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Salmonella and campylobacter contamination of raw retail chickens from different producers: a six year survey

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SUMMARY

Between 1995 and 2000, a prospective survey was undertaken to investigate the levels of contamination of raw retail chickens ($n = 1127$) with salmonella and campylobacter. The levels of contamination over the 6-year period were 11% (95% CI ± 6·5%) for salmonella, and 57% (95% CI ± 9·5%) for campylobacter. *S.* Bredeney (20%) and *S.* Enteritidis (18%) were the dominant serovars. Although salmonella contamination was higher than in an earlier survey we conducted (7%), since 1998 it has declined to 6%. Many *S.* Enteritidis isolates (43%) were associated with one large integrated poultry organization that appears to have successfully managed the contamination, and the serovar has not been isolated since 1998. Contamination ranged from 0 to 44% between different producers. There was no significant difference between producers contributing large and small numbers of samples, although some small producers had much poorer contamination rates than others. *S.* Bareilly, *S.* Bredeney, *S.* Enteritidis and *S.* Virchow showed associations with particular producers. Campylobacter contamination remains high. Contamination ranged from 47 to 81% between different producers. This study did not show a temporal association between contamination of chickens and human campylobacter infections, indicating that many cases of human campylobacteriosis, particularly during seasonal peaks, do not originate from chickens. Control measures that have reduced salmonella contamination have been largely ineffective against campylobacter and new interventions are needed. Most raw chickens are contaminated with these pathogens, and communicating the importance of minimizing this risk to caterers and the public is vital in reducing human infections.

INTRODUCTION

The production of chickens has long been associated with the presence of salmonella and campylobacter that cause human enteric illness, and the problems of the poultry industry have been described in depth [1]. In the UK, most human salmonella infections with identified vehicles are caused by eggs (generally by *S.* Enteritidis) [2]. Chicken is a vehicle for a wider range of salmonella serovars in 18·5% of all outbreaks of foodborne infection, and 67% of salmonella outbreaks are attributed to chicken [3]. Risk factors for flock infection by salmonella have been identified [4], but some remain unclear for campylobacter [5] due to practical reasons including poor recovery of sublethally injured organisms [6], genomic instability [7, 8], and untypability [9]. Campylobacter is the most common bacterial cause of human gastro-intestinal infections, and is responsible for a notable health burden due to acute illness, and to a lesser extent, death and long-term morbidity [10, 11]. Campylobacters are commonly found on chickens and these are an

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important vehicle for human infections [5, 12, 13]. The incidence of human campylobacter enteritis in Northern Ireland has been lower than the rest of the United Kingdom, although the reasons for this are unclear [1, 14].

Many studies have examined chicken to determine the levels of contamination with these pathogens with great variation in the results depending on the samples taken (whole, portions, fresh, frozen, organic, free range, etc), point of sampling in the processing chain, country [15], use of serological assays [16], carcass sampling and microbiological methods used in testing [17]. Direct comparisons between different reports are seldom possible. Reviews of earlier studies have shown the prevalence of campylobacter to vary between 0 and 100% in different surveys [1, 5, 18, 19]. At both extremes the number of samples was very small, and very few surveys have examined more than a few hundred chickens. On many carcasses the counts of campylobacter are high [20], increasing the likelihood of transmission by poor hygiene [21].

Likewise, salmonella contamination varies widely depending on the nature of the survey and methods used. An earlier study in 1994 designed to compare contamination rates between chilled and frozen chickens sold from large and small retailers reported salmonella contamination in 140 chickens sampled [22] in 1994. Chilled birds, and those sold from butchers' shops were more likely to be contaminated. The overall contamination rate was around 7%, lower than figures reported in England and Wales, and may partly have accounted for the lower levels of human infections in Northern Ireland at this time. Human salmonellosis and campylobacteriosis subsequently rose, and chicken contamination also increased. Little information is available about variations in contamination rates between producers. If large differences exist, producers with particularly poor contamination rates might potentially contribute an excessive proportion of human infections, and identify themselves in need of stricter control. Conversely, producers with low contamination will have systems in place that should be used more widely by other producers if the number of human cases is to be reduced.

