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A 17-year review of foodborne outbreaks: describing the continuing decline in England and Wales (1992–2008)

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SUMMARY

Systematic national surveillance of foodborne disease outbreaks effectively serves the development of public health policy on food safety. The Health Protection Agency has maintained a collaborative surveillance system for foodborne outbreaks in England and Wales since 1992. Up to 2008, 2429 foodborne outbreaks were identified, described and analysed for changes over time. *Salmonella* spp. accounted for half of the outbreaks, although the proportion of these decreased over the surveillance period. Similarly, the proportion of outbreaks caused by *Clostridium perfringens* decreased, while those attributed to *Campylobacter* spp. and Vero cytotoxin-producing *Escherichia coli* O157 increased. Although poultry meat was the most frequently implicated food vehicle in outbreaks followed by miscellaneous foods and red meats, the proportion of outbreaks attributed to meats in fact decreased over time but those linked to miscellaneous foods did not. Over the surveillance period, the proportion of outbreaks linked to eggs and *S.* Enteritidis non-phage-type 4, particularly in food service establishments, increased, highlighting the importance of this organism/setting/vehicle association. Contributory factors in most outbreaks were cross-contamination, inadequate heat treatment, and inappropriate food storage. This study describes the overall decline in foodborne outbreaks, providing evidence that the introduction and adherence to effective control measures provide the best means of minimizing the risk of foodborne infection.

Key words: Food poisoning, food safety, foodborne infections, outbreaks.

INTRODUCTION

Infectious intestinal disease (IID) acquired through consumption of food represents a significant health and financial burden to the UK. In 2007, the Health Protection Agency (HPA) estimated it as 926 000 cases, with 18 900 hospitalizations and 440 deaths [1]. Foodborne disease (outbreaks and sporadic infections) has cost about £1·5 billion per year in England and Wales from 2005 [1]. In the UK, reducing foodborne illness has been a key target in the Food Standards Agency's (FSA) strategy on foodborne disease since its inception in 2000, and continues to be a focus in its new strategic plan for 2010–2015, where one of the five outcomes that the FSA aims to deliver is that 'food produced or sold in the UK is safe to eat' [2].

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Human foodborne illness has remained a persistent problem because of the tremendous complexity and dynamic nature of foods. Such complexities include the diversity of microorganisms that cause a wide range of human health outcomes, the vast array of foods that serve as vehicles for human infection, and the extensive causative and contributing factors that affect contamination, growth, and persistence of the microorganisms throughout the food chain. The HPA's surveillance system for general outbreaks of IID in England and Wales has been in operation since 1992 [3]. This system allows a more reliable evaluation of the contribution of different pathogens, foods, and settings than the biased sample represented by published investigations [4]. The type of evidence leading to the suspicion of the food vehicle is also documented, thereby allowing a distinction to be made between credibly identified food vehicles, and food vehicles assumed on the basis of, for example, biological plausibility. The European Food Safety Authority (EFSA) has more recently developed a classification system for foodborne outbreaks for the harmonized reporting of foodborne outbreaks through the community reporting system [5]. Under this classification 'verified foodborne outbreaks' are defined as those where 'the link between human cases and a food vehicle is supported by the detection of the causative agent in the implicated food vehicle and/or by analytical epidemiological evidence providing a statistically significant association between the food vehicle and human cases'. 'Possible foodborne outbreaks' are those 'compatible with descriptive evidence alone including those where the causative agent is unknown' [6].

Attributing food sources to human disease is recognized as increasingly useful in food-safety risk analysis in that reliable and accurate information provided to policy makers can be used to make well-informed decisions about their policies and advice, and evaluate their impact. Analysis of data from outbreak investigations for attributing human foodborne disease has been previously described [7–10] and such analysis has proven to be a valuable means of identifying novel food vehicles of infection as well as confirming the continued role of known vehicles and pathogens in current food-safety problems, such as *Salmonella* and *Campylobacter* infections [7, 11–14]. The existence of comprehensive foodborne outbreak data in England and Wales collected for almost two decades provided the opportunity to give an overview of foodborne outbreaks between 1992 and 2008, to track trends in foodborne disease against interventions that have taken place, and to explore the usefulness of the data for estimating source attribution of foodborne infections.

METHODS

The surveillance system for general outbreaks of IID in England and Wales

The HPA's surveillance system of general outbreaks of IID in England and Wales commenced in 1992 [3]. General outbreaks are those affecting members of more than one household or residents of an institution. Upon notification of an outbreak (from a variety of sources including local health protection teams, environmental health practitioners, reference laboratory microbiologists, food examiners, and government) a standardized surveillance form is sent to the lead investigator [usually a consultant in communicable disease control (CCDC)] with a request that it is completed once the outbreak investigation has ended. Designed to elicit a standard response, the questionnaire seeks to capture information about the setting of the outbreak (in foodborne disease outbreaks defined as the place where food was prepared), the mode of transmission, causative organism(s) and the results of epidemiological and environmental investigations. Up to three reminders are sent to non-responders and the response rate is consistently over 80% [15]. Upon receipt, the information is entered onto a bespoke database and the outbreak classified as either a verified or possible foodborne outbreak as defined by EFSA [6]. In some instances, e.g. the six outbreaks caused by *L. monocytogenes*, questionnaires were retrospectively completed following receipt of outbreak reports/papers.

