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A foodborne outbreak of *Campylobacter jejuni* (O:33) infection associated with tuna salad: a rare strain in an unusual vehicle

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SUMMARY

We report a foodborne outbreak of *Campylobacter jejuni* infection in a summer camp. Outbreak-related cases occurred in 79 persons including 3 secondary cases in campers. *Campylobacter jejuni* was isolated from stool specimens from 16 of 21 patients who submitted a sample; 13 viable isolates were serotyped and all were serotype O:33 (somatic O scheme) or HL:18 (heat-labile scheme), and biotype III (Lior scheme). This serotype is widely distributed geographically but rarely isolated from humans. Samples of water from the wells supplying the camp were negative for faecal coliforms, and raw milk had not been served in the camp. A matched (1:1) case-control study identified tuna salad served for lunch on 19 July as the likely food item associated with illness (matched odds ratio = 22; 95% confidence intervals (CI) = 3·6–908). Swimming in the camp pool and other recreational water use in area lakes by the campers were not statistically associated with illness. The precise mechanism of introduction of the organism into the tuna salad remains unknown; contamination most likely occurred through cross-contamination with another food product, the hands of a food handler, or a work surface. Several deficiencies in the operation of the camp kitchen were identified. In Wisconsin, kitchens of such camps are subject to different inspection rules than restaurants. Camp staff, administrators, counselors, food managers, and infirmary staff, should fulfil important roles in their respective areas to prevent future outbreaks.

INTRODUCTION

Recent studies have found *Campylobacter jejuni/C. coli* to the most common cause of human diarrhoeal disease in developed countries [1]. This observation is true in Wisconsin where, from 1986 through 1994, a mean annual number of 1390 cases of campylobacter infections (range: 1146–1607 cases) were reported to the Wisconsin Division of Health (DOH) [2]. Despite this frequency of reported infection and unlike infections associated with other enteric bacterial pathogens (e.g., *Salmonella* sp., *Escherichia coli* O157:H7), outbreaks of campylobacter infections are infrequent. During 1986–94, 181 foodborne and

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waterborne outbreaks with an identified pathogen were reported to DOH; only 3 (1·7%) were caused by campylobacter organisms [3].

Investigations of outbreaks of campylobacter infections have mainly implicated raw milk [4, 5], poultry [6, 7], contact with pets [8, 9] and water [10, 11] as vehicles or mechanisms associated with infection. Children are often affected during outbreaks of campylobacter enteritis associated with drinking raw milk because a majority of these outbreaks occurred during organized youth activities (e.g., school excursions, summer camps, or farm retreats) [12, 13]. Summer camps constitute a setting that facilitates the occurrence of foodborne outbreaks, and reports have described outbreaks of *Clostridium perfringens*, *E. coli* O157:H7, *Yersinia enterocolitica*, and *C. jejuni* in a summer camp setting [14–17].

We report the clinical, epidemiological, and bacteriologic features of an outbreak of gastroenteritis in a summer camp in Wisconsin caused by an unusual serotype of *Campylobacter jejuni* in which tuna salad was implicated epidemiologically as the vehicle for transmission.

METHODS

Background

On 24 July 1995, Sauk County Health Department (SCHD) officials were notified by a summer camp administrator of an increase in gastrointestinal illness among campers and staff. The coeducational camp held two 4-week sessions from 18 June through 15 July (Session 1) and from 16 July through 13 August (Session 2). Most of the 195 staff (counselors, administrative personnel, and kitchen personnel) stayed in the camp during both sessions. No increased occurrence of gastrointestinal illness was reported among Session 1 campers. Session 2 involved 503 campers, including 113 adolescents who resided and ate separately from the rest of the campers.

Epidemiological investigation

The investigations included only Session 2 campers and staff. The camp infirmary log was reviewed to detect cases of gastrointestinal illness. A probable case of campylobacter infection was defined as diarrhoea (more than 3 stools per 24 h) with onset from 16 July through 31 July in a camp resident. A confirmed case-patient additionally had a stool culture positive for *C. jejuni*. Listings of camper and staff names and their residences in the camp were obtained. Case residences were plotted on a map to determine whether geographic clustering occurred.