The earlier survey revealed the need for a large number of samples over a long period to achieve the statistical power necessary to have confidence in identifying changes in contamination over time, and to avoid seasonal effects. The survey reported here was established to determine changes in pathogen contamination of raw retail chickens over time and between producers.

MATERIALS AND METHODS

Sample collection and preparation

A prospective survey of raw retail chickens was organized between 1995 and 2000. Environmental Health Officers (EHOs) were instructed to collect each month 50 raw chickens, wrapped and unwrapped, from retail premises in Northern Ireland. Samples were transported to the laboratory in cool boxes at < 5 °C. EHOs recorded information that was available on sample request forms, and these details were entered into Specimen Control System software (Microft, Kew, England). Detailed information on market share of producers was not available and sampling was intended to reflect market share as seen by the shopper.

Neck skin (25 g) was removed from each chicken and cut in half for salmonella and campylobacter examinations. The portion for salmonella testing was added 1 to 10 (w/v) to buffered peptone water (BPW, Oxoid, England) non-selective enrichment broth before stomaching for 30 s (Stomacher 400, Seward, London, England).

Salmonella

The homogenate was decanted into a honey jar and incubated at 37 °C for 18 h. Selective enrichment for salmonella was done by pipetting 0·1 ml of the pre-enrichment culture into 10 ml Rappaport–Vassiliadis soya peptone broth (RVS, Oxoid) and incubating in a water bath at 41·5 °C for 22 h. RVS was streaked onto both xylose lysine desoxycholate (XLD, Oxoid) and modified brilliant green agar plates (BGA, Oxoid) and incubated at 37 °C for 22 h. Red colonies (BGA) and red colonies with or without black centres (XLD) were confirmed from both agars by serological and biochemical testing. Five suspect colonies from each plate were suspended in 1·0 ml sterile saline and inoculated onto MacConkey agar (Oxoid) purity plates and into urea broth before being incubated 37 °C for 18–24 h. Serological confirmation was performed by subculturing non-lactose-fermenting colonies to nutrient agar slopes with 0·5 ml peptone water added. These were incubated at 37 °C for 18–24 h. Saline suspensions from the slopes were tested by agglutination with polyvalent 'O' A-S, polyvalent 'O' A-I + Vi,

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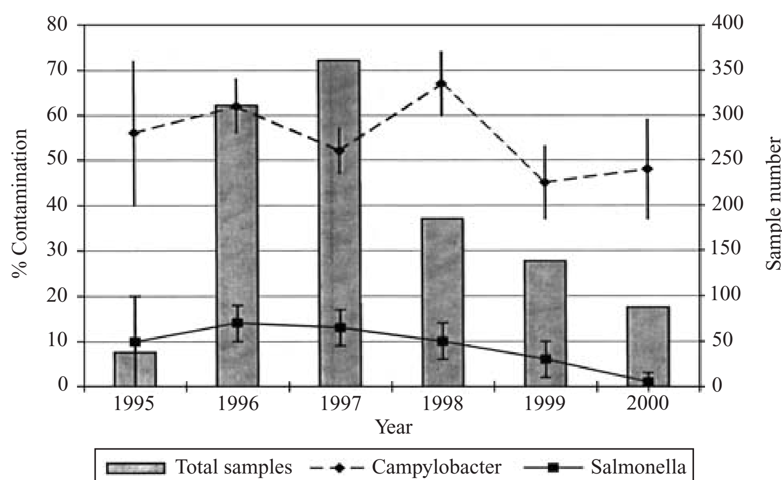

Fig. 1. *Annual trends in* salmonella *and* campylobacter *contamination.*

and polyvalent 'H' phase 1 and 2 salmonella antisera, followed by individual 'O' and 'H' antisera (Pro-Lab Diagnostics, Neston, England). Isolates were confirmed biochemically in API 20E test strips (Bio-Mérieux, Basingstoke, England), and *S.* Enteritidis, *S.* Typhimurium and any isolates not identified were sent to the Public Health Laboratory Service Laboratory of Enteric Pathogens (PHLS LEP).