Data abstraction and analysis

For the purpose of this study, outbreaks of foodborne origin were selected (a proportion of foodborne outbreaks are followed by person-to-person transmission). A foodborne outbreak is defined by European legislation as 'an incidence, observed under given circumstances, of two or more human cases of the same disease and/or infection, or a situation in which the observed number of human cases exceeds the expected number and where the cases are linked, or are probably linked, to the same food source' [5]. As defined by the European General Food Law Regulation [Regulation (EC) No. 178/2002], potable water is now classed as a 'food' and was therefore

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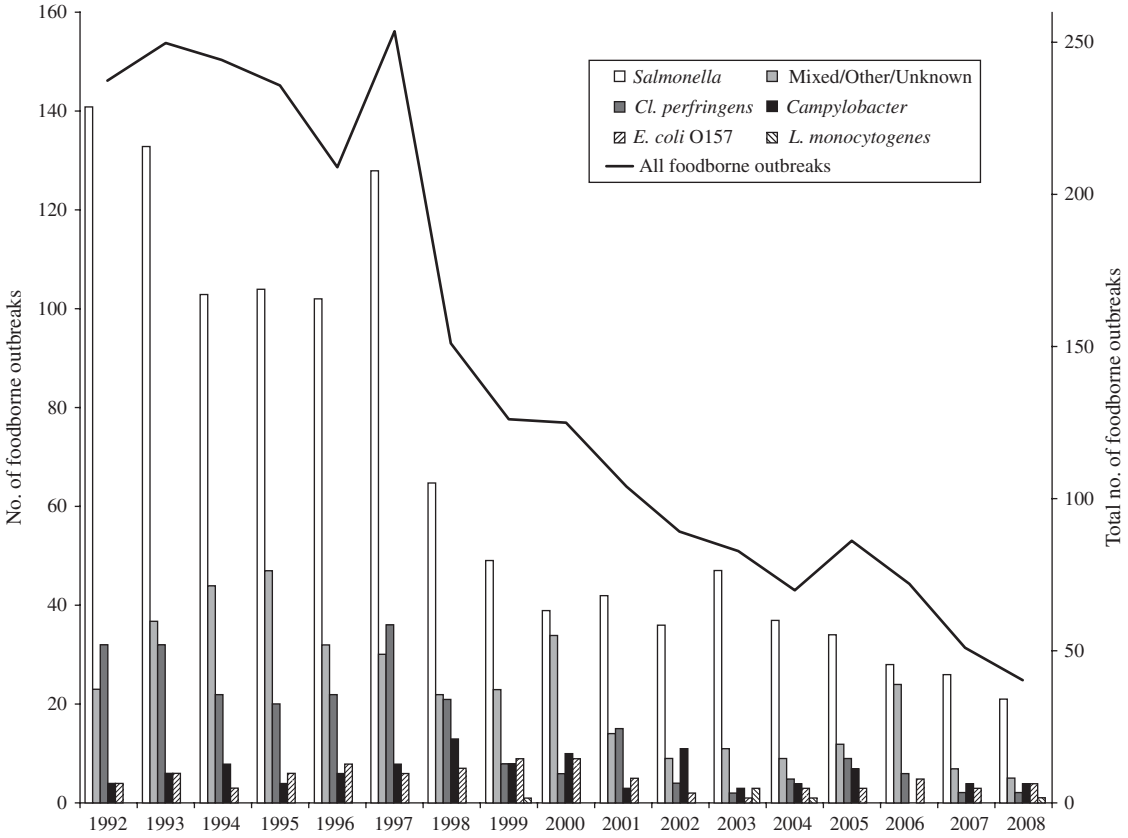

Fig. 1. Number of foodborne outbreaks in England & Wales (1992–2008) stratified by causative pathogen. Overall annual numbers of foodborne outbreaks are also shown.

included in the analyses. Household outbreaks were not included although private establishments, defined as private functions involving more than one household were. These data were extracted into Microsoft Excel to facilitate analysis.

Data analysis was carried out using Microsoft Excel and Stata version 10 (Stata Corporation, USA). The change in relative proportions over time were described using the χ^2 test for trend, relative proportions compared using the χ^2 test, and for smaller sample sizes Fisher's exact test was used. Univariate analysis was used to describe associations between the pathogen of interest (outcome) with food and outbreak setting as variables (exposures).

RESULTS

Between 1992 and 2008, 10 966 general outbreaks of IID were reported to the HPA, of which 2429 (22·2%) were foodborne. Over the surveillance period, the number of foodborne outbreaks progressively declined from 238 in 1992 to 40 in 2008, with the exception of 1997 and 2005 (Fig. 1).

Severity of causative agent

From 2429 foodborne outbreaks, a total of 58 424 individuals were affected (range 2–575, mean 24), 2141 were hospitalized (range 0–65, mean 1) and 127 died (range 0–13, mean 0·1). *Salmonella* spp. were responsible for the highest number of people affected ($n=27\,339$), hospitalizations ($n=1500$), and deaths ($n=97$) (Table 1). However, the mean number of people affected was highest for *Cryptosporidium* outbreaks ($n=101$). As a proportion of the total number of people affected, most hospitalizations were incurred through Vero cytotoxin-producing *Escherichia coli* (VTEC) O157 infection (286/1168, 24·5%), while the highest mortality rates were observed for *Listeria monocytogenes* (2/33, 6·1%).

