

The seasonality of human campylobacter infection and *Campylobacter* isolates from fresh, retail chicken in Wales

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SUMMARY

Seasonal peaks in both human campylobacter infections and poultry isolates have been observed in several European countries but remain unexplained. We compared weekly data on human campylobacter infections with thermophilic *Campylobacter* isolation rates from fresh, retail chicken samples ($n=514$) purchased weekly in Wales between January and December 2002. Human isolates ($n=2631$) peaked between weeks 22 and 25 (early June) and chicken isolates ($n=364$) between weeks 24 and 26 (late June). In the absence of a temporal association, we postulate that the seasonal rise in humans is not caused by a rise in isolation rates in poultry but that both are more likely to be associated with a common, but as yet unidentified, environmental source.

INTRODUCTION

Campylobacter is the commonest cause of bacterial gastroenteritis in most countries, yet many aspects of its epidemiology remain poorly understood. Thermophilic *Campylobacter* spp., including *C. jejuni*, *C. coli* and *C. lari*, are those most associated with human gastroenteritis [1]. The genus is widespread in the environment and has been isolated from a variety of animals and birds. The most important route for sporadic campylobacter infection in industrialized countries is eating or handling chicken, but the source for 30–50% of cases remains undetermined [2]. Although several other risk factors for campylobacter infection are recognized, including drinking raw milk, eating non-poultry meats, living or working on farms,

contact with chickens or with domestic pets, and cross-contamination from raw meat to cooked foods, these only account for a minority of cases [3–5].

Seasonal peaks in human campylobacter infection have been observed in several European countries, particularly the United Kingdom and the Nordic countries, and in New Zealand [6, 7]. These peaks have two distinctive features: they are consistent year-on-year and they vary in prominence and timing from country to country (weeks 20–22 in Wales, weeks 24–27 in Scotland, weeks 31–32 in Denmark, weeks 29–31 in Finland, and weeks 32–34 in Sweden) [7]. A better understanding of this seasonal pattern could provide important clues to the aetiology of human campylobacter infection.

Several studies have investigated the seasonality of *Campylobacter* in poultry [8–10], and one has attempted to correlate data on human and chicken isolates [10]. However, most studies have focused on broiler flocks rather than on retail chicken, and

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weekly data on *Campylobacter* isolates have not been compared. We carried out a microbiological survey of fresh chicken on retail sale in Wales, between 1 January 2002 and 31 December 2002, together with a review of human campylobacter infections reported in Wales over the same period.

METHODS

Chicken sampling, preparation and examination

Fresh, raw, chickens were purchased on a weekly basis from retailers selected at random by environmental health officers from 20 out of 22 local authorities in Wales. Seventy per cent of samples were obtained from supermarkets and retail grocery stores and 30% from local, independent butchers. All samples were purchased directly from chilled cabinets or displays and were examined within the use-by date. Weekly reports were based upon the week the chicken was purchased. General information on the length of time between chicken slaughter and expiry of the use-by date was obtained by one of the participating environmental health departments from a large national chicken producer within their locality.

Chicken samples were submitted to one of four public health laboratories and stored at $<5^{\circ}\text{C}$ before microbiological examination. The neck skin was removed and divided into two pieces (no specific weight required). The carcass was then placed into a sterile bag and manually rinsed for 2 min in 225 ml of buffered peptone water (BPW), ensuring that all surfaces, internal and external, had contact with the rinse. The rinse was then poured into a sterile jar and a portion of neck skin added. 25 ml of this rinse was then pipetted into 225 ml of campylobacter enrichment broth (CEB) to which the remaining part of the neck skin had been added. The CEB was incubated for 24 h (± 6 h) at 37°C ($\pm 1^{\circ}\text{C}$), followed by incubation at 41.5°C ($\pm 1^{\circ}\text{C}$) for 24 h (± 6 h). The CEB was subcultured onto charcoal cefoperazone desoxycholate agar (CCDA) plates and incubated in a microaerophilic atmosphere at 37°C ($\pm 1^{\circ}\text{C}$) for 48 h (± 6 h). Presumptive positive colonies were confirmed by oxidase reaction, growth under microaerophilic conditions and determination of cell morphology using phase-contrast microscopy.

