Campylobacter infection of commercial poultry

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Summary
Campylobacter jejuni, a widespread food-borne pathogen is responsible for enteritis in the populations of both industrialised and developing nations and is acquired by consumption of contaminated water, milk and food products. Contaminated poultry meat is regarded as an important source of campylobacteriosis, with both commercial broiler and turkey growing flocks infected at two to three weeks of age by direct and indirect horizontal exposure. Non-chlorinated water is regarded as a vehicle of infection, followed by rapid intraflock dissemination. Intensification in the poultry industry has contributed to the increased prevalence rates on carcasses associated with increased stocking density and mechanised processing which are inherent to the high efficiency dictated by a competitive market. Currently, pre- and post-harvest control measures may ameliorate the problem of Campylobacter infection in consumers. Refrigerated storage and transport of red meat and poultry, appropriate handling and food preparation, and thorough cooking reduce the possibility of food-borne infection. In view of the world-wide distribution of C. jejuni infection and the multiplicity of sources, including non-pasteurised milk and contaminated water, it is inappropriate to impose trade restrictions on poultry meat based on the detection of campylobacters.

Keywords
Avian diseases — Campylobacter coli — Campylobacter jejuni — Food-borne infection — Post-harvest control — Poultry — Pre-harvest control — Prevalence.

Introduction
Campylobacter is responsible for food-borne enteric infection among consumers world-wide (32, 196). The infection may be acquired by consumption of non-chlorinated, contaminated surface water or water from wells (97), unpasteurised milk (181), and consumption of undercooked poultry (153) or red meat (172). In addition, campylobacteriosis may be acquired by direct contact with infected human shedders in the family environment. Nosocomial infection occurs and cases of congenital transmission are rarely documented. Campylobacteriosis in children is often acquired from immature diarrhoeic pets (31).

In the context of international trade, the ubiquitous nature of Campylobacter jejuni and the multiple reservoirs and sources of infection mitigate against impeding trade on the basis of contamination. Establishing an import barrier against poultry or red meat contaminated with Campylobacter would be unjustified. Invoking sanitary and phytosanitary measures would be blatantly protectionist and inconsistent with the rules of the World Trade Organization (100).

The characteristics of the thermophilic Campylobacter spp. of food-borne significance are reviewed in relation to isolation and identification, epidemiology in poultry and human populations, and current and future methods of control.

Isolation and identification of thermophilic Campylobacter spp.

The genus Campylobacter was established in the early 1970s (193), based on morphological and biochemical characteristics including serological typing (26). Subsequent developments in molecular biology have facilitated revision of the genus and differentiation from Helicobacter and Arcobacter (187).
The three thermophilic *Campylobacter* species of human health significance, *C. jejuni*, *C. coli* and *C. lari*, require selective media, incubation at 42°C and a microaerobic environment comprising a low level of oxygen (3% to 10%) and elevated carbon dioxide (1% to 10%). Methods of specimen collection to avoid desiccation, and subsequent culture and identification are reviewed in laboratory manuals (159). The three thermophilic species of *Campylobacter* can be differentiated by biochemical characteristics (167) and hydrogen sulphide production (105). The Penner serotyping scheme is based on heat-stable antigens derived from lipopolysaccharides (135). The alternative Lior serotyping scheme using heat-labile H antigens (106) is practical under laboratory conditions to differentiate among *C. jejuni* isolates derived from flocks and patients (131).

The relative efficiency of ten alternative methods to distinguish among *Campylobacter* isolates in epidemiological investigations was based on extensive studies undertaken at the United States Centers for Disease Control and Prevention, Atlanta (132). Techniques included Penner and Lior serotyping, multilocus enzyme electrophoresis, deoxyribonucleic acid (DNA) restriction endonuclease analysis, phage typing, plasmid analysis and ribotyping. Serotyping was determined to be the most discriminating phenotypic method, but all the procedures required specialised laboratory equipment and trained technicians consistent with reference centres. Pulse field gel electrophoresis is frequently applied to distinguish *C. jejuni* from *C. coli* and in molecular epidemiological studies (201). Flagella typing using restriction fragment length polymorphism (RFLP) analysis can discriminate among isolates and is regarded as a practical typing method for epidemiological investigations (124). Highly sensitive polymerase chain reaction (PCR) procedures are being developed to detect *C. jejuni* in food products (199). This has specific implications for regulations which impose a zero tolerance for *C. jejuni* on imported poultry, since the high sensitivity of this technique will detect the organism at extremely low prevalence. In a study conducted in Switzerland, *Campylobacter* was detected in 4% of a series of 231 litter samples using conventional microbiology, compared to 68% detection using PCR (175). In a trial conducted on faecal samples derived from hospitalised patients with enteric infections, the sensitivity and specificity of the PCR procedure as compared to conventional culture was determined to be 91% and 97% respectively (192). Alternative methods of detection and identification of *Campylobacter* include immunomagnetic separation and identification of pathogen-specific ions by mass spectrometry (114).

*Campylobacter jejuni* and *C. coli* produce a cytotoxic toxin which has immunological similarities to cholera toxin (109). This toxin is probably responsible for the diarrhoea associated with submucosal oedema noted in three- to four-day-old chickens inoculated with *C. jejuni* isolated from diarrhoeic patients (151). The cytotoxin produced by toxigenic strains of *C. jejuni* is dose dependent and is not neutralised by shiga-toxin immune serum. The toxin is regarded as a unique compound, lethal to HeLa and CHO cells (63) and chicken embryos (111).

In vitro assays involving adhesion and cytotoxicity have demonstrated that *C. jejuni* isolates from surface water are less pathogenic than strains derived from diarrhoeic patients (126). Pathogenic isolates are thought to develop the ability to colonise and to produce toxin as a result of passage in a susceptible host. This hypothesis was confirmed using a neonatal mouse model to demonstrate increased virulence following successive passage of *C. jejuni* isolates in chicks (150).

**Epidemiology of *Campylobacter jejuni* infection in commercial poultry**

Infection of commercial poultry, including ducks (93), broilers (140), turkeys (2), egg production flocks (55) and parent breeding stock (161) with thermophilic *Campylobacter* spp. is widespread (155). Of the three species, *C. jejuni* predominates, with *C. coli* and *C. lari* infrequently recovered from the intestinal tract of poultry. A review lists forty-eight reports of isolations from five species of poultry in thirty countries from 1981 to 1990 (153).