Outbreak duration

The dates of onset of the first and last cases were reported in 82·5% (2003/2429) of outbreaks. The duration of these outbreaks ranged from a single day to 373 days, with a mean of 10 days; however, those caused by VTEC O157 (mean 16·9 days), *Salmonella*

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Table 1. *Foodborne outbreak setting in England and Wales, morbidity by pathogen*

Causative agent	No. of outbreaks	Affected				Hospitalized				Deaths			
		Total	Mean	Min	Max	Total	Mean	Min	Max	Total	Mean	Min	Max
Salmonella spp.	1135	27 339	24·1	2	530	1500	1·8	0	65	97	0·13	0	13
Mixed/other/unknown	383	8300	21·7	2	426	56	0·2	0	21	2	0·007	0	1
Viruses	263	8745	33·3	2	200	32	0·2	0	6	3	0·02	0	2
Cl. perfringens	244	5559	23·0	2	400	31	0·3	0	16	10	0·09	0	2
Campylobacter	103	2331	22·6	2	281	18	0·3	0	4	0	0	0	0
VTEC O157	84	1168	13·9	2	114	286	3·7	0	28	12	0·18	0	3
Bacillus spp.	69	588	8·5	2	106	4	0·2	0	2	0	0	0	0
Scombrotoxin	66	318	4·8	2	46	62	1·5	0	10	0	0	0	0
Staph. aureus	35	505	14·9	2	125	32	2·1	0	13	0	0	0	0
Cryptosporidium	31	3115	100·5	9	575	105	5·5	0	26	1	0·06	0	1
Shigella spp.	10	423	42·3	9	171	12	1·3	0	5	0	0	0	0
L. monocytogenes	6	33	5·5	2	17	3	3·0	0	3	2	0	2	2
Total	2429	58 424	24·1	2	575	2141	1·3	0	65	127	0·1	0	13

VTEC, Vero cytotoxin-producing *Escherichia coli* O157.

spp. (mean 12·0 days), and *Shigella* spp. (mean 11·9 days) were more prolonged.

Causative agent

Salmonella spp. were in the main implicated in almost half of all foodborne outbreaks (1135/2429, 46·7%). Of all *Salmonella* serotypes, *Salmonella enterica* serovar Enteritidis phage-type (PT) 4 was the most common, accounting for 50·9% (578/1135), *S.* Enteritidis non-PT4 accounted for 26·2% (297/1135), *S.* Typhimurium for 12·8% (145/1135), and other salmonellas for the remaining 10·1% (115/1135). The proportion of *Salmonella* spp. outbreaks collectively decreased significantly over the surveillance period ($P=0·0075$) (Fig. 1). Specifically, the proportion of *S.* Enteritidis PT4 followed this trend ($P<0·0001$) whereas those attributed to *S.* Enteritidis non-PT4 increased (mainly PT1 and PT14b) ($P<0·0001$). In addition to *S.* Enteritidis PT4 outbreaks, the proportion of outbreaks attributed to *Clostridium perfringens* also decreased significantly ($P=0·0021$). In contrast, the proportion of outbreaks attributed to *Campylobacter* ($P<0·001$) and VTEC O157 ($P<0·007$) increased over the surveillance period while no trend was observed for those attributed to viral pathogens (mainly norovirus), *Cryptosporidium* and unknown agents ($P>0·05$).

Evidence implicating food vehicles

In 1193/2429 (49·1%) outbreaks one type of evidence was provided to implicate a food vehicle, in 247 (10·2%) there was more than one, and in 989 (40·7%) none. There was analytical epidemiology plus microbiological evidence (laboratory detection of the causative pathogen or toxin in food taken in the course of the investigation) in 3·4% (83/2429), microbiological evidence alone in 10·1% (246), analytical epidemiology alone in 17·2% (419), descriptive epidemiology plus microbiological evidence in 3·4% (82) and descriptive epidemiology alone in 25·1% (610). The proportion in which both analytical and microbiological evidence or microbiological evidence alone were reported remained unchanged during the surveillance study ($P=0·344$). However, analytical evidence reported alone decreased up to 2005 (15·0% to 3·0%, $P<0·0001$), but was then followed by an increase up to 2008 (15·0%). In contrast, the proportion in which descriptive evidence was reported increased (18·0–22·5%, $P=0·00026$), as did that with microbiological and descriptive evidence (1·0–2·5%, $P=0·00074$). On the basis of the evidence provided, 57·6% (830) and 42·4% (610) of these outbreaks would be classified as verified and possible outbreaks, respectively, for the purposes of reporting on foodborne outbreaks to EFSA.

Implicated food vehicles

At least one food vehicle was identified in 75·6% (1836/2429) of all foodborne outbreaks. Poultry meat was most frequently implicated, accounting for 19·1% (350/1836), with miscellaneous foods (17·6%), red meat (15·7%) and fish and shellfish (12·4%) being among the next most commonly identified (Table 2).

