* infection (including poultry meat and eggs) are frequently handled in domestic kitchens and that contamination can be widely disseminated within the kitchen environment under experimental conditions [4–6].

The key to developing preventative strategies, however, is to identify how kitchens used by a case of *Salmonella* are different to those used by the remainder of the population. Our work has compared case and control kitchens in terms of foods handled, food hygiene precautions and microbiology.

We have previously reported that we did not identify food-handling risk factors for sporadic *Salmonella* infection in domestic kitchens [3]. The large study of infectious intestinal disease in England also found that domestic kitchen practices had little effect on the risk of acquiring an infectious intestinal disease [9]. Nevertheless, we would still expect that case kitchens would be more likely to be contaminated with *Salmonella* than controls if infection was acquired from home-prepared food.

Commonly, dishcloths are used to wipe kitchen surfaces and should be a good indicator of kitchen contamination. However, we could not demonstrate that the presence of *Salmonella* in the dishcloth was a good indicator of risk of *Salmonella* infection from a kitchen. We did isolate *Salmonella* from both case and control dishcloths under normal domestic kitchen conditions. Case and control samples were transported identically, in the same insulated boxes and were examined blind. Case dishcloths (10%) were twice as likely to be contaminated as controls (5%) but this difference did not reach statistical significance. In six of the 10 case households with serotype

and phage-type information available the dishcloth isolate was the same as the clinical isolate. These cases could, in theory, have contaminated the cloth after their symptoms had started. However, case households where the infected person had most opportunities to handle the cloth (i.e. was the main food handler and an adult) were not more likely to have a positive cloth when compared to other case households. An alternative explanation is that the *Salmonella* infection originated in the kitchen and salmonella remained in the dishcloth following the initial contamination. In all six case households with the same clinical and dishcloths strain, interviewees reported that the cloth had been changed or bleached between the onset of symptoms in the index case and sampling. However, widespread contamination of the kitchen may have occurred with the new dishcloth picking up the bacteria as it was used. In four kitchens different strains of *Salmonella* were isolated from the case and from the household kitchen dishcloth suggesting new contamination. We also found four positive (5%) control dishcloths suggesting background level of contamination; although the numbers of positive dishcloths are too few to draw firm conclusions.

Our controls had been questioned about which foods had been handled in the kitchen in the 7 days prior to the cloth being sampled (cases were asked about the period prior to onset which was several weeks previously). We examined the food-handling data for the four controls with contaminated cloths for potential sources of *Salmonella*. Chicken had been handled in all four control kitchens and eggs in three of the four. These represent the most likely sources of contamination. Two controls had handled fresh chicken and two had handled frozen chicken. All four households adopted reasonable hygienic precautions in the cleaning of the dishcloths. One did not bleach the cloth but changed it every 3 days and the remainder bleached the cloth daily or weekly. All four food handlers reported that they washed their hands with soap after handling raw poultry although we observed hand-washing soap in only two of the kitchens. None reported using an antibacterial cleaning agent on kitchen work surfaces. In all four kitchens, reasonable food safety precautions had failed to stop *Salmonella* contaminating the dishcloth.

We report similar findings for refrigerator microbiology. *Salmonella* spp. was isolated from the internal surface of both case and control refrigerators but there was no difference in the proportions of

refrigerators which were positive. In one case and one control both the refrigerators and the dishcloth were contaminated.

We conclude that there is no convincing evidence that kitchens which are contaminated with *Salmonella* are more likely to give rise to a case of *Salmonella* infection than those which are not contaminated.

It is often assumed that *Salmonella* infection occurs in homes which are unhygienic and have higher bacteria levels and our second hypothesis was that higher colony counts are recorded in case kitchens compared to control kitchens. The distributions of Enterobacteriaceae counts and aerobic colony counts were similar in case and control dishcloths and case and control refrigerators. It was not surprising that many dishcloths were heavily contaminated with counts of  $10^{11}$ , as the dishcloth provides an ideal moist environment for bacterial multiplication [7, 8] but some dishcloths also had undetectable levels ( $< 1250$ ). Generally, bacterial levels on the internal surface of the refrigerator were lower, with many  $< 5$  c.f.u.

There was some evidence that counts recorded in the summer months were higher than in winter and higher ambient room temperatures would explain this. However, counts did not vary with the time of day of sampling.

Our case group was broadly representative of all reported *Salmonella* infections meeting our case definition over the study period, although it is accepted that reported cases are only a small proportion of all cases in the community. Our response rate for our controls, 77%, was high considering the fairly intrusive nature of the inquiry. Interviews were carried out on pre-arranged days of the week to coordinate with the transport of samples to the laboratory. The majority of cases and controls were, therefore, visited by making a prior appointment affording equal opportunity to clean the kitchen and change or bleach the dishcloth. Most case/control pairs were interviewed by the same Environmental Health Officer.

Household kitchens with dirty (in terms of bacteriological quality) dishcloths and refrigerators are not more likely to give rise to an episode of *Salmonella* infection than clean kitchens. We have previously reported [3] that the presence of visible dirt in the kitchen refrigerator was not a risk factor for sporadic *Salmonella* infection.

The third hypothesis was that dishcloths and refrigerators with higher colony counts are more likely to be contaminated with *Salmonella*. It is widely

believed that poor kitchen hygiene results in dirtier dishcloths and refrigerators and that this in turn increases the chances of contamination with pathogens. Our results suggest that this is not the case. We isolated salmonella from dishcloths with relatively low total counts and from refrigerators with < 5 c.f.u. We found TAC counts and Enteriobacteriaceae counts to be poor predictors of *Salmonella* contamination in a kitchen environment.

In conclusion, our studies to date have found no evidence that hygienic practices or food-handling patterns differ between the households of cases of *Salmonella* infection and controls [3]. Here, we have found no evidence that kitchens in households where a case of *Salmonella* had occurred were more likely to be contaminated with the bacterium or to be 'dirtier' in terms of total bacterial counts. We also found no evidence that 'dirtier' kitchens are more likely to harbour *Salmonella*.

Clearly, the presence of *Salmonella* in the domestic kitchen is necessary but not sufficient to result in transmission and an episode of foodborne illness. It is possible that there are further risk factors that could not be identified by a study of this type which increase the chances of transmission occurring. We did question the main food handler in detail about food purchasing practices, food handling, temperature control and opportunities for cross contamination and made observations in the domestic kitchen but found no differences between cases and controls [3]. We did not focus on host-specific risk factors such as recent treatment with H<sub>2</sub> antagonists, antibiotics and gastric surgery [10], which have been shown by previous studies to predispose individuals to *Salmonella* food poisoning, and these factors may be more important in the community than previously acknowledged. Alternatively, the assumption that the vehicle for infection is usually food consumed in the home may be wrong. Sixty-five per cent of our cases, including all of the cases with *Salmonella* isolated from the kitchen had eaten meals outside the home in the 72 h prior to onset of symptoms.

Further studies focusing on individuals who had only eaten at home and recording factors which may

make some individuals more vulnerable to infection would advance this important area of research.

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