A matched (1:1) case-control study involved 35 camp residents with illness meeting the case definition (13 confirmed and 22 probable cases) and who stayed at least 1 night in the infirmary. Controls were selected from the list of healthy room-mates residing in the same sleeping quarters as the case-patient and were matched to cases by age and gender. This age and gender matching was clear-cut in non-staff cabins as they lodged campers of similar age ($\pm$1 year) and gender. In staff cabins, the age-matching was more complex and less perfect as occupants were of different age groups; the healthy person with the age closest to case-patient was selected. In addition to the age and gender matching, residents of the same cabin had a similar seating placement in the cafeteria. The brief, self-administered questionnaire with simple questions was given to case-patients and controls under the guidance of their residence counselors. Cases were compared with their controls regarding consumption of food and beverages during the period 16–22 July and regarding recreational water use during the week before the day of interview.

Environmental investigation

Sanitary inspections occurred on 24 July and 11 August. The first inspection involved the camp swimming pool, kitchens, and food-storage facilities and included evaluation of food-preparation methods, food-serving techniques, and the hygiene standards of the kitchen personnel. Drinking water samples from each of the camp's two private wells were obtained on 24 July. A list of food items and beverages served from 16 July through 22 July was obtained from the food manager; we asked specifically whether raw milk was served during any camp functions or day excursions. Food items from that period were not available for bacteriology. During the second inspection, the evaluation focused on the specific food item epidemiologically identified as the likely cause of the outbreak. Questions focused on the food item's ingredients, preparation method, storage and carry-over, and the hygiene and illness history of the food handler(s) directly involved in the item's preparation.

Laboratory investigation

Stool specimens from 21 ill camp attendees and staff were submitted by infirmary staff to a referral hospital

for routine culture of enteric bacterial pathogens. The campylobacter isolates were referred to the Microbiology Section, Wisconsin State Laboratory of Hygiene (WSLH), for confirmatory testing and pulsed-field gel electrophoresis (PFGE) using *Sal*I for restriction of DNA. Subsequently, the WSLH referred isolates to the Epidemic Investigations and Surveillance Laboratory, Foodborne and Diarrheal Diseases Section, CDC, for serotyping using the somatic O (formerly heat-stable) scheme of Penner and Hennessy [18]. Isolates were also referred to the Bureau of Microbiology, Canadian National Laboratory for Bacteriology and Enteric Pathogens (CNLBEP), for serotyping using the heat-labile (HL) scheme of Lior and colleagues [19], and biotyping using the method developed by Lior [20].

Statistical analysis

For the univariate analysis, maximum likelihood estimates of matched odds ratios with exact 95% CI were used. For the stratified analysis, stratum specific estimates and summary adjusted matched odds ratios were calculated. Continuous variables were analysed using the ANOVA test (Epi-Info, version 6.02, CDC).

RESULTS

Epidemiological Investigation

On the basis of laboratory reports and infirmary log book review, a total of 16 confirmed cases (9 in campers, 7 in staff) and 63 probable cases (45 in campers, 18 in staff) were identified during this outbreak (Fig. 1). The three cases with onsets after 27 July occurred in residents of three different cabin groups and were probably secondary cases. Based on the time the epidemiologically implicated meal was served, the median incubation interval was 4 days (range: < 1–8 days), excluding these three secondary cases. Thirty-nine (49%) case-patients spent at least one night in the infirmary, an indication of the relative seriousness of their illnesses. No cases were reported among residents of the adolescent cabin group who ate exclusively in a separate dining hall with its own food preparation facility. Cabin-group-specific attack rates ranged from 6–22% (mean = 13%). The temporal distributions of cases in each of the nine cabin groups were similar.

Two culture-confirmed campylobacter infections occurred in campers with onsets before 16 July and were, therefore, not included in the case count. We were unable to assess whether these two cases were outbreak related; their isolates were discarded before initial notification of the outbreak and were not serotyped. The first early case had onset on 11 July in a Session 1 male camper who left the camp before Session 2 campers arrived on 16 July. The second early case occurred in a Session 2 female camper who had illness onset while at home on 13 July and who boarded the bus to the camp despite her illness.