Campylobacter

The portion of neck skin for campylobacter examination was added to Exeter broth (1 in 10 w/v) [23], stomached for 30 s, and poured back into a 250 ml jar, and headspace minimized with additional broth. Broths were incubated at 37 °C for 18 h, and then at 42 °C to give a total incubation of 48 h. Enrichment broth was streaked onto campylobacter Charcoal Deoxycholate Agar (CCDA) plates (Oxoid) and incubated microaerophilically using Oxoid Campygen gas packs at 37 °C for 48 h. Five suspect colonies were tested for oxidase production, and each subcultured onto two Columbia blood agar plates (CBA, Oxoid). One plate was incubated in air, and the other with a Campygen gas pack (Oxoid) at 37 °C for 22 h. The Oxoid latex agglutination kit was used for serological confirmation. *C. jejuni* was differentiated from *C. coli*/*lari* by reaction in hippurate.

RESULTS

Satisfactory information was received to allow the analysis of 1127 samples. For various logistical reasons the target of 50 samples per month was not achieved. Supermarkets/grocers contributed 678 samples, 235 came from butchers, and the rest from a variety of small outlets. Salmonella was detected in 123 samples (7 samples had 2 or 3 serovars), campylobacter in 632. The levels of contamination over the 5-year period were 11% (95% CI $\pm$6·5%) for salmonella, and 57% (95% CI $\pm$9·5%) for campylobacter. The annual trend in contamination is shown in Figure 1. Analysis of annual samples provided sufficient numbers to enable confidence intervals to be calculated. For monthly and quarterly figures the confidence intervals were very wide.

There was no significant difference in contamination between producers and retailers (own brand or no brand label) from which >10 samples were received ($n=803$) and those from which <10 samples were examined ($n=311$) (Table 1). Thirteen samples were excluded because of inadequate information on their origin. The overall salmonella contamination was 11% for both large and small producers ($P=0·73$). For campylobacter, contamination was 56·9% for large and 56·3% for small producers ($P=0·96$).

The seasonal relationship between contamination with salmonella, campylobacter and either organism is shown in Figure 2. The quarterly trend in contamination was analysed by logistic regression with re-scaling for over-dispersion performed using GLIM (Generalised Linear Interactive Modelling) statistical software (Royal Statistical Society). The decline was significant only for salmonella ($P=0·0012$). The apparent decline in campylobacter was not significant ($P=0·11$), and there was no

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Table 1. *Salmonella and campylobacter contamination by producer*

Producer	Number examined	Salmonella detected *n*	Salmonella detected %	Campylobacter detected *n*	Campylobacter detected %
> 10 samples					
A*	37	4	10·8	24	64·9
B	31	3	9·7	18	58·1
C	196	6	3·1	110	56·1
D	51	16	31·4	31	60·8
E	16	5	31·3	12	75·0
F	16	2	12·5	13	81·3
G*	41	3	7·3	27	66·0
H	48	4	8·3	28	58·3
I	114	15	13·2	56	49·1
J	137	9	6·6	74	54·0
K	36	16	44·4	22	61·1
L*	43	6	14·0	21	49·0
M*	15	2	13·3	7	47·0
N*	22	0	0	14	64·0
Total	803	91	11·3	457	56·9
< 10 samples					
Various small producers/ retailers	311	32	10·3	175	56·3

* Retailer.

