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Campylobacter jejuni: Incidence in Processed Broilers and Biotype Distribution in Human and Broiler Isolates

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Campylobacter jejuni was isolated from 18 of 40 processed broiler carcasses and 134 of 327 cloacal swabs obtained at four processing plants in Sydney, Australia. Three of four flocks examined carried *C. jejuni*. Eighty-two percent of chicken and 98% of human isolates from the area were of identical biotypes.

Pathogenic bacteria of the genus *Campylobacter* have been identified in up to 14% of patients with acute gastrointestinal symptoms (2). Domestic pets (4), farm animals (15), poultry (2, 10), and sea- and freshwater (6) have been reported to be reservoirs of *C. jejuni*. Outbreaks of *Campylobacter* infection have succeeded the consumption of unpasteurized milk (9). Circumstantial evidence has implicated undercooked poultry in small-group or family infections (1, 10). Contaminated water has been implicated in a large outbreak in Vermont (18). Studies in the United States (5, 17) and the United Kingdom (3, 7, 11) have established that *C. jejuni* was present in the gastrointestinal tracts of 1.8 (17) to 91% (7) of broilers. There is no published data on the incidence of *C. jejuni* in poultry in Australia. One aim of this study was to determine the incidence of *Campylobacter* sp. in commercially processed broilers in Sydney. Surface contamination of processed carcasses and interflock variation in cloacal carriage within a locale were also assessed.

Poultry and cattle isolates have been reported to share characteristics of human isolates of enteropathogenic *Campylobacter* sp. (13). Skirrow and Benjamin (14) have proposed a simple biotyping scheme that includes *C. fetus*, *C. coli*, two biotypes of *C. jejuni*, and a possible new species, nalidixic acid-resistant thermophilic *Campylobacter*. One hundred and sixty-five thermophilic *Campylobacter* sp. of human and chicken origin were obtained from Sydney and biotyped with the method of Skirrow and Benjamin (14).

Broiler samples were collected from four processing plants handling chickens from separate grower farms within a 15-mile (24.14-km) radius. In commercial processing, broilers are slaughtered, scalded, mechanically plucked, eviscerated, washed, chilled, and packaged. Random cloacal swabs were obtained from 327 carcasses before the evisceration procedure. At the same time, 10 chilled, freshly packaged, whole carcasses of similar size were selected from each plant. Cloacal specimens were placed in transport medium (Medical Wire Equipment Co. Ltd., Corsham, Wilts, England), stored at 5°C, and processed at the laboratory within 2 h of collection. Carcasses were stored at 5°C and transported immediately to the laboratory. Cloacal swabs were plated onto Skirrow selective medium (12; Oxoid BA base no. 2, 7% lysed horse blood, 10 μg of vancomycin per ml, 2.5 IU of polymixin B sulfate per ml, 5 μg of trimethoprim lactate per ml), and the plates were incubated at 42°C for 48 h under microaerophilic conditions (5% O_2, 10% CO_2, 85% N_2). Each carcass was surface rinsed by shaking in polyethylene bags containing 300 ml of 1% peptone water. Samples of 0.1 ml of rinse solution were plated in duplicate on Skirrow medium, and the plates were incubated as described above. Typical colonies were identified and counted. The growth of one colony on the selective plate represented a count of 1.5×10^3 colony-forming units per carcass.

Forty-six isolates of thermophilic *Campylobacter* sp. were obtained by coproculture from patients with enteritis. The cultures were collected during a 12-month period and stored at −70°C until biotyped.

Retail chicken outlets serving this nonindigent patient population were predominantly supplied by the plants sampled in this study.

Biotyping was performed on all human isolates and 119 broiler isolates. Cultures morphologically characteristic of *Campylobacter* sp. were tested for the production of catalase and oxidase, temperature tolerance, nalidixic acid sensitivity, H_2S production, and hippurate hydrolysis. These tests differentiated *C. fetus*, *C.*

TABLE 1. Recovery of *C. jejuni* from broilers at four processing plants

Processing plant	Cloacal isolations[a]	Processed carcass contamination[a]	
	No.[b] %	No.[b] %	*C. jejuni* count, per carcass
A	11/11 (100)	8/10 (80)	$(1.5–4.5) \times 10^3$
B	71/102 (70)	10/10 (100)	$1.2 \times 10^4–4.8 \times 10^6$
C	0/105 (0)	0/10 (0)	$<1.5 \times 10^3$
D	52/109 (52)	0/10 (0)	$<1.5 \times 10^3$

[a] Samples from a single flock were taken at each processing plant.

[b] Number of positive cultures/number of samples.

jejuni biotypes 1 and 2, *C. coli*, and a nalidixic acid-resistant group of thermophilic *Campylobacter* sp. (13, 14).

Thermophilic *Campylobacter* sp. were isolated from 134 of 327 (41%) of the cloacal swabs and 18 of 40 (45%) of the processed carcasses. The results from individual processing plants are summarized in Table 1. The mean *C. jejuni* count per contaminated carcass was 8.6×10^4. No *C. coli*, nalidixic acid-resistant thermophilic *Campylobacter* strains or thermotolerant *C. fetus* (16) were isolated from chicken or human sources. Of 119 chicken isolates, 98 (82%) were identified as *C. jejuni* biotype 1, compared with 45 of 46 (98%) of human isolates. Eighteen percent of chicken and 2% of human isolates were identified as *C. jejuni* biotype 2.

The incidence of *C. jejuni* in broilers has been reported to range from 1.8 (17) to 83% (5) in the United States and 14 (11) to 91% (7) in the United Kingdom. The variations may be due to differences in sample size, isolation methodology, or variation in flocks from different localities, or all of these factors. Our data indicate that carriage of the organism occurs in discrete flocks and that the incidence varies within a locale. This has not been previously reported.

C. jejuni was detected in levels up to 4.8×10^6 colony-forming units per carcass. The clinical significance of these results is not clear since the minimal infective dose for humans has not been determined. However, experimental infection in humans has been induced by the ingestion of 500 organisms of a *C. jejuni* strain from a milkborne outbreak (8). Further studies are required to determine the public health risk associated with *Campylobacter* sp. contamination of commercially available chickens.

Our finding that 82% of chicken isolates and 98% of human enteric isolates from the same locale were of an identical biotype suggests an epidemiological link. In contrast to our data, Skirrow and Benjamin (Abstr. Int. Workshop *Campylobacter* Infect. 1981, University of Reading, United Kingdom) reported that in Great Britain *C. jejuni* biotype 1 was the most frequent enteropathogenic *Campylobacter* sp. in humans and that poultry isolates were mostly of biotype 2. *C. jejuni* biotype 1 predominated in cattle and sheep, *C. coli* predominated in pigs, and the nalidixic acid-resistant thermophilic *Campylobacter* strains were common in wild birds. Further studies on the distribution of biotypes among animals in Sydney should determine whether the biotyping scheme of Skirrow and Benjamin will identify discriminatory, epidemiological markers.

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