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Table 2. *Foodborne outbreaks recorded in England and Wales from 1992 to 2008 showing implicated food vehicle by year*

	Implicated food vehicle, *n* (%)											
Year	Poultry meat	Red meat	Fish/ shellfish	Salad vegetables/ fruit	Sauce	Dessert	Milk/milk products	Water	Miscellaneous*	Eggs	Rice	Total
1992	45 (20·1)	45 (20·1)	20 (8·9)	16 (7·1)	4 (1·8)	25 (11·2)	5 (2·2)	6 (2·7)	41 (18·3)	12 (5·4)	5 (2·2)	224
1993	35 (21·2)	40 (24·2)	13 (7·9)	7 (4·2)	5 (3)	26 (15·8)	4 (2·4)	7 (4·2)	20 (12·1)	8 (4·8)	0 (0)	165
1994	21 (12·9)	33 (20·2)	21 (12·9)	18 (11)	4 (2·5)	21 (12·9)	5 (3·1)	5 (3·1)	19 (11·7)	10 (6·1)	6 (3·7)	163
1995	33 (17·9)	33 (17·9)	26 (14·1)	5 (2·7)	6 (3·3)	22 (12)	6 (3·3)	1 (0·5)	34 (18·5)	10 (5·4)	8 (4·3)	184
1996	42 (22·8)	24 (13)	22 (12)	20 (10·9)	3 (1·6)	23 (12·5)	5 (2·7)	3 (1·6)	21 (11·4)	11 (6)	10 (5·4)	184
1997	46 (22·2)	28 (13·5)	32 (15·5)	5 (2·4)	12 (5·8)	30 (14·5)	7 (3·4)	5 (2·4)	22 (10·6)	13 (6·3)	7 (3·4)	207
1998	22 (19)	16 (13·8)	12 (10·3)	7 (6)	7 (6)	16 (13·8)	3 (2·6)	4 (3·4)	21 (18·1)	6 (5·2)	2 (1·7)	116
1999	22 (23·2)	13 (13·7)	12 (12·6)	8 (8·4)	3 (3·2)	7 (7·4)	6 (6·3)	3 (3·2)	17 (17·9)	4 (4·2)	0 (0)	95
2000	15 (17)	14 (15·9)	11 (12·5)	10 (11·4)	1 (1·1)	9 (10·2)	3 (3·4)	2 (2·3)	21 (23·9)	0 (0)	2 (2·3)	88
2001	21 (24·4)	16 (18·6)	9 (10·5)	6 (7)	1 (1·2)	3 (3·5)	0 (0)	0 (0)	22 (25·6)	5 (5·8)	3 (3·5)	86
2002	10 (16·7)	4 (6·7)	2 (3·3)	4 (6·7)	3 (5)	6 (10)	3 (5)	5 (8·3)	13 (21·7)	8 (13·3)	2 (3·3)	60
2003	6 (10)	3 (5)	2 (3·3)	6 (10)	0 (0)	4 (6·7)	2 (3·3)	1 (1·7)	21 (35)	13 (21·7)	2 (3·3)	60
2004	12 (24)	6 (12)	8 (16)	3 (6)	1 (2)	5 (10)	0 (0)	0 (0)	8 (16)	4 (8)	3 (6)	50
2005	5 (10·4)	6 (12·5)	17 (35·4)	3 (6·3)	1 (2·1)	5 (10·4)	0 (0)	2 (4·2)	8 (16·7)	0 (0)	1 (2·1)	48
2006	4 (9·3)	4 (9·3)	12 (27·9)	1 (2·3)	2 (4·7)	3 (7)	0 (0)	0 (0)	11 (25·6)	5 (11·6)	1 (2·3)	43
2007	9 (22·5)	3 (7·5)	7 (17·5)	1 (2·5)	0 (0)	0 (0)	0 (0)	0 (0)	14 (35)	5 (12·5)	1 (2·5)	40
2008	2 (8·7)	1 (4·3)	2 (8·7)	2 (8·7)	0 (0)	2 (8·7)	0 (0)	1 (4·3)	11 (47·8)	2 (8·7)	0 (0)	23
Total	350 (19·1)	289 (15·7)	228 (12·4)	122 (6·6)	53 (2·9)	207 (11·3)	49 (2·7)	45 (2·5)	324 (17·6)	116 (6·3)	53 (2·9)	1836

* Miscellaneous food includes buffet foods, sandwiches and other dishes comprising multiple ingredients.

Table 3. *Foodborne outbreaks recorded in England and Wales from 1992 to 2008 showing causative agent by implicated food vehicle*