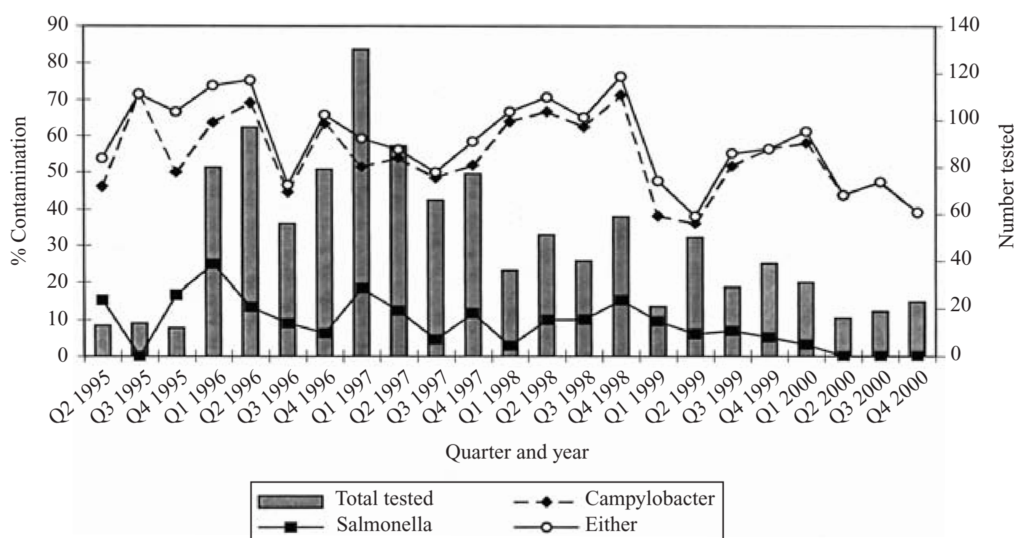

Fig. 2. *Quarterly* campylobacter *trends in chickens and humans.*

seasonal effect ($P = 0{\cdot}75$). A salmonella peak around the first quarter of each year was a significant seasonal trend ($P = 0{\cdot}019$). There was a more significant declining trend ($P = 0{\cdot}0023$) in salmonella contamination over the years. *S.* Enteritidis PT4 has not been isolated since 1998, and the annual decline is significant ($P = 0{\cdot}027$).

The quarterly incidence of human campylobacter cases and chicken isolations is shown in Figure 3. There was an insignificant negative correlation ($-0{\cdot}26$) between these, and they clearly lacked a seasonal relationship.

Six serovars constituted 70% of salmonella isolates (Table 2). Many of the most common serovars showed

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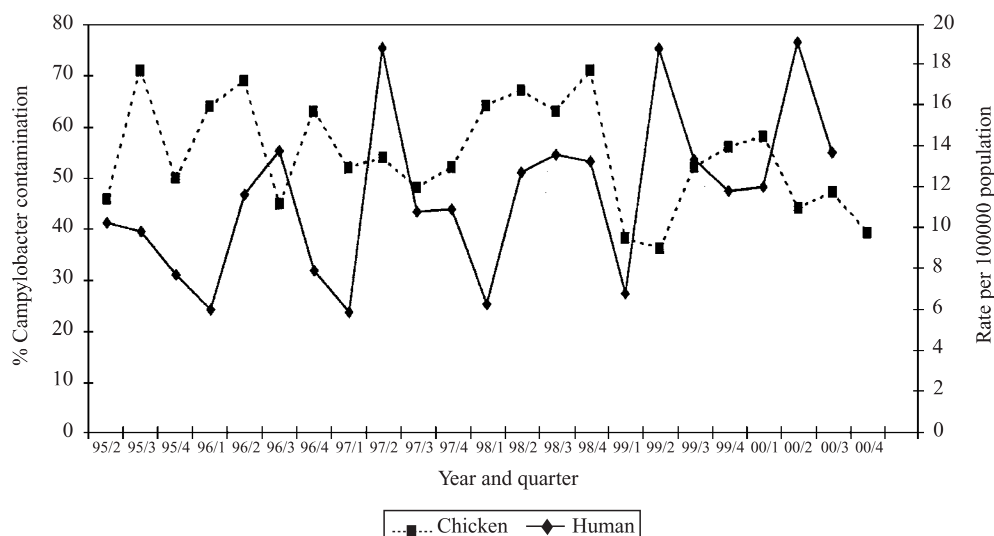

Fig. 3. *Quarterly contamination rates with* salmonella *and* campylobacter.