Experimental infection may induce mortality and transient diarrhoea in chicks following infection at one-day-old with a known enteroinvasive and pathogenic strain of *C. jejuni* (149). Subsequent exposure to the organism results in colonisation of the intestinal tract resulting in either watery dropings (125) or the absence of clinical signs (194). Neonatal infection with pathogenic strains of *C. jejuni* possessing virulence factors may produce focal hepatic necrosis and distention of the jejenum (39) or focal haemorrhage (194). Generally, flocks infected with *C. jejuni* show no clinical abnormality.

Oral infection results in colonisation of the distal jejenum, caecum and cloaca (39), with the organism located in the mucosal film. An outer membrane protein component, with a molecular weight of 69 kDa, is associated with colonisation (118). *Campylobacter jejuni* is attracted to L-fucose, a terminal sugar of the glycoprotein constituent of mucin (74). Infected broiler flocks excrete *C. jejuni* from the second or third week during the growing cycle (1). Prevalence rates among flocks vary, with values ranging from 17% to 90% in surveys conducted between 1984 and 1996, as documented in Table I.
Seasonal differences in prevalence rate can be detected, with higher recovery during summer compared to winter in Norway (90), the Netherlands (82), Sweden (24) and Yugoslavia (13), which is reflected in corresponding levels of contamination on processed broiler carcasses (198). Generally, intraflock transmission is rapid following introduction of infection. A field study showed an infection rate, based on cloacal isolation, increasing from 2% on the tenth day of the growing cycle to 80% on the twentieth day and persisting until the eightieth day (57). Data from commercial broiler processing plants in Israel confirm prevalence rates within flocks ranging from 58% to 100% of representative birds selected at slaughter. These field surveys are supported by the results of experimental infection which show rapid horizontal transmission of \textit{C. jejuni} among contacts (120, 163, 162).

Appropriate pre-harvest control is dependent on an understanding of the reservoirs of infection, mechanisms of transmission to flocks and the interaction of \textit{C. jejuni} with the host and commercial housing.

Contaminated water has been demonstrated to be a source of infection for flocks (146). Non-chlorinated water supplied to broilers has been implicated as a vehicle of transmission in Sweden (51), England (133) and Norway (92). The occurrence of viable but 'non-culturable' \textit{C. jejuni} in surface water may be significant in introducing infection onto farms (146). Open water receptacles, including troughs and suspended drinkers, contribute to intraflock dissemination of \textit{C. jejuni} infection (168). This observation is based on experimental studies demonstrating rapid horizontal transmission of \textit{C. jejuni} among contacts (120, 163, 162).

Wild birds are a potential source of \textit{C. jejuni}, with a 10% recovery rate from 445 cases representing 13 orders (205). Species most likely to introduce infection into commercial poultry flocks include passeriformes (11, 62) and columbiformes (205). Anseriformes (107) may contaminate surface water used to supply flocks (129).

Insects (90), and especially darkling beetles (82) (\textit{Alphatobius diaperinus}) and houseflies (\textit{Musca domestica}) (13, 198) may transmit \textit{C. jejuni}. The role of houseflies in transmitting \textit{C. jejuni} infection has been demonstrated under controlled conditions (156).

Rodents may serve as reservoirs of \textit{C. jejuni} (13, 24), although recent surveys in the United States of America (USA) have not detected \textit{C. jejuni} in trapped rats and mice on farms with infected flocks (62, 86). The role of rodents in introducing or perpetuating infection in successive broiler flocks with appropriate inter-cycle decontamination has not been defined.

The presence of domestic livestock on broiler farms has been implicated as a risk factor in infection of flocks with \textit{C. jejuni} (13, 92, 189). Recent studies, applying flagellin-A gene RFLP assays, have demonstrated a commonality among isolates obtained from the intestinal tract of broilers, houseflies, boots of farm personnel and cattle (174). Indirect mechanical transmission of \textit{C. jejuni} from cattle resident on a farm to successive broiler flocks, by farm personnel, wild birds, vermin, rodents and domestic pets is possible in the absence of appropriate biosecurity procedures and facilities to exclude wildlife.

Feed is not regarded as a source of infection because of the low moisture content and water activity below 0.8, which is inconsistent with survival of \textit{C. jejuni} (47). Broiler feed, which is generally pelleted, is subjected to pasteurisation temperatures expected to destroy \textit{C. jejuni} (77). Surveys have consistently failed to show the presence of \textit{C. jejuni} in broiler feed delivered to farms (13, 51, 93). Isolation of \textit{C. jejuni} from

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<th>Authors</th>
<th>Country</th>
<th>Prevalence rate (%)</th>
<th>Reference number</th>
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<tbody>
<tr>
<td>Prescott and Gellner</td>
<td>Canada</td>
<td>48</td>
<td>140</td>
</tr>
<tr>
<td>Altmeyer et al.</td>
<td>Germany</td>
<td>54</td>
<td>11</td>
</tr>
<tr>
<td>Engvall et al.</td>
<td>Sweden</td>
<td>17</td>
<td>51</td>
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<tr>
<td>Pokamunski et al.</td>
<td>Israel</td>
<td>31</td>
<td>138</td>
</tr>
<tr>
<td>Evans (1992)</td>
<td>England</td>
<td>50</td>
<td>52</td>
</tr>
<tr>
<td>Kjaipenud et al.</td>
<td>Norway</td>
<td>18</td>
<td>92</td>
</tr>
<tr>
<td>Humphrey et al.</td>
<td>England</td>
<td>76</td>
<td>77</td>
</tr>
<tr>
<td>Jacobs-Reitsma et al.</td>
<td>The Netherlands</td>
<td>82</td>
<td>83</td>
</tr>
<tr>
<td>Stern et al.</td>
<td>United States of America</td>
<td>90</td>
<td>173</td>
</tr>
<tr>
<td>Berndtson et al.</td>
<td>Sweden</td>
<td>27</td>
<td>24</td>
</tr>
<tr>
<td>Pearson et al.</td>
<td>England</td>
<td>36</td>
<td>134</td>
</tr>
<tr>
<td>Van de Giessen et al.</td>
<td>The Netherlands</td>
<td>57</td>
<td>189</td>
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feed in pans and troughs within a house has been documented (103). This is attributed to contamination by regurgitation, or introduction of litter or faeces into receptacles.

Fomites may be responsible for indirect mechanical transmission of C. jejuni, as determined by field surveys (13). Movement of personnel and equipment between breeder, broiler and turkey growing farms, associated with modern integrated production, may contribute to introduction of infection if clothing, boots and equipment are contaminated with moist faecal material from a flock excreting C. jejuni. Abiotic transmission is facilitated on multi-age farms or where units are in close proximity.