	Implicated food vehicle, *n* (%)										
Causative agent	Poultry meat	Miscellaneous*	Red meat	Fish/ shellfish	Desserts	Salad vegetables/ fruit	Eggs	Rice	Sauces	Milk/milk products	Water	Total
Bacillus spp.	14 (17·3)	19 (23·5)	9 (11·1)	3 (3·7)	2 (2·5)	5 (6·2)	0 (0)	28 (34·6)	1 (1·2)	0 (0)	0 (0)	81
Campylobacter spp.	30 (38)	13 (16·5)	4 (5·1)	1 (1·3)	0 (0)	6 (7·6)	0 (0)	0 (0)	2 (2·5)	8 (10·1)	15 (19)	79
Cl. perfringens	69 (29·7)	19 (8·2)	117 (50·4)	2 (0·9)	3 (1·3)	9 (3·9)	0 (0)	4 (1·7)	9 (3·9)	0 (0)	0 (0)	232
Cryptosporidium	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	1 (3·4)	28 (96·6)	29
VTEC O157	3 (6·8)	7 (15·9)	17 (38·6)	0 (0)	1 (2·3)	3 (6·8)	0 (0)	0 (0)	0 (0)	13 (29·5)	0 (0)	44
L. monocytogenes	0 (0)	4 (80)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	1 (20)	0 (0)	5
Staph. aureus	13 (33·3)	8 (20·5)	11 (28·2)	1 (2·6)	0 (0)	1 (2·6)	1 (2·6)	3 (7·7)	1 (2·6)	0 (0)	0 (0)	39
Scombrotoxin	0 (0)	4 (6·2)	0 (0)	61 (93·8)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	65
Shigella spp.	0 (0)	2 (33·3)	0 (0)	0 (0)	1 (16·7)	3 (50)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	6
Viruses	12 (10·1)	32 (26·9)	7 (5·9)	41 (34·5)	7 (5·9)	16 (13·4)	0 (0)	0 (0)	2 (1·7)	2 (1·7)	0 (0)	119
Salmonella spp.	190 (20·5)	183 (19·7)	100 (10·8)	41 (4·4)	186 (20)	46 (5)	114 (12·3)	12 (1·3)	35 (3·8)	21 (2·3)	0 (0)	928
Mixed/other/unknown	19 (9·1)	33 (15·8)	24 (11·5)	78 (37·3)	7 (3·3)	33 (15·8)	1 (0·5)	6 (2·9)	3 (1·4)	3 (1·4)	2 (1)	209
Total	350 (19·1)	324 (17·6)	289 (15·7)	228 (12·4)	207 (11·3)	122 (6·6)	116 (6·3)	53 (2·9)	53 (2·9)	49 (2·7)	45 (2·5)	1836

VTEC, Vero cytotoxin-producing *Escherichia coli* O157.
* Miscellaneous food includes buffet foods, sandwiches and other dishes comprising multiple ingredients.

Desserts were implicated in 11·3% of foodborne outbreaks, with raw shell egg used as an ingredient in 70·5% of these (146/207).

The proportion of foodborne outbreaks associated with poultry meat, red meat and desserts decreased significantly over the 17 years ($P=0·02$, $P<0·001$ and $P=0·003$, respectively), while those linked with eggs and miscellaneous foods increased ($P=0·04$ and $P=0·0002$, respectively) (Table 2). The proportion of outbreaks linked to fish and shellfish did not significantly change ($P>0·05$).

Over half (54·2%) of poultry meat-linked outbreaks were attributed to *Salmonella* spp. as were 56·5% of outbreaks linked with miscellaneous foods (Table 3). *Salmonella* outbreaks were strongly associated with consumption of desserts [89·9%; odds ratio (OR) 11·9, 95% confidence interval (CI) 7·4–19·1] and eggs (98·3%; OR 72·1, 95% CI 17·1–304·4) (Tables 3 and 5). Specifically, *S.* Enteritidis PT4 accounted for 58·6% of the 116 egg-associated outbreaks and *S.* Enteritidis non-PT4 for 35·3%.

Cl. perfringens outbreaks were more strongly associated with consumption of red meats (40·5%; OR 10·8, 95% CI 7·8–14·8) compared to other implicated food vehicles, as were VTEC O157 outbreaks (5·9%; OR 1·9, 95% CI 1·1–3·3), with the latter also being strongly associated with milk and milk product consumption (26·5%; OR 11·7, 95% CI 5·9–23·4) (Tables 3 and 5). *Campylobacter* outbreaks were strongly associated with consumption of poultry meat (38·0%; OR 2·6, 95% CI 1·7–4·0), but significant associations with potable water (33·5%; OR 13·0, 95% CI 6·7–25·5) and milk or milk products (16·5%; OR 4·7, 95% CI 2·1–10·3) were also observed (Tables 3 and 5).

Outbreak setting

Foodborne outbreaks were more likely to take place in food service establishments (52·6%, 1279/2429) (Table 4), with restaurants and hotels accounting for 67·6% (864/1279) of these. The proportion of foodborne outbreaks linked to food service establishments increased significantly during the surveillance period ($P<0·0001$), whereas those linked to residential homes and private establishments decreased significantly ($P=0·01$ and $P<0·0001$, respectively). The proportion of outbreaks associated with food retailers remained relatively unchanged.

Salmonella spp. were most commonly implicated in outbreaks linked to food service (42·5%),

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Table 4. *Foodborne outbreaks recorded in England and Wales from 1992–2008 showing outbreak setting by causative agent*