Table 2. *Salmonella serovars isolated from chickens*

Serovar	Number isolated	Association with producer
Agona	1	
Anatum	1	
Arizona	1	
Bareilly	15	K
Bovis mobificans	1	
Brandenburg	1	
Bredeney	26	E, L, D
Daula	1	
Enteritidis	23	I
Eschweiler	2	
Essen	1	
Glostrup	1	
Hadar	1	
Heidelberg	1	
Indiana	4	
Infantis	4	
Kentucky	16	
Kralingen	1	
London	2	
Mbandaka	2	
Meunster	1	
Montevideo	2	
Nachshonin	1	
Orion	2	
Panama	1	
Saint-paul	1	
Schwarzengrund	4	
Trachau	1	
Typhimurium	5	
Unknown	1	
Virchow	6	G

associations with certain producers or retailers. For *S*. Bareilly (11·5% of total), 8/15 isolates came from one small-medium producer (K). For *S*. Bredeney (20%), 4/26 came from one large producer outside NI (E), 3/26 came from a supermarket chain (L), and 9/26 came from one small producer in NI (D).

DISCUSSION

Salmonella

A statistically significant reduction in salmonella contamination was seen over the 6-year study period. Since 1997–8, large, but possibly not small, producers in Northern Ireland have been vaccinating certain flocks (e.g. broiler breeders but not broilers) against salmonella depending on their destination market. It is likely that this has played an important part in the reduction of contamination, but since it was introduced selectively and as part of a programme of improved biosecurity, vaccination cannot be identified clearly as the main reason for any improvement. In England and Wales the role of vaccination in reduction of infection is clearer [24]. In Scotland, salmonella isolations from humans have also fallen sharply since 1998, and poultry isolations since 1999 [25]. During the same period in Northern Ireland, human salmonella isolations continued to increase until late in 1999 [26]. The decline in salmonella isolations from chickens, in particular *S*. Enteritidis which has not been isolated since 1998, while human

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salmonellosis continued to rise may indicate the importance of sources other than chicken in NI.

The poultry industry has complex inter-relationships in company ownership, rearing, trade and branding. Certain companies distribute their products widely throughout the United Kingdom and beyond. A compulsory slaughter and compensation policy was introduced in Northern Ireland in 1990 for breeder flocks infected with *S.* Enteritidis or Typhimurium. This did not apply to broiler flocks, although precautions are taken for infected flocks such as processing them at the end of the day. Legislation requires traceability and surveillance of breeders and hatcheries, but the details of bird movements are usually unclear to those outside the industry and are potential confounding factors in a large survey such as this. The survey was intended to be representative of raw chickens purchased by consumers in a wide variety of retail stores. This was not achieved fully due to factors too complex for detailed discussion here, but which included incomplete information, market changes and sample availability in relation to staff rotas and sample quotas. However, the survey shows that certain producers are associated with particular salmonella serovars, and improved co-ordination between veterinary and public health officers could reduce the incidence of human and animal illness.

No reason was clear for the peak in salmonella during the first quarter of most years, but it may possibly have been due to a change in the age profile of flocks after the increased Christmas market for poultry. It has been noted by veterinary officers but has no obvious explanation. Although statistically significant, it may be artefactual. From 123 salmonella contaminated samples, *S.* Enteritidis was found in 23 during the 5-year period (21 × PT4, 1 × PT5a, 1 × PT6a). *S.* Enteritidis PT4 rose from 1/38 samples in 1995 to 11/361 samples in 1997, then began to decrease. Since 1998 we have not isolated this phage type. An increase in this serovar might have been expected from breeding flocks if an influx of broiler chicks had been the reason for the seasonal peak, but the incidence of PT4 was distributed throughout the year. This indicates that flocks infected with *S.* Enteritidis are increasingly being excluded from food products. *S.* Enteritidis PT4 tends to be transmitted vertically and has high endemicity. If found, it probably indicates that the majority of that flock is infected. Prior to the mid 1980s, *S.* Enteritidis was not frequently encountered [27]. Its prevalence subsequently rose, possibly due to poor crate hygiene during trade in breeding flocks and eggs. Only the largest poultry companies have breeder flocks, and smaller producers buy from these. Improved awareness, feed preparation, biosecurity, hygiene and control measures have reduced the prevalence of this serovar [28].