Data on human infections

Weekly reports (based on the date the specimen was received by the laboratory) of human *Campylobacter*

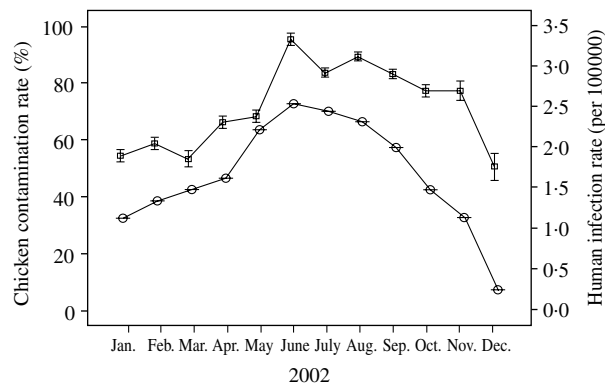


Fig. 1. Comparison of monthly human campylobacter infection and chicken *Campylobacter* isolation rates, with 95% confidence intervals, Wales, 2002. \square , Chicken isolate rate (%); \circ , human infection rate (rate per 100 000 population).

isolates in Wales were obtained from routine, laboratory-based surveillance that has complete coverage throughout Wales [11]. The report week was based on the date that the patient submitted the stool sample for primary isolation. Human *Campylobacter* isolates in Wales are not routinely speciated but since over 90% of those that are tested yield *C. jejuni*, all isolates were included in the study. Human infection rates were compared with *Campylobacter* isolation rates from chickens. Data were analysed by month and by week. In order to allow for random fluctuations in the numbers of positive isolates, weekly data series were smoothed using kernel density smoothing.

RESULTS

Altogether, 364 of the 514 (71%) fresh chicken samples were positive for thermophilic *Campylobacter* spp. and 2631 human campylobacter infections were reported during the 12-month sampling period. The mean number of chickens sampled per calendar month was 42.8 (range 20–54) and the mean number sampled per week was 10.5 (range 4–19). The estimated time period between slaughter and expiry of the use-by date was 7–10 days, indicating a sampling window of 7–10 days between packing (the final point of potential contamination) and removal from retail sale (expiry of use-by date).

The monthly data series for chickens and human isolates were similar, with a coincident peak in June (Fig. 1). However, weekly data indicate that the first peak in human infections slightly preceded that in chickens (week 24 compared to week 25) (Fig. 2). During the first half of the year approximately

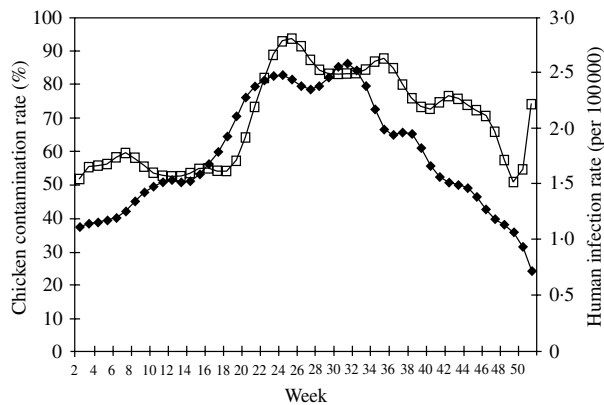


Fig. 2. Comparison of weekly human campylobacter infection rates and chicken *Campylobacter* isolation rates, Wales, 2002. Data subject to kernel density smoothing. □, Chicken isolate rate (%); ◆, human infection rate (rate per 100 000 population).

50–60% of chicken isolates were *Campylobacter* positive, but over a 6-week period from early May to mid-June contamination rates rose rapidly, peaking in late June (between weeks 24 and 26). They then stabilized at 80–90% until the end of August (week 35), when they slowly decreased again to approximately 50% by mid-December (week 50). Human infections increased slowly until early April (week 14), then increased at a faster rate reaching a peak late May to mid-June (between weeks 22 and 25). There was a secondary, and relatively higher, peak in human infections in late July and early August (between weeks 30 and 32), thereafter numbers of human isolates fell rapidly, mirroring the decrease in the chicken isolation rate.

DISCUSSION

Our findings indicate that the seasonal peak in human campylobacter infection in Wales in 2002 coincided with or preceded, rather than followed, the peak in retail chicken isolates. This suggests that the source of the excess in human infections at this time of year is not poultry but that both are associated with a common environmental source or reservoir.

By focusing on retail chicken and correlating weekly data on both human and chicken isolates we were able to analyse more precisely the relationship between human and chicken peaks. Our data do not take account of the time lag between exposure to infection and submitting a sample to the laboratory. This is probably around 10 days if we take into account the delay between exposure and onset of

symptoms (an incubation period of usually 2–5 days [12], and the delay between onset and receipt of the faecal specimen by the laboratory (median 6 days according to the Study of Infectious Intestinal Disease in England [13]). However, there is a similar time lag in chicken sampling that we estimate to be around 5 days, if we assume that chickens are distributed and made available for retail sale within 48 h of slaughter and packing. Therefore, if the graphs were redrawn to reflect estimated date of acquisition of infection in humans and slaughter date in poultry (the last point at which a carcass could become contaminated), the relationship between peaks in humans and chickens would diverge further with a peak in humans in week 22 and in chickens in week 24.

Another shortcoming in our data is that they do not distinguish indigenous human campylobacter cases from those acquired abroad, although only approximately 10% of Welsh cases are acquired abroad (Communicable Disease Surveillance Centre, unpublished data) and these present predominantly in summer and early autumn (accounting for some of the secondary peak between weeks 30–32) rather than at the time of the observed spring peak which, in 2002, was not associated with any public or school holidays.