Causative agent	Outbreak setting, *n* (%)												
	Food service establishments	Club/ centre	Community	Hospital	Other*	Private	Residential establishments	School	University/ college	Work place	Food retailer	Farm shop	Total
Salmonella spp.	544 (47·9)	28 (2·5)	33 (2·9)	25 (2·2)	16 (1·4)	157 (13·8)	168 (14·8)	46 (4·1)	10 (0·9)	2 (0·2)	96 (8·5)	10 (0·9)	1135
Mixed/other/ unknown	253 (66·1)	19 (5)	1 (0·3)	13 (3·4)	6 (1·6)	22 (5·7)	45 (11·7)	13 (3·4)	4 (1)	5 (1·3)	2 (0·5)	0 (0)	383
Viruses	150 (57)	21 (8)	0 (0)	14 (5·3)	1 (0·4)	13 (4·9)	44 (16·7)	7 (2·7)	6 (2·3)	2 (0·8)	4 (1·5)	1 (0·4)	263
Cl. perfringens	115 (47·1)	12 (4·9)	3 (1·2)	10 (4·1)	9 (3·7)	11 (4·5)	74 (30·3)	6 (2·5)	0	1 (0·4)	3 (1·2)	0	244
Campylobacter spp.	61 (59·2)	5 (4·9)	1 (1)	1 (1)	0	4 (3·9)	17 (16·5)	3 (2·9)	2 (1·9)	1 (1)	0	8 (7·8)	103
VTEC O157	19 (22·6)	0	10 (11·9)	0	3 (3·6)	8 (9·5)	12 (14·3)	6 (7·1)	0	0	15 (17·9)	11 (13·1)	84
Bacillus spp.	58 (84·1)	3 (4·3)	0	0	2 (2·9)	2 (2·9)	2 (2·9)	0	0	0	2 (2·9)	0	69
Scombrotoxin	52 (78·8)	4 (6·1)	0	1 (1·5)	1 (1·5)	2 (3)	1 (1·5)	1 (1·5)	0	0	4 (6·1)	0	66
Staph. aureus	22 (62·9)	2 (5·7)	1 (2·9)	1 (2·9)	1 (2·9)	3 (8·6)	2 (5·7)	2 (5·7)	0	0	1 (2·9)	0	35
Cryptosporidium	0 (0)	1 (3·2)	23 (74·2)	0	2 (6·5)	1 (3·2)	1 (3·2)	1 (3·2)	0	0	0	2 (6·5)	31
Shigella spp.	5 (50)	0	1 (10)	0	0	0	0 (0)	2 (20)	0	0	2 (20)	0	10
L. monocytogenes	0 (0)	0	0	5 (83·3)	1 (16·7)	0	0 (0)	0	0	0	0	0	6
Total	1279 (52·7)	95 (3·9)	73 (3)	70 (2·9)	42 (1·7)	223 (9·2)	366 (15·1)	87 (3·6)	22 (0·9)	11 (0·5)	129 (5·3)	32 (1·3)	2429

VTEC, Vero cytotoxin-producing *Escherichia coli* O157.
* Other settings include singletons such as rugby club, funeral, bowling alley and outbreaks where the setting was not specified.

residential (45·5%) and private (70·4%) establishments (Table 4). Most *Cl. perfringens* outbreaks occurred in food service (47·1%) and residential establishments (30·3%) (Table 4) and *Cl. perfringens* outbreaks were three times more likely to occur in residential establishments compared to those caused by other pathogens (OR 2·8, 95% CI 2·1–3·8) (Table 5). Although implicated in outbreaks at various settings, outbreaks attributed to VTEC O157 were significantly more likely to be linked to farm shops (OR 16·7, 95% CI 7·6–36·4) and food retailers (OR 4·3, 95% CI 2·4–7·7) than other pathogens (Tables 4 and 5). All but one of the six reported *L. monocytogenes* outbreaks (83·3%) occurred in hospitals (Table 4), and were linked to consumption of pre-packed sandwiches [16]. Outbreaks caused by viral pathogens (mainly norovirus) also occurred significantly more in food service establishments (57·0%; OR 1·2, 95% CI 0·9–1·6), as did those caused by *Bacillus* spp. (84·1%; OR 4·9, 95% CI 2·6–9·5), scombrotoxin (78·8%; OR 3·4, 95% CI 1·9–6·3), *Staphylococcus aureus* (62·9%; OR 1·5, 95% CI 0·8–3·1), and *Campylobacter* (59·2%; OR 1·3, 95% CI 0·9–2·0) (Table 5). Three-quarters (74·2%) of *Cryptosporidium* outbreaks were linked to community-based settings (Table 5), reflecting their strong association with potable water (96·6%, Table 3).

Contributory factors

Factors thought to have contributed to the outbreak were reported in 62·3% (1529/2455) of foodborne outbreaks. Where this was the case, one (55·9%, 855/1529) or two (31·7%, 485) factors were reported most commonly. In 152 (9·9%) outbreaks three factors were reported, in 34 (2·2%) four factors were reported and in three (<0·1%) five factors were reported. In those outbreaks associated with potable water the main contributory factors identified were agricultural pollution (11·1%, 5/45), heavy rain (11·1%, 5/45), a water system failure (8·9%, 4/45) and consumption of untreated/partially treated water (8·9%, 4/45), Cross-contamination was the most frequently reported factor (41·6%, 631/1518) in other foodborne outbreaks, and particularly so in those occurring in food service establishments (27·3%, 348/1277). The next most common reported factors overall were inadequate heat treatment (38·4%, 583/1518) inappropriate food storage (37·8%, 574/1518) and an infected food handler (19·4%, 295/1518). Inappropriate food storage was the most commonly identified

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Table 5. *Single exposures positively associated with outbreaks of foodborne disease attributed to causative pathogen, in England and Wales, 1992–2008* (*univariate analysis*)