As C. jejuni is intolerant to desiccation (108), recovery from broiler litter is limited to substrate with a water activity value exceeding 0.85. Contaminated litter has been implicated in infection of flocks of broilers (57, 137) and turkeys (1). Campylobacter jejuni has been recovered inconsistently from the substrate of flocks excreting the organism (62). This suggests that litter is not a suitable medium for survey of C. jejuni infection in broiler flocks. The ability of contaminated litter to transmit C. jejuni under controlled experimental conditions was confirmed using Horsfall isolator units (120). The recovery of the organism from litter is a function of the water activity value of the litter, stocking density, techniques used to collect and transport samples, and methods of enrichment and isolation (159).

The results of numerous field studies generally disfavour the acceptance of vertical transmission of C. jejuni from breeding flocks to progeny via the egg under practical conditions (153). A survey of eggs derived from commercial egg-producing flocks, known to be faecal excretors of C. jejuni, failed to yield the organism from the shell surface or from homogenates of yolk or albumen. Contamination of the surface of shells with a faecal suspension of C. jejuni (1.4 x 10⁹ colony forming units [CFU/g]) resulted in shell penetration in 3/70 eggs and recovery from the contents of only 1/70 eggs (157). This study confirmed previous investigations which demonstrated that shell membranes serve as an effective barrier to penetration of C. jejuni from the shell to albumen (46). A concurrent study showed that survival of C. jejuni in albumen was limited to six hours. The organism could not be recovered from dead-in-shell embryos, or from the intestinal tracts of neonatal specific-pathogen-free chicks derived from eggs experimentally contaminated with C. jejuni (125).

Turkey pouls, brooded in an isolator unit, remained free of C. jejuni for twenty-one days, in contrast to commercially reared birds which excreted the organism by the fifteenth day, concurrently with the recovery of C. jejuni from the drinking water of the birds (2). Both cross-sectional and longitudinal studies of commercial egg-producing flocks and broilers in Sweden failed to demonstrate C. jejuni excretors in day-old chicks. The organism was recovered from the faeces of the laying-strain pullets at five weeks, but broilers remained free of infection through to slaughter at five weeks of age (103). Consistent with field experience, studies in Sweden showed that chicks derived from sixteen broiler flocks were free of C. jejuni at placement. In the case of eight flocks, excretion of the organism commenced at three weeks and persisted until slaughter at six weeks (51). A survey conducted in Australia failed to detect C. jejuni in 185/187 eggs derived from a breeder flock with a 74% prevalence of faecal excretion. Fourteen placements of broilers, derived from breeders known to be infected, were free of C. jejuni during a six-week growing cycle (161). In a parallel laboratory study, C. jejuni could not be recovered from 162 chicks hatched from eggs contaminated with a suspension of C. jejuni, suggesting that vertical transmission was unlikely under commercial conditions.

Field studies in Yugoslavia confirmed the observations made previously in Australia. Broiler chicks derived from known infected parent flocks (60% to 80% prevalence) were free of infection at day old but excreted C. jejuni when sampled at twenty-one days (13). A longitudinal study conducted in the Netherlands (188) demonstrated C. jejuni infection in one broiler flock, but C. jejuni was absent in six subsequent placements. Evidence against vertical transmission was predicated by the fact that parent flocks in the Netherlands are frequently infected (81) and that broiler placements are derived from eggs delivered to hatcheries from a large number of parent flocks (188). Freedom from infection in successive flocks was attributed to thorough intercycle decontamination. In a concurrent study, C. jejuni was isolated from seven consecutive broiler flocks. Penner serotyping and random amplification polymorphic DNA-typing denoted identical C. jejuni isolates, suggesting a common source of infection or residual infection in the poultry house.

Recently, the results of a number of investigations based on more sensitive assays for C. jejuni using molecular biological techniques have again raised the question of vertical transmission. Applying a DNA hybridisation procedure, investigators in Japan were able to demonstrate C. jejuni infection in day-old broiler chicks at the time of placement and over the following three-week period. Conventional microbiological assays with enrichment failed to detect the organism in cloacal swabs (38).

A recent study applying DNA sequencing of the variable region of the flagella antigen fla A gene confirmed that C. jejuni isolates from a breeder flock and the broiler progeny of this flock were identical (40). The fact that the farms were separated by a distance of 30 km suggests congenital infection, either vertically through the egg, or associated with infection during incubation, handling or delivery.
Previous studies have demonstrated the susceptibility of day-old chicks to infection with C. jejuni (149), especially by the intra-cloacal route (39). Horizontal transmission occurred rapidly among chicks in the hatching trays of commercial incubators and also in chick delivery boxes. Attempts at culture showed that two of fifteen samples of the water in the humidity pans in the hatcher were contaminated with C. jejuni. Given the high rate of air displacement by fans in hatchers, C. jejuni introduced into the environment of an incubator may be disseminated rapidly among the hatchlings. It was previously noted that a small proportion of eggs yield C. jejuni following experimental infection (46). Faecally soiled eggs, especially with damaged shells, which are subjected to incomplete decontamination by either disinfectant solutions or fumigants may introduce C. jejuni into incubators and the hatchery environment. This may result in infection of chicks at the time of pipping and thereafter, as the hatch is subjected to 37°C and 70% relative humidity for the holding period which frequently exceeds twelve hours, conditions which are conducive to survival of C. jejuni.

The recent series of DNA-based assays should be extended to define the mechanisms relating to possible vertical infection. To confirm this route of infection, the identification of a common serotype in parent flocks, eggs and progeny is required. The presence of the same gene for a flagella antigen, in both parent stock and progeny does not necessarily eliminate congenital infection through exposure in the hatchery environment, independently of direct vertical transmission through the egg.

Evaluating the results of field studies on commercial flocks, including a large number of prevalence surveys and laboratory experiments, the following conclusions are relevant to the epidemiology of C. jejuni infection in breeders, broilers and growing turkeys:

- Campylobacter jejuni is prevalent in all types of commercial flocks in all regions of the world where poultry is raised and where surveys have been conducted
- although the organism is sensitive to desiccation, intensive systems of production, especially in integrated operations, facilitate transmission to both floor and cage-housed flocks
- the major reservoirs of infection include the intestinal tracts of mature breeding flocks, commercial broilers and turkeys and replacement pullet flocks over three weeks of age
- the known routes of abiotic transmission include non-chlorinated surface water or water from wells, faecally contaminated clothing, footwear and equipment and exposure of a young flock to a contaminated environment
- rodents, vermin, insects, wild birds, domestic livestock and companion animals may serve as reservoirs or sources of infection
- conventional methods of sampling, followed by enrichment and culture may not be capable of isolating viable but non-culturable C. jejuni. Sophisticated DNA-based techniques will, in the future, contribute to a greater understanding of the molecular epidemiology of C. jejuni infection at the commercial flock level.