Data from previous studies on the seasonality of campylobacter in poultry and humans leave many questions unanswered. In Finland, data indicate that the peak incidence in retail chicken occurs in late summer and that pulsed-field gel electrophoresis genotypes from chicken and humans are the same [14], but these findings cannot distinguish whether human infection is derived from poultry or whether there is a common environmental source. In Denmark, simultaneous seasonal increases in human and broiler chicken isolates have been observed in some years, but studies have not attempted to correlate them more closely or to examine contamination in retail chickens [9]. Data reported from Northern Ireland between 1995 and 2000 suggest that there is no temporal association between raw, retail chicken contamination and human infection, particularly during the seasonal peaks, but this observation was based on comparing quarterly data, rather than monthly or weekly isolates [10].

Infection in humans can be caused by a wide variety of factors, but there are none yet identified that specifically explain the seasonal peak in human infections during late spring or early summer. It may be that the seasonal increase in campylobacter contamination of retail chicken explains the increase in

human infections, but our observations provide evidence against a direct causal link between the peak in chickens and in human infections. An alternative explanation is that the peak in both chickens and humans is due to a common but unidentified environmental source or seasonal behavioural change.

Although *Campylobacter* spp. is ubiquitous in the environment, no correlation between possible common reservoirs, such as wild birds or fresh surface waters, and chicken colonization or human infection has ever been documented. Other previously unexplored environmental reservoirs should, therefore, be considered. The consistent seasonal rise at the same time each year suggests the presence of a very sensitive environmental stimulus such as the seasonal increase in hours of daylight that may reactivate latent *Campylobacter* cells. In order to corroborate our findings and explore these hypotheses, more detailed exploration of correlations between weekly human and animal *Campylobacter* isolation rates and hours of daylight at different latitudes are required. Case-control studies focusing on human cases during the peak season might also provide important clues about the source of infection.

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REFERENCES

1. Park RWA, Griffiths PL, Moreno GS. Sources and survival of campylobacters: relevance to enteritis and the food industry. *J App Biol* 1991; **70** (Suppl): 97S–106S.
2. Allos BM. *Campylobacter jejuni* infections: update on emerging issues and trends. *Clin Infect Dis* 2001; **32**: 1201–1206.
3. Altekruze SF. *Campylobacter jejuni* in foods. *J Am Vet Med Assoc* 1998; **213**: 1734–1735.
4. Altekruze SF, Stern NJ, Fields PI, Swerdlow DL. *Campylobacter jejuni* – an emerging foodborne pathogen. *Emerg Infect Dis* 1999; **5**: 28–35.
5. Studahl A, Andersson Y. Risk factors for indigenous campylobacter infection: a Swedish case-control study. *Epidemiol Infect* 2000; **125**: 269–275.
6. Hudson JA, Nicol C, Wright J, Whyte R, Hasell SK. Seasonal variation of campylobacter types from human cases, veterinary cases, raw chicken, milk and water. *J Appl Microbiol* 1999; **87**: 115–124.
7. Nylen G, Dunstan F, Palmer SR, et al. The seasonal distribution of campylobacter infection in nine European countries and New Zealand. *Epidemiol Infect* 2002; **128**: 383–390.
8. Bang DD, Nielsen EM, Knudsen K, Madsen M. A one year study of campylobacter carriage by individual Danish broiler chickens as the basis for selection of *Campylobacter* spp. strains for a chicken infection model. *Epidemiol Infect* 2003; **130**: 320–323.
9. Wilson IG. Salmonella and campylobacter contamination of raw retail chickens from different producers: a six-year survey. *Epidemiol Infect* 2002; **129**: 635–645.
10. Evans MC, Christiansen LE, Wedderkop A, Gerner-Smidt P, Wegener HC. Effects of climate on *Campylobacter* spp. in humans and broilers in Denmark. In: Abstracts of the 3rd International Conference on Emerging Infectious Diseases. Atlanta: 20–24 March 2002 (board 65). Atlanta, Georgia: Centers for Disease Control and Prevention.
11. Henry R. CoSurv: a regional computing strategy for communicable disease surveillance. *PHLS Microbiol Digest* 1996; **13**: 26–28.
12. Chin J, ed. Control of communicable disease manual, 17th edn. Washington, DC: American Public Health Association, 2000.
13. Food Standards Agency. Report of the study of infectious intestinal disease in England. London: The Stationery Office, 2000: 87.
14. Hanninen ML, Perko-Makela P, Pitkala A, Rautelin H. A three-year study of *Campylobacter jejuni* genotypes in humans with domestically acquired infections and in chicken samples from the Helsinki area. *J Clin Microbiol* 2000; **38**: 1998–2000.