Outcome (outbreak linked to pathogen)	Exposures	OR (95% CI)	*P* value
Bacillus spp.	Food service establishments	4·9 (2·6–9·5)	<0·001
Campylobacter spp.	Poultry meat	2·6 (1·7–4·0)	<0·001
	Milk/ milk products	4·7 (2·1–10·3)	<0·001
	Water	13·0 (6·7–25·5)	<0·001
	Miscellaneous*	1·6 (1·2–2·0)	0·0002
	Food service establishments	1·3 (0·9–2·0)	0·1726
	Residential establishments	1·1 (0·7–1·9)	0·677
	Farm shops	8·1 (3·5–18·6)	<0·0001
	Cross-contamination	2·4 (1·6–3·7)	<0·001
Cl. perfringens	Red meat	10·8 (7·8–14·8)	<0·001
	Poultry meat	2·7 (2·0–3·6)	<0·001
	Residential establishments	2·8 (2·1–3·8)	<0·001
	Inappropriate storage	2·8 (2·1–3·7)	<0·001
	Inadequate heat treatment	1·8 (1·3–2·4)	<0·001
Cryptosporidium	Water	1307·0 (190·4–8974·0)	<0·001
	Community	135·0 (51·3–355·3)	<0·001
Salmonella spp.	Eggs	72·1 (17·1–304·4)	<0·001
	Dessert	11·9 (7·4–19·1)	<0·001
	Poultry meat	1·4 (1·1–1·8)	0·0022
	Food retailers	3·5 (2·3–5·3)	<0·001
	Cross-contamination	3·9 (3·2–4·7)	<0·001
	Inadequate heat treatment	3·0 (2·5–3·7)	<0·001
Scombrotoxin	Fish/shellfish	160·4 (56·0–459·4)	<0·001
	Food service establishments	3·4 (1·9–6·3)	<0·001
	Food retailers	1·2 (0·4–3·2)	0·7831
	Inappropriate storage	3·3 (2·0–5·5)	<0·001
Staph. aureus	Food service establishments	1·5 (0·8–3·1)	0·2235
VTEC O157	Red meat	1·9 (1·1–3·3)	0·0163
	Milk/milk products	11·7 (5·9–23·4)	<0·001
	Farm shops	16·7 (7·6–36·4)	<0·001
	Food retailers	4·3 (2·4–7·7)	<0·001
	Cross-contamination	2·4 (1·6–3·8)	<0·001
Viruses	Fish/shellfish	2·0 (1·4–2·8)	0·0003
	Food service establishments	1·2 (0·9–1·6)	0·1321
	Residential establishments	1·3 (1·0–1·7)	0·0543
	Infected food handler	4·1 (3·0–5·5)	<0·001

OR, Odds ratio; CI, confidence interval; VTEC O157, Vero cytotoxin-producing *Escherichia coli* O157.
* Miscellaneous food includes buffet foods, sandwiches and other dishes comprising multiple ingredients.

contributory factor in private establishments (45·7%, 101/221); and in residential establishments, inadequate heat treatment was the most common contributory factor (27·1%, 97/358).

Cross-contamination events were significantly more likely to have been reported in outbreaks of VTEC O157 (44·0%; OR 2·44, 95% CI 1·6–3·8), *Campylobacter* (38·8%; OR 2·4, 95% CI 1·6–3·7) and *Salmonella* (39·6%; OR 3·8, 95% CI 3·2–4·7) and those of inadequate heat treatment in outbreaks of *Cl. perfringens* (35·7%; OR 1·8, 95% CI 1·3–2·4) and *Salmonella* (34·6%; OR 3·0, 95% CI 2·5–3·7) (Table 5). Inappropriate storage was most commonly observed in scombrotoxin (86·8%; OR 3·3, 95% CI 2·0–5·5) and *Cl. perfringens* outbreaks (51·2%; OR 2·8, 95% CI 2·1–3·7), while infected food handlers were the main contributory factor in viral pathogen outbreaks (58·0%; OR 4·1, 95% CI 3·0–5·5).

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DISCUSSION

The number of foodborne outbreaks reported to the HPA in England and Wales has continued to decline markedly since 1992. The main reason for this decline has been the reduction in outbreaks attributed to *Salmonella* spp. The decline in *S.* Enteritidis PT4 outbreaks in particular clearly indicates the effect of successful intervention measures, i.e. improved biosecurity and vaccination of UK poultry flocks introduced in the late 1990s [12], and is reinforced by similar trends in salmonellosis post-introduction of similar control measures in other countries [17, 18]. In contrast, outbreaks attributed to *S.* Enteritidis non-PT4 increased over the surveillance period (mainly PT1 and PT14b), with the greatest increases occurring from 2002, with a preponderance of these associated with eggs or egg dishes linked to food service establishments. Surveillance of salmonellosis from 1998 to 2003 also showed upsurges in *S.* Enteritidis non-PT4 in other European countries [19]. These major resurgences were associated with substantive changes in market supply with the sourcing of eggs from other egg producers in member states, where there is a lack of vaccination of layer flocks against *Salmonella* or controlled assurance [20]. This continues to be a public health concern with a substantial rise in the number of outbreaks and sporadic cases of *S.* Enteritidis PT14b still occurring during the latter part of 2009 and associated with non-UK eggs linked to food service establishments [21]. Despite almost 20 years of national guidance on the safe handling and use of eggs [22–25], eggs have continued to be implicated as a source or vehicle of *S.* Enteritidis infection in outbreaks associated with food service establishments which implies that government advice has not been followed.