**Contamination of poultry meat with Campylobacter**

The high prevalence of Campylobacter on poultry meat and derived products is of significance to consumers (8). Records of the occurrence of C. jejuni and the less frequently isolated species of *Campylobacter* (C. coli, C. lari and C. fetus) during the period 1980 to 1990 are documented in a review of forty-two publications concerning seventeen countries and five poultry species (153).

Live broilers (128), turkeys (206) and ducks (93) are delivered to processing plants with high levels of faecal contamination (195). A study conducted in the USA confirmed that 20% of live broilers yielded *C. jejuni* from cloacal swabs obtained at the time of delivery to two plants (86). Unwashed transport coops may contribute to surface contamination of plumage and feet (71), resulting in recovery rates of 80% to 100% from the caecum for clinically healthy broiler flocks (195).

The practice in the USA of withholding feed from broiler flocks for periods of 6 h to 10 h to reduce contamination of carcasses with ingesta during evisceration, may exacerbate the introduction of *C. jejuni* contamination into plants via the crop. The recovery of the organism increased from 23% of 360 crops before feed withdrawal, to 62% at the time of harvesting. During an eight-hour period, levels of *C. jejuni* in the caeca of the subject birds remained constant (34). Subsequent studies using a fluorescent dye gavaged into the crop confirmed the extent of dissemination of ingesta among carcasses and the eviscerating environment (65, 66). Surveys conducted at three processing plants demonstrated cross contamination of carcasses during defeathering and evisceration, but a decrease in level of *C. jejuni* on the skin surface associated with scalding and immersion chilling (79). On-line washing of turkey carcasses with chlorinated water reduced levels of contamination (2).

A recent study of whole, processed packed and refrigerated carcasses and portions at point of sale yielded a Campylobacter recovery rate of 26%. The products were derived from five countries of the European Union (EU) with similar methods of flock management and processing (185). The values recorded in the study in Belgium are generally in agreement with surveys which yielded a 28% recovery rate in a survey conducted in Germany (15). Higher levels of *C. jejuni* were documented in a study in the USA using enrichment culture of samples derived from whole carcasses offered for sale. Recovery ranged from a low of 7% in December, to 97% in June and July, with an average annual rate of 64% among...
the thirty samples examined by whole carcass wash (198). Previous surveys over the period from 1980 to 1988 to quantify the levels of C. jejuni contamination on broiler carcasses reveal a generally high rate of recovery ranging from 14% to 88%, with an unweighted mean of 57% (Table II).

The recovery rate from carcasses may be influenced by the proportion of flocks infected, the degree of intraflock colonisation, seasonal and climatic factors, configuration and operation of immersion tanks and processing plant equipment, chlorination and chemical treatment of water and carcasses and microbiological techniques used for sampling, isolation and identification.

Quantification of the level of Campylobacter on carcasses, portions and derived products can be influenced by handling and storage (130). Freeze-thaw and heat stress injury following exposure to disinfectants or acids can lower recovery of C. jejuni, unless appropriate enrichment and isolation techniques are applied.

Campylobacter jejuni is relatively tolerant to freezing (64). A reduction of 0.5 to 2.0 log was recorded over a two-week period on broiler carcasses held at -20°C, with inoculation levels of $10^3$ to $10^5$ CFU/g. Viability of C. jejuni persisted on drumsticks contaminated at a level of $4.8 \times 10^2$ CFU/cm$^2$, for an extreme shelf life of ten days at both 9°C and -12°C. At -20°C, the level of C. jejuni declined from $9.9 \times 10^2$ CFU/cm$^2$ to $4.5 \times 10^2$ CFU/cm$^2$ in seven days, but persisted through the twenty-sixth week of storage with a terminal level of $0.2 \times 10^2$ CFU/cm$^2$ (203).

Campylobacter jejuni survived for up to twenty-eight days in vacuum-packed processed turkey rolls and hams held at 4°C (143). A statistically significant decrease was reported in the level of C. jejuni over time, and differences in viability were recorded among three isolates. The organism survived in sliced turkey roll under carbon dioxide enriched packaging for eighteen days at 4°C, confirming that processed poultry products may serve as a vehicle for infection.

Campylobacter jejuni infection on whole broiler carcasses is sensitive to cooking for 90 minutes at 190°C when subjected to moderately high levels of contamination corresponding to $10^6$ CFU/carcass. Some recontamination from mishandling of cooked carcasses was demonstrated when an inoculum of $10^6$ CFU was applied (59).

The potential of C. jejuni contamination of carcasses to be disseminated over hands and work surfaces was demonstrated in institutional kitchens surveyed in England (42). The organism was recovered from 88% of chilled and 10% of frozen broiler carcasses respectively, and from the kitchen environment (34%) and hands (4%) during preparation of chicken. In contrast, the environment was free of contamination before processing and after cleaning. A variety of Skirrow biotypes were identified on the carcasses which were recovered from hands and the kitchen environment. Similar results were obtained in the Netherlands where extensive cross contamination was demonstrated in a structured trial simulating transfer of C. jejuni from carcasses to work surfaces, raw vegetables and cooked products (43).

### Commercial table eggs

In contrast to the high prevalence of C. jejuni infection on poultry meat, extensive surveys have failed to demonstrate the potential pathogen in table eggs destined for consumption. Commercial hens known to be faecal shedders of C. jejuni did not produce infected eggs using conventional microbiological detection (157). A parallel study did not detect C. jejuni in 276 eggs derived from twenty-three farms in the State of New York (17). A survey conducted in two commercial egg processing plants did not demonstrate C. jejuni in eggs, derived products or from samples of water collected from the overflow of the egg washer (78). The investigators were able to detect C. jejuni from experimentally inoculated control specimens.