Other serovars tend to produce low-grade, intermittent infections in birds and be transmitted horizontally, often by infected feedstuffs that have not been thermally processed. Thirty-one different salmonella serovars were isolated (Table 2) with *S.* Bareilly, *S.* Bredeney, *S.* Enteritidis, *S.* Kentucky, *S.* Typhimurium and *S.* Virchow accounting for 70% of the isolates. Legislation governs only hatcheries and breeder flocks and there is neither legislation nor funding for broiler surveillance. A proposed EU Zoonoses Directive may require surveillance of broiler flocks. One infected bird can contaminate other birds on the production line for the rest of the working day. The relatively low level of salmonella contamination indicates that infected flocks were generally being excluded from the line.

The control of contamination seen in broilers has not been evident for layer flocks and eggs continue clearly to be implicated in food-borne outbreaks. Chicken contamination was not related closely to human salmonellosis by this study since the number of human cases was fairly static between 1995 and 1997 [26] when chicken contamination was highest. The rise in human cases during a period of decline in chicken contamination probably indicates the increasing importance of eggs as a source between 1997 and 1999. Although *S.* Enteritidis was not found in chickens after 1998, the number of clinical laboratory reports of this serovar rose from 169 in 1997 to 272 in 1998 and 462 in 1999, before dropping equally sharply to 235 in 2000 and 180 in 2001. Of the 272 laboratory reports of *S.* Enteritidis in 1998, 207 (76%) were PT4 and 5 outbreaks accounted for 101 (37%) of *S.* Enteritidis reports. *S.* Enteritidis PT4 was responsible in 4 outbreaks; in 2 of these eggs were the vehicle, in 2 the vehicle was chicken. In the fifth outbreak eggs used to make sauce contained *S.* Enteritidis of another phage type (Brian Smyth, Communicable Disease Surveillance Centre Northern Ireland (CDSCNI), personal communication). Negligible information is available on the infection of layer flocks, but outbreak data show that eggs remain a major source of *S.* Enteritidis infections. This is possible because of the separation of the broiler and

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egg-layer industries. Broiler houses operate on an all-in, all-out basis and are amenable to effective cleaning. Egg layer farms have multi-age houses to provide a constant supply of eggs. The cages and other equipment are impossible to clean effectively and re-infection of new birds routinely occurs.

Our earlier survey [22] was designed to compare fresh and frozen chickens from supermarkets and butchers' shops. The sampling was different from the present survey and results cannot be compared directly. However, salmonella contamination increased considerably for around 2 years after the first survey (from 7% to over 20% in late 1995, early 1996), but more recently has returned to lower levels of 5–6%. A large survey of chicken carcasses and products in Belgium found contamination levels of a similar order to the overall figure we found, but also noted a lack of improvement over a 4-year period [29].

While it is important to improve on contamination rates of 11%, previous surveys have often found levels of contamination that are much higher, e.g. 23% in England in 1995 [30] and 26% in the Republic of Ireland (ROI) in 1999 [31]. The lower prevalence of contamination since 1998 is undoubtedly due to a combination of factors whose importance cannot be identified clearly. Pressure from large supermarket chains has probably been an important driver of these improvements in the industry. Due to the limited number of breeder flocks, these improvements may eventually filter down to smaller producers if they give attention to feedstuffs, biosecurity and vaccination.