Despite the significant health burden that sporadic campylobacteriosis places on the community [7], the number of outbreaks caused by this pathogen was relatively low (4%). This is likely to reflect the fact that *Campylobacter* outbreaks are difficult to detect in the first place [26]. Most outbreaks were the result of poultry meat consumption (38%), in accord with Canadian data [27] (56% of *Campylobacter* outbreaks), in contrast to that in the USA where dairy products were more frequently implicated in these outbreaks (45%) with poultry meat accounting for less (14%) [28]. Poultry meat on UK retail sale are commonly contaminated with *Campylobacter* spp. [29] and cross-contamination was significantly associated with *Campylobacter* outbreaks in this study. Reducing *Campylobacter* in chicken continues to be a key target in the UK Food Standards Agency's strategy on foodborne disease [2]. In New Zealand, which had the world's highest rate of *Campylobacter* infection, recent successful national control measures through targeted poultry strategies has reduced the number of human cases of campylobacteriosis by 50% [30]. Such interventions included better education of the public with regard to safe barbeque cooking, through to educating poultry farmers on farm biosecurity and using hyperchlorinated water to cool birds post-dressing [31]. The New Zealand experience in understanding and controlling *Campylobacter* in poultry meat is to be utilized in developing international guidelines on good hygienic practice and hazard control measures targeting *Campylobacter* in poultry meat [32].

The other organisms that stood out in terms of disease burden were VTEC O157 and *L. monocytogenes*. Although the number of foodborne outbreaks of these pathogens was small, there was substantial associated morbidity. *L. monocytogenes* is recognized as being the most frequent cause of death from foodborne infections in industrialized countries [33] and this is now also reflected in England and Wales.

Given the impact which information from outbreaks may have on national and international food-safety policy, the quality of the outbreak investigation is important [1, 6]. However, the degree of evidence that links a food vehicle with human illness in foodborne outbreaks varies. Identifying a food vehicle through microbiological testing depends very much on the speed with which the outbreak comes to official attention, i.e. the longer this is the less likely that the suspected foods will be available for testing. The overall increase in the proportion of outbreaks providing descriptive evidence is almost certainly influenced by familiarity with links between food vehicles and organisms, notably *Salmonella* and eggs, and is therefore subject to biases arising from the belief of the investigators. However, well conducted case-control or cohort studies should overcome this bias if all possible foods available to cases are investigated. During the 17-year period of this study, the use of analytical epidemiology in investigation outbreaks declined from 1992 to 2005 with a reversal in this trend observed towards the end of the surveillance period. The recent increase in the use of analytical epidemiology in outbreak investigations follows the

creation of the HPA in 2003. Prior to the inception of the HPA most foodborne outbreaks were investigated by health protection teams led by CCDCs based in district health authorities. When the HPA was created the local health protection teams in England were integrated to form the Agency's Local and Regional Services Division (LaRS). This led to the development of a series of initiatives within LaRS to introduce standardized guidelines and operating procedures designed to disseminate best practice across the organization. It is likely that these initiatives have resulted in an increase in the use of analytical epidemiology as part of field investigations. Increased vigilance following unprecedented publicity following notable outbreaks, such as both the Central Scotland and South Wales outbreaks of VTEC O157 infection [34, 35], can also prompt greater effort in investigation or reporting of outbreaks as seen in the peaks in outbreaks reported in 1997 and 2005 [36].

The analysis of foodborne outbreak data over time is extremely useful in that it may reflect changes which occur in food production, handling and service [27]. Few countries hold such a large collection of standardized outbreak information which details pathogen–food causal factors as England and Wales; however, in other countries, integrated collections of foodborne outbreaks from wide time periods have been analysed, including major pathogen–food associations. In a Canadian study [27], almost a third (30%) of *Salmonella* foodborne outbreaks between 1976 and 2005 were associated with fresh vegetables and fruit (produce) and 15% with poultry meat. However, we showed that in England and Wales most salmonellosis outbreaks were linked to poultry meat (21%) and miscellaneous foods (20%) with fresh vegetables and fruit (produce) accounting for only 5%. In the USA between 1998 and 2002, most foodborne outbreaks linked to *Salmonella* were associated with complex multi-ingredient foods and eggs [28]. However, classification of foods often varies depending on the source of the data. This lack of harmonization of food categories can make it difficult to compare outbreak data in different countries and to evaluate the contribution of specific food commodities to human illness. Painter *et al*. [37] and Greig & Ravel [9] have both recently published approaches to classifying and grouping implicated foods but differ in their proposed hierarchy of food commodities. In Europe, EFSA is also currently developing a food classification and description system for exposure assessment to harmonize analyses and interpretation of such data in Europe.

The HPA surveillance system of general foodborne outbreaks has proved a useful resource for source attribution of gastrointestinal illness to specific foods or situations that caused them over the 17 years of its operation. In 2009, this was further improved by obtaining information on food provenance and the place of the problem (e.g. for manufactured and/or imported foodstuffs), the level of the causative pathogen/toxin in the food, and by refining and incorporating a hierarchical system for food classification and description to enhance its use for exposure assessments. In 2009, the surveillance system was also converted to an electronically based system to facilitate the collation of outbreak information from investigators and communication of information back to them and other stakeholders.

Although the downward trend in general foodborne outbreaks reported here is encouraging and mirrors the national decrease in *Salmonella* infection, the proportion of outbreaks linked specifically to food service establishments increased (restaurants and hotels in particular). Our analysis found much evidence that outbreaks associated with these settings are related to cross-contamination in the kitchen and this is supported by studies showing how easy it is for the environment to become contaminated [38]. Improving hygiene and lowering the risk of introducing *Salmonella* and other pathogens into the food service establishment are needed in order to realize further public health benefits. The food service sector needs to adopt appropriate control measures, and follow advice provided by national food agencies in order to reduce the risk of infection.

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DECLARATION OF INTEREST

None.

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