#### Table II

Recovery rate of Campylobacter jejuni from broiler carcasses

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<th>Authors</th>
<th>Country</th>
<th>Recovery (%)</th>
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<tr>
<td>Chowdhury et al. (1984)</td>
<td>India</td>
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<tr>
<td>Dawkins et al. (1984)</td>
<td>United Kingdom</td>
<td>68</td>
<td>42</td>
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<tr>
<td>Rosel et al. (1984)</td>
<td>Norway</td>
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<tr>
<td>Stern et al. (1984)</td>
<td>United States of America</td>
<td>30</td>
<td>171</td>
</tr>
<tr>
<td>Harris et al. (1986)</td>
<td>United States of America</td>
<td>56</td>
<td>67</td>
</tr>
<tr>
<td>Juven and Rogol (1986)</td>
<td>Israel</td>
<td>70</td>
<td>97</td>
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<tr>
<td>Humphrey and Lanning (1987)</td>
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<tr>
<td>Hood et al. (1988)</td>
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<td>70</td>
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<tr>
<td>Lammerding et al. (1988)</td>
<td>Canada</td>
<td>38</td>
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</tr>
<tr>
<td>DeBoer and Hahne (1990)</td>
<td>The Netherlands</td>
<td>61</td>
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</table>
Based on epidemiological studies involving numerous outbreaks of campylobacteriosis, eggs have not been identified as a primary source of infection. Accordingly, regulations aimed at preventing international movement of eggs on the basis of potential Campylobacter infection would be unjustified.

The relation between poultry and campylobacteriosis in humans

Incidence of Campylobacter infection in humans

The United States Department of Health, Centers for Disease Control and Prevention has recently completed an extensive survey of food-borne disease in the USA (117). Figures were collected from ten national and regional databases including the Food-borne Disease Active Surveillance Network ('Food Net') established in 1996. An estimate of the incidence of campylobacteriosis in 1998 was based on active surveillance among a population of 20 million. The incidence rate from 1996 to 1997, of 24/100,000 was extrapolated to the entire population of the USA following application of a multiplication factor of thirty-eight to represent the proportion of non-reported to diagnosed cases. The total estimated number of cases in the USA exceeded 2.5 million, with 13,000 hospital admissions and 124 deaths in 1997. Campylobacteriosis represented 14.2% of all diagnosed food-borne infections including bacterial, viral, protozoal and metazoal aetiologies, and exceeded paratyphoid salmonellosis (9.7%) in incidence. The most recent compilation of data on food-borne Campylobacter infections in the USA updates previous reports on occurrence and causation of outbreaks covering the period from 1973 to 1992 (20, 21, 179). The campylobacteriosis incidence rate in the USA of 1,020/100,000 population, estimated in 1992 (176), is strongly supported by the latest, more structured evaluation.

The differential between diagnosed and non-reported cases of campylobacteriosis complicates estimates of economic losses associated with infection. Based on incidence rates and hospital records pertaining to the mid-1980s, the direct and indirect cost of the disease ranges from US$700 million to US$1,400 million (121). A comparative value of US$150 million was estimated for the United Kingdom (UK), based on an incidence rate of 1,100/100,000 and prevailing medical costs in that nation (166).

Epidemiology of Campylobacter infection in humans

A recent review of C. jejuni infection as a food-borne disease, provides a perspective of the history, epidemiology and prevalence of the condition in human populations (10), supplementing information contained in earlier reviews (136, 154, 177). Campylobacter jejuni is responsible for over 95% of the diagnosed cases of campylobacteriosis, whilst C. coli and C. lari are occasionally isolated from cases of haemorrhagic enteritis in industrialised countries which maintain appropriate surveillance systems (179). Campylobacter coli represented 19% of the isolates in a survey in Portugal (35), 11% in Singapore (101) and 35% in Yugoslavia, with a predominance of this species from patients in rural areas (139).

Following the first recognised cases of enteritis attributed to 'related vibrios' in 1952 (94), an association was recognised between infection and either the consumption of contaminated food products of animal origin or direct contact with livestock. Structured investigations were later facilitated by pivotal advances in isolation, culture and identification of the 'related vibrios' from faeces (44). By the late 1970s, Campylobacter enteritis was recognised as an emerging food-borne disease (165). At this early stage in the understanding of the disease, the recognised risk factors included close contact with domestic flocks or processing of poultry, handling diarrhoeic companion animals or foreign travel by residents of urban areas of industrialised countries in the northern hemisphere (142).

The first documented outbreak of campylobacteriosis which was directly attributed to consumption of chickens occurred in the Netherlands among a group of cadets who experienced an 80% attack rate (28). Subsequent surveys confirmed the high prevalence of C. jejuni in the faeces of patients and on the carcasses of broilers and turkeys (61, 127). The introduction of serotyping schemes (106, 135) and biotyping (105) facilitated epidemiological studies (110) to establish relationships between sporadic outbreaks in communities and consumption of processed poultry in Australia (160), the UK (85), the Netherlands (18), Yugoslavia (13) and Germany (200).

Campylobacteriosis may be regarded as an occupational infection of processing plant workers (60). A single outbreak was documented in a poultry abattoir in Sweden, affecting thirty-seven workers of whom 71% were young, untrained temporary summer labourers (37). A comparison of titres in a survey in the UK revealed that 27% to 68% of workers in poultry and red meat plants demonstrated antibodies to C. jejuni compared to 3% among field labourers (84). A survey conducted in Italy confirmed antibody titres to C. jejuni in 12% of abattoir workers, compared to 2% in a cohort of blood donors (113).

Young adults, and specifically male college students (123), demonstrate high incidence rates for Campylobacter infection, approaching 15/100,000 during the mid-1980s (178). An outbreak at the University of Victoria, British Columbia, Canada, in the autumn of 1984, involved an attack rate of 30% among 1,076 students. Salads and fried chicken were implicated as vehicles, with evidence of mishandling and
improper storage, preparation and serving of food (4). A subsequent case control study conducted at the University of Georgia, USA, identified the consumption of undercooked and raw chicken and contact with a cat as risk factors for infection (odds ratio: 9), resulting in an overall C. jejuni isolation rate of 24/10,000 students per academic quarter of fifty days (45).

Contact with raw chicken during food preparation is regarded as a significant risk factor for campylobacteriosis among consumers (72, 91). Raw poultry carcasses and portions and seepage during thawing frequently contaminate hands, storage areas, work surfaces and utensils, leading to transfer of C. jejuni to salads and other non-cooked foods. Improper and unhygienic procedures during storage and preparation (29, 68) contribute to outbreaks of campylobacteriosis in catering and institutional units (9) and also in domestic kitchens. Educational initiatives undertaken by the Food Safety and Inspection Service of the United States Department of Agriculture (USDA) address the safe handling of poultry meat (184).

Children are at risk of campylobacteriosis, based on age-prevalence data. An incidence rate of 36/100,000 was determined for infants in the USA in 1997 (14). Reports from various countries confirm the susceptibility of children, including Taipei China (102), the USA (180), Mexico (186), Chile (54), Guatemala (41), Peru (122), Singapore (101), Portugal (35), Israel (152), Liberia (119), Nigeria (3), South Africa (144) and Bangladesh (25). The high rates recorded in surveys conducted in non-industrialised countries reflect deficiencies in sanitation, hygiene, housing, consumption of unchlorinated water, unpasteurised milk and non-refrigerated food, and contact with domestic livestock including free-ranging chickens (116). In industrialised countries, contact with diarrhoeic pets, food-borne infection and direct contact transmission have been reported to occur in day-care centres employing suboptimal procedures for food handling and hygiene (19).