Campylobacter

Previous surveys in Northern Ireland have shown that 38% of 120 packs of chicken sampled from a single producer's premises over 1 year were contaminated with campylobacter [32]. The present survey has shown that while this producer has controlled salmonella contamination since 1998, campylobacter contamination has not changed. In an earlier survey of 153 packs of chicken wings from different producers sampled at retail over 10 weeks, 65% were contaminated with campylobacter [19].

Significant seasonality in the numbers of campylobacters in chicken intestines has been reported [33], but not confirmed by others [34]. Hours of sunshine and temperature were important influences, and reduced recovery of *C. jejuni* has been described in December and January [35]. Alterations in sub-types have been reported throughout the year [36]. There is evidence from previous surveys conducted in Northern Ireland that the complexity of the campylobacter flora may reduce between killing and selling of poultry [37], although, arguably, a wider range of viable but non-culturable campylobacters may continue to present a risk. The present retail survey did not enumerate campylobacters and was not designed to investigate seasonality. However, the seasonal variation it showed was not statistically significant and could not be related to the findings of some other workers [32–35].

Campylobacter is endemic in chicks at much higher levels than salmonella. It pervades all stages of chicken production [38], and its initial appearance by 4 weeks of age may be related to the decline in maternal antibodies that possibly mediate strain-specific killing of campylobacters in chicks [39]. The range of contamination was smaller between producers than with salmonella. In raw retail chickens campylobacter contamination ranged from 47 to 81% between different producers. The mean was 56·9% for large producers/retailers and 57·6% for small producers, and this difference was not significant. It has been demonstrated that skinning poultry may not significantly reduce the presence of campylobacter on broiler parts [40], so the risk will remain high to consumers of prepared chicken portions.

Figure 3 shows that no temporal correlation between human cases of campylobacteriosis and infected chickens was found. Despite human campylobacteriosis being attributed to poultry in 50–70% of cases [41], the relationship demonstrated here is a negative correlation. A recent case-control study of over 200 cases found only chicken eaten in restaurants to be associated with campylobacteriosis [42]. Strong associations, both positive and negative [43], have been found in case-control studies and investigations of illness. However, the majority of cases are sporadic and difficult to investigate successfully since discriminatory, reliable and standardized sub-typing has yet to be achieved. The epidemiology is complicated by co-infection with different strains and this may invalidate certain typing investigations [44]. A study using pulsed-field gel electrophoresis (PFGE) found that limitations were placed on traceback by extensive polymorphism, more than one genotype per carcass, and genetic instability [45]. Most cases therefore remain unexplained. To compare self-reporting human cases with randomly selected chickens is an unbalanced comparison, but if chicken were

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responsible for a large proportion of campylobacter cases, some seasonal relationship might be seen. A similar comparison for salmonella is of no value since there are fewer isolates, and more direct epidemiological associations are made by serotyping and phage typing. Campylobacter subtypes vary during the year and certain subtypes are selected through the production chain [37, 46]. It is possible that particular subtypes which are more likely to cause human infection and less easy to culture dominate the chicken flora periodically. While campylobacters from chickens may cause much human illness, it seems likely that peaks in human infection originate from sources other than chicken.

Producer effects on hygiene

Little information is available in the literature on variations between producers. Previous investigators have generally sampled from a single farm, killing plant or producer to simplify collection and analysis or for reasons of commercial sensitivity, and retail sampling has not been related to producers [31, 32]. For this analysis, samples were grouped into large and small producers depending on whether >10 or <10 samples were received. A minor proportion of imported samples from producers classed as small for the purpose of this survey may have come from producers that are economically large in their home market. There was no significant association between salmonella and campylobacter as co-contaminants (6·5%; χ^2: $P=0{\cdot}44$). The low level of mixed contamination has been shown by other workers in both flocks [47] and carcasses [48], and indicates that cross-contamination by evisceration equipment or scalding baths is not a general problem. Studies of co-infection in flocks are rare [47], but a Dutch study reported 25% of broiler flocks were infected with both organisms [49]. Such variations may be due to national differences in poultry-keeping practices or epidemiology. Wedderkop et al. [47] concluded that intensive cleaning was ineffective and that improved biosecurity which prevents the entry of campylobacter into broiler houses is important. Slader et al. [46] also showed that disinfection of transport crates is limited in the absence of thorough cleaning. Our results suggest that the biosecurity which contributed to reducing salmonella infection did not impact on campylobacter. This indicates that most campylobacter contamination has a different vehicle from salmonella.