Detailed clinical information was available from 31 (49%) probable case-patients and 14 (88%) confirmed case-patients who completed a questionnaire. Persons with confirmed cases and probable cases had similar clinical manifestations except for nausea and vomiting (Table 1). There were no significant differences between these confirmed and probable case-patients regarding sex (53% *vs.* 67% female, $P = 0{\cdot}6$) and mean age (15·2 years *vs.* 14·2 years, ANOVA calculated *P*-value: 0·4). Information for the remaining 32 persons with probable cases was retrieved from the infirmary log book in which signs and symptoms were not recorded in a standard manner.

The 35 case patients enrolled in our case control study had a mean age of 15·2 years (range 9–30); the 35 control subjects' mean age was 13·5 years (range 10–22). Between 16 July and 19 July, approximately 45 food items and beverages were served in the summer camp; raw milk was not served at any time to the campers or staff. Although univariate analysis associated two meal-specific items with illness (tuna salad served during lunch and dinner on 19 July), stratified analysis identified the tuna salad served for lunch as the only statistically significant vehicle of infection ($P < 0{\cdot}0003$). The tuna salad served for lunch was eaten by 23 (66%) of 35 patients versus 2 (6%) of 34 controls (matched odds ratio = 22; 95% CI = 3·6–908); one control did not recall his food history. Swimming in the camp pool and other recreational water use in area lakes after arriving in the camp were not statistically associated with illness.

Environmental investigation

Several deficiencies were observed in the main dining facility and among the practices of the main dining hall kitchen staff, including failure to wear hair restraints, improper use of gloves, inadequate hand washing, the absence of sneeze-guards and inadequate temperature controlling devices at the salad bars.

The camp employed 28 food handlers in the central kitchen. Thirteen were foreign students, residents of

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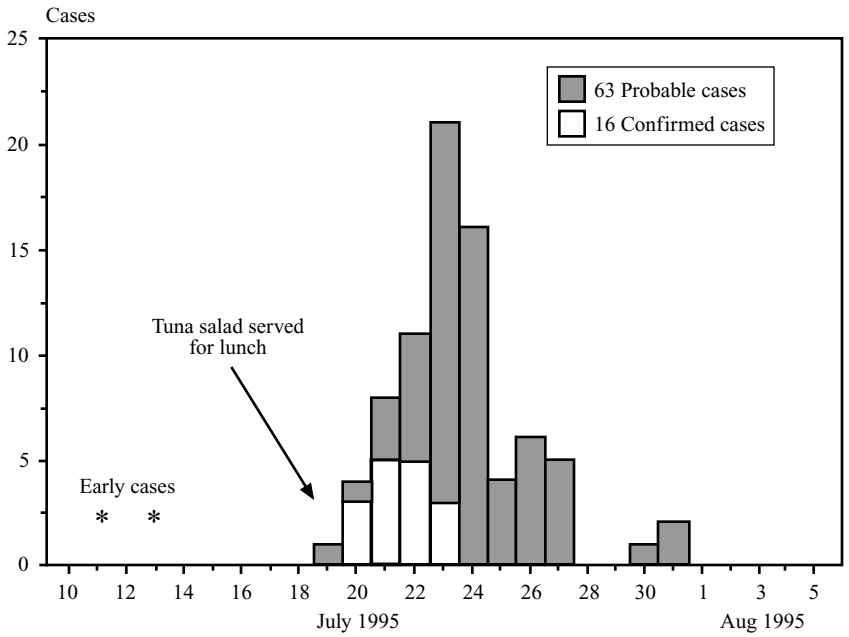

Fig. 1. Confirmed and probable cases of *Campylobacter jejuni* infection in a summer camp by date of onset, Wisconsin, July 1995.

Table 1. *Signs and symptoms of illness in persons with confirmed and probable cases of* Campylobacter jejuni *infections who completed a detailed questionnaire following an outbreak at a summer camp, Wisconsin, 1995*

Sign or symptom	Confirmed cases ($n = 14$) number (%)	Probable cases ($n = 31$) number (%)
Diarrhoea*	14 (100%)	31 (100%)
Weakness	12 (86%)	21 (68%)
Abdominal cramps	11 (79%)	21 (68%)
Fever	11 (79%)	27 (87%)
Headache	9 (64%)	23 (72%)
Vomiting†	8 (57%)	7 (21%)
Muscle aches	8 (57%)	14 (44%)
Nausea†	7 (50%)	25 (78%)
Chills	7 (50%)	14 (42%)
Sweats	6 (43%)	16 (48%)
Bloody diarrhoea	3 (21%)	7 (23%)