Immunosuppressed patients, including those infected with human immunodeficiency virus (HIV), are extremely susceptible to campylobacteriosis, especially when concurrent exposure to opportunistic fungal and protozoal pathogens occurs (23). Isolation rates of C. jejuni increase with age. Data for the period 1982-1986 in the USA confirm a rise from 1/10^5 in the age range 40-45 to 5/10^5 in the 60-65 year cohort. The frequency of isolation of C. jejuni from blood increases exponentially with age (180).

**Antibiotic resistance**

The isolation of antibiotic resistant strains of *Campylobacter* from poultry represents an emerging public health problem. Plasmid-mediated resistance to tetracycline was demonstrated in Canada in 1983. The studies revealed a close relationship between isolates from humans and domestic animals (182). The occurrence of erythromycin resistant strains of *C. jejuni* was documented in Israel, using the agar dilution technique which was considered superior to the less sensitive disc susceptibility method (145). A survey of the antibiotic sensitivity of twenty-one *C. jejuni* isolates from healthy chickens in Canada showed resistance to sulphonamides and bacitracin (both intrinsic), streptomycin, tetracycline and penicillin G (27). The isolates were all susceptible to erythromycin, kanamycin and ampicillin.

The mechanisms of antibiotic resistance in *Campylobacter*, and the prevalence of resistant strains was comprehensively reviewed during the late 1980s (183). *Campylobacter jejuni* strains were noted to be highly susceptible to quinolones, but plasmid-mediated resistance to tetracyclines and aminoglycosides (in the case of *C. coli*) was documented.

A significant paper confirming a parallel increase in quinolone resistance of *C. jejuni* isolates from human patients and from poultry was attributed to the extensive use of enrofloxacin in the production of broilers in the Netherlands. During the period between 1982 and 1989, quinolone resistance in human isolates increased from 0% to 11.5%. During the same period, poultry isolates increased in resistance from 0.5% to 14% (50). In contrast, a survey in Sweden showed no increase in resistance to antibiotics used therapeutically for gastroenteritis among isolates of *C. jejuni* from patients during the period 1978 to 1988. In 1989, 14% of *Campylobacter* isolates were resistant to quinolones, with corresponding values of 7.3% and 12.4% for erythromycin and doxycycline, respectively (164).

A study on quinolone resistance of species of *Campylobacter* derived from poultry abattoir effluent and sewage plants was conducted in the Netherlands in 1995. Of the isolates derived from the poultry plant outflow, 28% were resistant to quinolones. In contrast, 11% to 18% of isolates from a sewage treatment plant were resistant to quinolones. A second plant receiving effluent from various sources, including a poultry abattoir, yielded 17% to 33% quinolone resistant *Campylobacter* (95). The difference between the Netherlands and Sweden with respect to prevalence of quinolone resistance was attributed to the patterns of drug use in poultry in the respective countries.

During the period from 1990 to 1994, a study in Spain demonstrated that resistance to quinolones increased from 45% to 88% of *C. jejuni* isolates obtained from patients. During the same period, the proportion of isolates resistant to chloramphenicol, amoxycillin and tetracycline remained constant (148). Quinolone resistance in *Campylobacter* involves a single point mutation around residues Ser 83 and Asp 87, located near the N terminus of the gyrase A subunit (197). Inappropriate and
excessive administration of antibiotics, including quinolones, to poultry is regarded as a major source of drug resistant *C. jejuni*. In the USA, where quinolones have been licensed for restricted therapy of poultry since 1995, under strict veterinary supervision and employing the 'principles of prudent use' of the Food and Drug Administration, ciprofloxacin resistant *C. jejuni* can be recovered from processed broilers (10).

**Autoimmune conditions following campylobacteriosis**

During the 1990s, epidemiologists confirmed a relationship between infection with *C. jejuni* and post-recovery autoimmune conditions. These include Guillain-Barré syndrome (GBS) (80), Fisher’s syndrome, a variant of GBS (96), and Reiter’s syndrome, a non-purulent reactive arthritis (48).

Guillain-Barré syndrome is an acute neuro-muscular paralysis associated with an inflammatory demyelinating polyneuropathy. Following an increase in anecdotal reports on the possible relationship between *C. jejuni* infection and subsequent GBS (22), surveillance studies established a serological basis for the association (7). A case-control study determined a significantly higher antibody titre in patients with GBS, compared to fifty-five controls (odds ratio: 12.5 with a 95% confidence interval of 0.6 to 33) in five centres in the USA during the summer months of 1983 to 1990. Similar results were obtained from a field study conducted in the north of the People’s Republic of China, which showed a statistically significant difference (*P* = 0.001) in *C. jejuni* titre in patients with GBS (66%), compared to controls (16%) (69). Microbiological studies have confirmed the presence of *C. jejuni* in patients at the time of onset of GBS (3). According to estimates, 30% to 40% of GBS cases are preceded by *C. jejuni* infection, and one case of GBS follows 1,050 *C. jejuni* cases (33).

Microbiological studies have confirmed the presence of *C. jejuni* in patients at the time of onset of GBS (3). According to estimates, 30% to 40% of GBS cases are preceded by *C. jejuni* infection, and one case of GBS follows 1,050 *C. jejuni* cases (33). The lipopolysaccharides of *C. jejuni* and *C. coli* have been shown to stimulate an inappropriate immune response to the gangliosides GM1 and GD1b incorporated in the myelin sheath of peripheral nerves. This mechanism may be common to infection with Epstein-Barr virus, cytomegalovirus and *Mycoplasma pneumoniae*, which are also associated with GBS (80).

The annual economic impact of GBS in the USA was calculated to be US$0.2 to US$1.8 billion on the basis of 3,000 to 10,000 reported cases. Between 500 and 3,500 cases of GBS were assumed to be initiated by *C. jejuni* infection (33).

Reactive arthritis and associated conjunctivitis and stomatitis occur following *C. jejuni* infection in patients demonstrating HLA-B27 antigen (191). No reports have been published on the incidence or cost of autoimmune arthroses following food-borne *C. jejuni* infection.