With some exceptions, the similarity between contamination at different producers was noteworthy (Table 1). Salmonella contamination ranged between 0 and 44% and the difference between means of large and small producers was not significant (11·3 and 10·3%). One large and two small-medium sized producers (D, E and K) had worse than average salmonella contamination which was significant ($P=<0{\cdot}001$) and indicates poor processing practices. Information was not available to exclude the use of more highly infected flocks by these producers, but this was suspected in at least one of the three cases.

The association of four serovars with six particular processors (Table 2) probably indicates that most contamination is due to infections in the live birds which came from a limited number of breeders and rearers. *S.* Bredeney was the most common serovar, showing association with a large producer outside NI, and a small producer and supermarket chain in NI. It was also the most common serovar in a recent survey in the Republic of Ireland (ROI) of retail meat products where 26% of chicken samples contained salmonellae [31]. There was no information to identify the supermarket's and small producer's sources, but as this is an otherwise uncommon serovar, it may have come from the same large producer. For *S.* Enteritidis (17·7%), 10/23 (43%) came from one large producer (I). For *S.* Kentucky (12·3%), 6/16 were associated with producer D, and 2/16 with producer K that also had a problem with *S.* Bareilly. No single producer was associated with *S.* Typhimurium (3·8%). For *S.* Virchow (4·6%), 5/6 isolates were from a large supermarket chain (G) and sampled 2 days apart. This may indicate an infected flock, or that contamination of the production line by even a single bird occurred at this time. Some associations between other producers and serovars persisted throughout the survey and indicate that certain serovars can be associated with particular producers, although not definitively. In some cases, poor plant hygiene may have been a factor, but more generally this indicates persistent or recurrent flock infection which may be related to broiler or broiler-breeder flocks and their feedstuffs.

Over the 6-year period, one large integrated poultry organization (I, 114 samples) had salmonella contamination slightly worse than average and it contributed 43% of the *S.* Enteritidis PT4 isolates. It appears to have successfully controlled this problem since no salmonella of any serovar was isolated during examination of 19 samples from this producer

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after 1998. Some of the controls [50] for salmonella, which included breeding, biosecurity and vaccination [1], may have played a part in the reduction in campylobacter by producer I, which was slightly lower than most producers. Producer I was not sampled evenly throughout the survey and most samples were taken prior to 1998. Before 1998, 46 of 95 samples contained campylobacter; after 1998, 10 of 19 samples were thus contaminated ($P = 0{\cdot}846$). Control measures, particularly in breeder flocks, that improved salmonella infection did not significantly change campylobacter contamination in broilers.

This large retail survey shows that the industry is having some success in reducing salmonella contamination, in particular *S*. Enteritidis PT4. The reduction in broiler contamination should free resources to tackle the ongoing problem of *S*. Enteritidis in eggs. The lack of correlation between campylobacter and salmonella contamination indicates generally different routes of infection and this is important for future intervention strategies. The evidence here does not closely relate samples of the chicken population to human cases of these pathogens. Nevertheless, raw chickens should continue to be regarded as a high risk source of human infection since over 60% of birds carry these pathogens, and evidence from many epidemiological investigations has related chickens to many human cases. It is unfortunate that the movement of live and killed birds within the industry is not more transparent to the veterinary authorities since there is scope for improved control. This information should be integrated to assist epidemiology and control. Great care is needed in preparation and cooking, and the risks of handling poultry must be communicated to caterers and the public if human infections are to be reduced.

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