* Required to meet the case definition.
† Significant difference in prevalence, $P < 0{\cdot}05$ (Fisher Exact 2-Tailed *P*-value).

other countries (England, France, Poland, Hungary) who were in the United States only for the summer. Most food handlers did not work in food service during the rest of the year. They had not received formal training in safe food handling practices when starting their jobs in the camp. Four food handlers were among the case patients; 2 had submitted stool samples and 1 specimen was positive for *C. jejuni*. No food handler reported signs and symptoms of gastrointestinal illness before the outbreak; the reported dates of illness onset among these 4 food handlers were 21 July or later.

The food manager reported that any of 10 different food handlers could have prepared the implicated tuna salad. No kitchen logs were maintained identifying the preparer of food items and tuna salad preparation dates. Tuna salads were separately prepared in the central kitchen and the kitchen used for preparing food for adolescents. For each batch of tuna salad the ingredients were canned tuna fish (6 cans at 4 pounds, 2 ounces per can), salt, and mayonnaise (1 pound of mayonnaise obtained from a 4 gallon pail). The mayonnaise was a commercially prepared product which was made with pasteurized ingredients. The ingredients for the tuna salad were mixed in a large bowl by using gloved hands. These large batches of tuna salad were made every 2–3 days and a portion would be served; the remainder was kept in the cooler at 4 °C. The tuna salad was served in bowls at four salad bars. Bowls were placed on ice cubes at the start of the meal. No refilling of ice cubes occurred during the meal. Leftover tuna salad was added back to the portion kept in the cooler and served on the next occasion. Tuna salad was served during lunch and dinner on 16, 17 and 19 July. As no

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logs were kept, it was not possible to conclude if these servings belonged to the same batch nor to determine its preparation date. The kitchen staff had prepared a meal that included a chicken item on 18 July and 19 July.

No deficiencies in the operation and maintenance of the camp swimming pool were observed. The results of the water samples from the two wells supplying the camp drinking water were negative for faecal coliforms.

Laboratory investigation

The WSLH received 16 isolates of *Campylobacter* sp. from the referral hospital; all were identified as *C. jejuni*. PFGE assays yielded an identical banding pattern for all 16 isolates. Thirteen of the 16 isolates were referred to CDC and CNLBEP for serotyping; 3 isolates became non-viable. At CDC, all 13 isolates had a positive reaction to O:33 antigen; 2 of the isolates also had a weak reaction to the O:40 antigen. At CNLBEP, all 13 isolates reacted to HL:18 antigen and were identified as biotype III.

DISCUSSION

This foodborne outbreak in a summer camp was clearly derived from a point-source likely to be the tuna salad served for lunch on 19 July. The incubation period (4 days), nature, and duration of illness was consistent with campylobacter gastroenteritis. The finding of campylobacter bacteria in the stools of 16 of 21 persons cultured, and the clinical similarities in illness among persons with confirmed and probable cases, provide strong evidence this organism was the cause; serotyping techniques identified the bacterium as *Campylobacter jejuni* serotype O:33 (somatic O scheme), HL:18 (heat-labile scheme), biotype III (Lior scheme).

From 1973 through 1992, 80 foodborne outbreaks of campylobacter infections were reported to CDC (unpublished data, Foodborne and Diarrheal Diseases Branch, CDC). A vehicle was implicated in 47 (60%) of these outbreaks. Only one outbreak investigation implicated tuna salad; this occurred in 1986 at a New York State correctional facility. The *C. jejuni* associated with that outbreak was not serotyped.

Although there appear to have been multiple opportunities for contamination of the tuna salad during this outbreak, the precise mechanism of introduction of the organism into the tuna salad remains unknown. An intrinsic contamination of the tuna salad ingredients (tuna or mayonnaise) is unlikely, but cannot be ruled out. *Campylobacter jejuni* has been isolated from freshwater, saltwater, and shellfish [21, 22]. Although reports of *C. jejuni* gastroenteritis associated with the consumption of shellfish and raw or rare fish have been reported [23–25], *Campylobacter* sp. has never been isolated from raw or unprocessed tuna fish to our knowledge. The kitchen in the main dining hall used large (4 gallon) pails of mayonnaise. Smaller quantities were scooped from the pail for the preparation of separate food items as necessary; contaminated equipment could have introduced *C. jejuni* in the pail after opening. Kitchen staff used mayonnaise in the preparation of several other dishes, but the tuna salad was the only food item associated with illness.