**Reduction of *Campylobacter jejuni* contamination in poultry meat**

**Pre-harvest control of *Campylobacter***

The amelioration of *C. jejuni* contamination must be based on control during both the pre-harvest and processing components of the chain of production.

If confirmation is obtained that vertical transmission of campylobacteriosis can occur from parent flock to broiler progeny, the implementation of programmes to limit the introduction of infection into grandparent and parent level breeder flocks will be necessary. Intensification of biosecurity to suppress *Salmonella* should also reduce exposure to *Campylobacter*, as many of the mechanisms of transmission are common to the two organisms. The biosecurity precautions appropriate to breeding farms have been reviewed (158) and incorporate both structural and operational procedures. Poultry houses should be designed and constructed to eliminate the entry of rodents and wild birds which are reservoirs of infection. Showering of personnel, provision of clean clothing and footwear, and placement of disinfectant boot dips, are basic procedures which can reduce the probability of introduction of infection.

Although feed is not regarded as an important vehicle for *Campylobacter*, pelletisation with heat pasteurisation and the addition of organic acids will effectively eliminate infection in feed.

The introduction of mechanical egg collection eliminates the need to use nest litter which is often associated with faecal contamination of shells. Frequent collection of eggs using a mechanical installation with self-cleaning belts, followed by decontamination after collection using either formalin fumigation or a phenolic disinfectant, will reduce the probability of mechanical transmission of *Campylobacter*.

Since breeding stock is intrinsically valuable, with each female potentially capable of producing 130 broiler chicks, expenditure on competitive inhibition cultures is justified. As water has been demonstrated to be an important source of *Campylobacter*, chlorination of the central supply at a level of 2 ppm to 3 ppm is strongly recommended (146). Removal of the biofilm in water supply lines using frequent cycles of flushing will reduce the potential for infection. Recent studies implicating domestic livestock, including cattle, as reservoirs of infection, suggests that farms should be securely fenced to exclude food animal and companion species.
The hatchery is a potential link in the chain of transmission from breeder flock to broilers, and accordingly, intensification of biosecurity procedures and decontamination should be emphasised in a control programme appropriate for an integrated poultry producer (158).

Litter additives, including sodium bisulphate (112), acidify the upper 5 cm to 10 cm of substrate and may reduce exposure to Campylobacter by eliminating sporadic introduction of low grade infection by defects in biosecurity. Field trials of sodium bisulphate, added to litter at 2 kg to 3 kg/10 m², delayed the onset of infection in flocks, compared to controls on untreated litter. Litter treatment did not prevent infection in thirty-five-day-old broilers which were assayed at processing (56). Intensifying biosecurity will reduce but not eliminate the possibility of introduction of infection (190).

Applying competitive exclusion cultures, immunological adjuvants and stimulants, and dietary supplements can reduce the prevalence and intensity of Campylobacter colonisation in broilers under controlled laboratory conditions (16). Trials with undefined caecal cultures have confirmed the studies on the 'Nurmi effect' (141) and subsequent work conducted in the USA (169) and the UK (76) in which inhibition of colonisation, but not absolute eradication of Campylobacter, could be achieved. The weight of literature suggests that competitive inhibition is a more effective mechanism against Salmonella than Campylobacter (104, 170).

Rearing broilers on plastic mesh to eliminate the possibility of coprophagy offers some potential in eliminating food-borne intestinal pathogens. Technical, financial and animal welfare restraints limit the possibility of applying off-litter growing as a means of eliminating Campylobacter infection in commercial broilers.

Currently, no vaccines are available for commercial control of Campylobacter in live parent stock or progeny. Although studies have been undertaken on the immunology and molecular biology of Campylobacter jejuni, products which are both effective and economically feasible have yet to be developed.

Post-harvest control of Campylobacter

Procedures to reduce the amount of Campylobacter infection entering processing plants on live broilers should be implemented. Thorous disinfection of coops and transport modules will reduce interflock contamination which may occur with partial flock depletion programmes.

Since introduction of mandated hazard analysis and critical control point (HACCP) programmes in the USA to reduce microbiological contamination of poultry and red meat, a significant decrease has occurred in contamination of carcasses with Salmonella (115).

This decrease is largely attributable to an increase in water utilisation for overflow of scalders, immersion chillers and improved 'inside-outside' spray washers. Concurrently, most processing plants have increased the levels of chlorine in immersion tanks and spray washers to achieve a reduction in Salmonella recovery from 20% of carcasses to a national average of 10% to 12%. Initial surveys conducted by the USDA Food Safety and Inspection Service and individual companies suggest that methods to reduce Salmonella contamination have not resulted in a corresponding decrease in the recovery of Campylobacter, which remains at high levels.

Immersion scalding is associated with a reduction in the level of C. jejuni on broiler carcasses providing the temperature of the water is maintained above 50°C at a pH range of 8 to 9. Additional reduction in the level of C. jejuni can be achieved by adding a quaternary ammonium disinfectant to water used for scalding, at a level of 50 ppm to 100 ppm (73). Concurrent studies showed that a reduction in the number of C. jejuni from 80 to 100 organisms/ml is possible, but with no effect on carcass contamination (75).

The introduction of an antimicrobial additive into 'inside-outside' bird washers can reduce the level of Salmonella on carcasses, and presumably also decrease C. jejuni levels. Trials conducted in the USA show that 0.5% cetylpyridinium chloride, 10% trisodium phosphate, 5% sodium bisulphate and 2% lactic acid are active against Salmonella when applied at a temperature of 35°C and a pressure of 400 kPa for 60 seconds. Of the range of chemicals, 0.5% cetylpyridinium chloride was the most effective (202). Trisodium phosphate is widely used as an antimicrobial rinse and is approved for application in poultry plants in the USA. Statistically significant reduction in the recovery rate of C. jejuni from carcasses has been documented under practical conditions. Application of trisodium phosphate in a commercial carcass washer (level not stated but presumed to be 10%) reduced Campylobacter levels from a prewash rate of 78% of carcasses examined to 46%. By comparison, Salmonella levels were reduced from 30% to 1% and Escherichia coli from 96% to 1%, confirming the greater susceptibility of these organisms to trisodium phosphate compared to Campylobacter (49). Previous studies on disinfectants have demonstrated the efficacy of 0.5% lactic and acetic acids, and chlorine at 100 ppm (204). Recent studies have focused on practical aspects of application and evaluation under operating conditions at line speeds exceeding 9,000 birds per hour. Chemical treatment is subject to approval by regulatory authorities in the USA and the EU. Registration requires data demonstrating the absence of either mutagenic or toxic effects of chemicals and to ensure that at practical levels, equipment is protected from damage and that organoleptic properties of poultry products are not compromised (153).
Gamma irradiation ('cold pasteurisation') is an effective method of eliminating *C. jejuni* from poultry meat and products (89). Experiments have established $D_{10}$ values of 0.3 kGy for *C. jejuni*, suggesting that a 2 kGy dose would effect a six log reduction in levels of *C. jejuni* (98). The literature on ionising radiation in relation to poultry supports the finding of the joint Food and Agriculture Organization/International Atomic Energy Agency/World Health Organization Committee, which established a 10 kGy level to eliminate *Salmonella* and other bacterial food-borne pathogens in poultry meat.

A petition to the Food and Drug Administration of the USA to amend the appropriate regulation (21 CFR 179.26) to allow irradiation of poultry products was filed in late 1986. Following approval by this agency, in 1990, the USDA Food Safety and Inspection Service developed specific regulations relating to procedures, controls and labelling to allow for irradiation at a level ranging from 1.5 kGy to a maximum dose of 3.0 kGy. Irradiation of poultry meat is not approved for use in all countries.

Feasibility studies on cobalt-60 isotope irradiation demonstrated the need to invest US$2 to US$4 million in a free standing plant which could irradiate poultry and other food products at a cost ranging from US$0.05 to US$0.1 per kg, depending on volume of utilisation, the interest rate and local factors (30). The development of electron beam technology will facilitate the adoption of 'cold pasteurisation', since the elimination of isotopes and the lower capital cost of an installation may represent a more acceptable alternative for poultry producers.

Despite adverse publicity by opponents of food irradiation, no evidence suggests a widespread consumer rejection of irradiated poultry (58). Trials conducted at the University of Georgia confirmed that education is an important motivator to accept 'cold pasteurised' breast and thigh portions. Over 80% of informed consumers accepted that 'cold pasteurisation' reduced the risk of food-borne infection. Half the subjects were willing to pay a 5% premium for treated product. Current information relating to food preservation using ionising radiation including packaging, and the sensitivity of bacteria including *Campylobacter* have been extensively reviewed (12, 53). Acceptance of 'cold pasteurisation' by both producers and consumers will be a necessary requirement for the widespread use of this technology. Establishing that poultry meat subjected to ionising radiation is both wholesome, nutritious and free of bacterial food-borne pathogens will encourage the commercialisation of the process.

At present, *Campylobacter* contamination of poultry products can be ameliorated by a combination of pre- and post-harvest procedures. Poultry products should be stored and transported at a temperature of 4°C or lower to prevent proliferation of *Campylobacter* and other food-borne bacterial pathogens. Appropriate precautions in handling and preparation of raw poultry, decontamination of utensils and work surfaces, and thorough cooking in accordance with health codes should reduce the risk to consumers.

**Significance for international trade**

*Campylobacter jejuni* and *C. coli* are widely distributed among commercial broiler, turkey and commercial egg-producing flocks in all countries where commercial poultry industries operate. The frequency of recovery of *Campylobacter* from broiler and turkey carcases and portions is a function of diagnostic intensity. Commercial table eggs are not involved in transmission of *Campylobacter* infections from caged flocks to consumers. In view of the widespread distribution of *Campylobacter* infection in foods derived from mammalian livestock and poultry, and the presence of the organism in untreated water, imposition of trade restrictions on poultry meat following recovery of *C. jejuni* or *C. coli* would be inappropriate.

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**Infection à *Campylobacter* chez les volailles commerciales**

S.M. Shane

**Résumé**

*Campylobacter jejuni*, largement répandu et transmis par les aliments, est à l'origine de cas d'entérite chez l'homme dans les pays développés et en développement. L'infection survient après consommation d'eau, de lait ou d'aliments contaminés. La viande de volaille contaminée est considérée comme une source importante de campylobactériose ; les poulets de chair et les dinde issus de l'élevage industriel s'infectent par exposition horizontale directe et
indirecte à l’âge de deux ou trois semaines. L’eau non chlorée est considérée comme une source de contamination, celle-ci se disséminant ensuite rapidement au sein de l’élevage. L’intensification de l’aviculture industrielle entraîne une présence accrue de la bactérie dans les carcasses, du fait de l’accroissement des capacités de stockage et de l’automatisation des processus de transformation, indispensables pour atteindre l’efficacité optimale requise par un marché de plus en plus concurrentiel.

Actuellement, des mesures prophylactiques avant et après abattage peuvent contribuer à protéger le consommateur contre l’infection à *Campylobacter*. Le stockage et le transport réfrigérés des viandes rouges et des volailles, la manipulation et la préparation d’aliments dans des conditions appropriées ainsi qu’une cuisson suffisante permettent de réduire les risques d’intoxication alimentaire. Compte tenu de la répartition mondiale de l’infection à *C. jejuni* et de la multiplicité des sources de contamination, y compris le lait non pasteurisé et l’eau, il serait vain d’imposer des restrictions aux échanges de viande de volailles sur la base de la détection de campylobacters.

**Mots-clés**

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**Infección por *Campylobacter* de aves de corral de explotaciones comerciales**

S.M. Shane

**Resumen**
La infección por *Campylobacter jejuni*, organismo transmitido por vía alimentaria, se contrae por consumo de agua, leche o alimentos contaminados y da lugar a brotes de enteritis entre las poblaciones de países tanto industrializados como en desarrollo. La contaminación de aves, que afecta sobre todo a los pollos asaderos y pavos de dos a tres semanas de edad de las explotaciones comerciales, infectados por exposición horizontal directa o indirecta, se considera una importante fuente de campilobacteriosis. Se piensa que el agua no clorada constituye un importante vehículo de la infección, que después se propaga rápidamente en el interior de las bandadas. La intensificación de la producción avícola, asociada a la mayor densidad de almacenamiento y a la mecanización de los sistemas productivos inherentes al elevado rendimiento que exige un mercado muy competitivo, han contribuido a elevar las tasas de prevalencia en las aves.

En las circunstancias actuales, la aplicación de medidas de control previas y posteriores al sacrificio puede paliar el problema que supone la infección por *Campylobacter* para los consumidores. El transporte y almacenamiento de carne roja y aves de corral en instalaciones refrigeradas, la manipulación y preparación correctas de los alimentos y su cocción completa reducen las probabilidades de infección alimentaria. Habida cuenta de la distribución mundial que presenta la
infección por *C. jejuni* y de la multiplicidad de fuentes (entre ellas la leche no pasteurizada y el agua contaminada), no resulta indicado imponer barreras comerciales a las aves basadas en la detección de *Campylobacter*.

**Palabras clave**


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**References**