Improper temperature-controlling devices and recycling of leftovers support the possibility that an infected camper might have contaminated the tuna salad at the salad bar. However, unlike salmonella, campylobacter does not multiply in foods left at ambient temperatures [26]. Campylobacter bacteria generally have a poor survival rate because of their sensitivity to different environmental factors such as high temperature, low pH, atmospheric concentration of oxygen, and the absence of moisture [27–29]. Moreover, *C. jejuni* does not propagate at temperatures below 30 °C. Hence, *C. jejuni* infection mainly occurs by transmission of organisms from contaminated materials directly to the mouth. This evidence makes it unlikely that 1 infected camper could have contaminated the tuna salad with sufficient organisms during serving to account for illness in the other persons associated with this outbreak.

The most likely explanation of the outbreak is contamination of the tuna salad during preparation in the main dining hall kitchen by cross contamination with another food product, or from the hands of a food handler or an environmental work surface. Poultry parts and carcasses have been found to be major sources of *C. jejuni*. Most retail chicken carcasses contain *C. jejuni*; in one study, a recovery rate of 98% was reported from raw chicken [30, 31]. Review of the full menu of meals served in the camp indicated that chicken had been served during 2 meals: the 18 July dinner included chicken patties; and the 19 July dinner included a chicken stir fry. Although the frozen chicken patties did not require much manipulation in the kitchen, the chicken stir fry required considerable preparation. It is possible that cross-contamination from the equipment or cutting

boards could have occurred during these preparation procedures. Another possibility is that the hands of foodhandlers contaminated from handling the raw chickens could have transferred campylobacter bacteria to already prepared foods. Such a mechanism has already been described [32].

The role of infected food handlers in campylobacter outbreaks has not been well studied but is probably minor. Blaser and colleagues described a foodborne outbreak of campylobacter infections in a summer camp likely associated with an ill food handler [17]. None of the food handlers in our survey reported illness before 21 July; however the foreign students might not have understood the importance of the questioning or might have been reluctant to reveal illness. No reports of transmission of campylobacter infection by asymptomatic carriers (i.e., food handlers) have been reported to date. Person-to-person transmission of campylobacter infection appears to be infrequent; in the rare occasions where it was reported, the index case has usually been a young child who is not toilet trained [33, 34]. Despite the suboptimal hygienic conditions and the young age of some campers, only three possible secondary cases were identified.

Campylobacter jejuni serotype O:33 is rarely isolated from human patients. Between July 1989 and June 1990, a group of 298 *C. jejuni* isolates was systematically collected from 19 representative counties in the United States to evaluate the serotype distribution and antimicrobial resistance patterns [34]. Two (0·7%) isolates of this group were identified as serotype O:33 [unpublished data, M. Nicholson, CDC]. Similarly, in a study conducted during 1980–2, Penner and colleagues identified only 12 (0·6%) serotype O:33 isolates among a group of 1586 *C. jejuni* strains [36]. An additional 4 isolates of this serotype have been identified in Penner's laboratory since 1982 (personal communication, J. L. Penner). The 16 O:33 isolates serotyped by Penner were from Canada (9), United States (2), United Kingdom (4), and South Africa (1). Other reports mention the isolation of the O:33 serotype in Pakistan (3%) and of the HL:18 serotype in France (1%) [37, 38]. These data confirm the geographic distribution and the relative rarity of campylobacter enteritis in humans caused by this serotype.

Foodborne enteric disease outbreaks in summer camps occur regularly. In the United States, 110 known foodborne outbreaks in camp settings were reported to CDC between 1973 and 1992 (unpublished data, Foodborne and Diarrheal Diseases Branch, CDC). Several factors may predispose summer camp settings to foodborne outbreaks: inexperienced kitchen staff (sometimes campers or counselors themselves), poor hygiene conditions, and overcrowded kitchens and cafeterias. In Wisconsin, kitchens of summer camps are subject to less stringent inspection rules than those imposed on restaurants; however, the same standards will be used in the near future.

Camp staff can help prevent outbreaks of foodborne illness. Camp administrators need to ensure that dining halls and kitchens are properly equipped and well maintained, and that hand-washing and toilet facilities are kept clean. Camp counselors need to encourage and supervise proper personal hygiene of campers (e.g., hand washing before meals and after use of toilets) and set a good example for the campers in their charge. Camp food managers need to provide appropriate educational sessions regarding basic food-handling practices to inexperienced food handlers and ensure ongoing compliance. Finally, camp infirmary staff need to be aware of the potential of foodborne or waterborne illness in camp settings. Requests for appropriate cultures/tests of stool specimens should be made with an additional provision to ensure that the laboratory retain isolates for at least 2 weeks.

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REFERENCES

1. Tauxe R. Epidemiology of *Campylobacter jejuni* infections in the United States and other industrialized nations. In Nachamkin I, Blaser MJ, Tompkins LS, *Campylobacter jejuni*: current status and future trends. Washington, DC: American Society for Microbiology, 1992: 9–19.
2. Annual reports, Communicable Disease Section, Wisconsin Division of Health, 1986–1994, Madison WI.
3. Communicable Diseases in Wisconsin, 1986–1996. Wisconsin Division of Health, Madison WI.
4. Centers for Disease Control. *Campylobacter* outbreak associated with raw milk provided on a dairy tour – California. MMWR 1986; **35**: 311–2.
5. Klein BS, Vergeront JM, Blaser MJ, et al. *Campy-*

lobacter infection associated with raw milk. JAMA 1986; **255**: 361–4.
6. Rosenfield JA, Arnold GJ, Davey GR, Archer RS, Woods WH. Serotyping of *Campylobacter jejuni* from an outbreak of enteritis implicating chicken. J Infect 1985; **2**: 159–65.
7. Istre GR, Blaser MJ, Shillam P, Hopkins RS. *Campylobacter* enteritis associated with undercooked barbecued chicken. Am J Publ Hlth 1984; **74**: 1265–7.
8. Blaser MJ, Waldman RJ, Barrett T, et al. *Campylobacter* enteritis associated with canine infection. Lancet 1978; ii: 979–81.
9. Deming MS, Tauxe RV, Blake PA, et al. *Campylobacter* enteritis at a university: transmission from eating chicken and from cats. Am J Epidemiol 1987; **126**: 526–34.
10. Vogt RL, Sours HE, Barrett T, Feldman RA, Dickinson RJ, Witherell L. *Campylobacter* enteritis associated with contaminated water. Ann Intern Med 1982; **96**: 292–6.
11. Sacks JJ, Lieb S, Baldy LM, et al. Epidemic campylobacteriosis associated with a community water supply. Am J Publ Hlth 1986; **76**: 424–9.
12. Joseph C, Noah N, White J, Hoskins T. A review of outbreaks of infectious disease in school in England and Wales, 1979–88. Epidemiol Infect 1990; **105**: 419–34.
13. Wood RC, MacDonald KL, Osterholm MT. *Campylobacter* enteritis outbreaks associated with drinking raw milk during youth activities – a 10-year review of outbreaks in the United States. JAMA 1992; **268**: 3228–30.
14. Hook D, Jalaludin B, Fitzsimmons G. *Clostridium perfringens* food-borne outbreak: an epidemiological investigation. Aust New Zealand J Publ Hlth 1996; **20**: 119–22.
15. Centers for Disease Control and Prevention. *Escherichia coli* O157:H7 outbreak at a summer camp – Virginia, 1994. MMWR 1995; **44**: 419–21.
16. Shayegani M, Morse D, DeForge I, Root T, Parsons LM, Maupin PS. Microbiology of a major foodborne outbreak of gastroenteritis caused by *Yersinia enterocolitica* serogroup O:8. J Clin Microbiol 1983; **17**: 35–40.
17. Blaser MJ, Checko P, Bopp C, Bruce A, Hughes JM. *Campylobacter* enteritis associated with foodborne transmission. Am J Epidemiol 1982; **116**: 886–94.
18. Penner JL, Hennessy JN. Passive hemagglutination technique for serotyping *Campylobacter fetus* subsp *jejuni* on the basis of soluble heat-stabile antigens. J Clin Microbiol 1980; **12**: 732–7.
19. Lior H, Woodward DL, Edgar JA, Laroche LJ, Gill P. Serotyping of *Campylobacter jejuni* by slide agglutination based on heat-labile antigenic factors. J Clin Microbiol 1982; **15**: 761–8.
20. Lior H. New extended biotyping scheme for *Campylobacter jejuni*, *Campylobacter coli*, and *Campylobacter laridis*. J Clin Microbiol 1984; **20**: 636–40.
21. Bolton FJ, Coates D, Hutchinson DN, Godfree AF. A study of thermophilic *Campylobacters* in a river system. J Appl Bacteriol 1987; **62**: 167–76.
22. Wilson IG, Moore JE. Presence of *Salmonella* spp. and *Campylobacter* spp. in shellfish. Epidemiol Infect 1996; **116**: 147–53.
23. Abeyta JR, Deeter FG, Kaysner CA, Stott RF, Wekell MM. *Campylobacter jejuni* in Washington State shellfish growing beds associated with human illness. J Food Protect 1992; **56**: 323–5.
24. Griffin MR, Dalley E, Fitzpatrick M, et al. *Campylobacter* gastroenteritis associated with raw clams. J Med Soc N J 1983; **80**: 607–9.
25. Harris NV, Weiss NS, Nolan CM. The role of poultry and meats in the etiology of *Campylobacter jejuni/coli* enteritis. Am J Publ Hlth 1986; **76**: 407–10.
26. Butzler JP, Oosterom J. *Campylobacter*: pathogenicity and significance in foods. Int J Food Microbiol 1991; **12**: 1–8.
27. Doyle MP, Roman DJ. Growth and survival of *Campylobacter fetus* subsp *jejuni* as a function of temperature and pH. J Food Protect 1981; **44**; 596–601.
28. Doyle MP, Roman DJ. Sensitivity of *Campylobacter jejuni* to drying. J Food Protect 1982; **45**; 507–10.
29. Hoffman PS, Krieg NR, Smibert RM. Studies on the microaerophilic nature of *Campylobacter fetus* subsp *jejuni*. Can J Microbiol 1979; **25**: 1–7.
30. Lammerding AM, Garcia MM, Mann ED, et al. Prevalence of *Salmonella* and thermophilic *Campylobacter* in fresh pork, beef, veal, and poultry in Canada. J Food Protect 1988; **51**: 47–52.
31. Stern NJ, Line JE. Comparison of three methods for recovery of *Campylobacter* spp. from broiler carcasses. J Food Protect 1992; **55**: 663–6.
32. Brown P, Kidd D, Riordan T, Barrell RA. An outbreak of food-borne *Campylobacter jejuni* infection and the possible role of cross-contamination. J Infect 1988; **17**: 171–6.
33. Blaser MJ, Waldman RJ, Barrett T, Erlandson AL. Outbreaks of *Campylobacter* enteritis in two extended families: evidence for person-to-person transmission. J Pediatr 1981; **98**: 254–7.
34. Oosterom J, den Uyl CH, Banffer JR, Huisman J. Epidemiological investigation on *Campylobacter jejuni* in households with a primary infection. J Hyg 1984; **93**: 325–32.
35. Patton CM, Nicholson MA, Ostroff SM, Ries AA, Wachsmuth JK, Tauxe RV. Common somatic O and heat-labile serotypes among *Campylobacter* strains from sporadic infections in the United States. J Clin Microbiol 1993; **31**: 1525–30.
36. Penner JL, Hennessey JN, Congi RV. Serotyping of *Campylobacter jejuni* and *Campylobacter coli* on the basis of thermostable antigens. Eur J Clin Microbiol 1983; **2**: 378–83.
37. Neogi PKB, Shahid NS. Serotypes of *Campylobacter jejuni* isolated from patients attending a diarrhoeal disease hospital in urban Bangladesh. J Med Microbiol 1987; **24**: 303–7.
38. Mégraud F, Gavinet AM, Camou-Junca C. Serogroups and biotypes of human strains of *Campylobacter jejuni* and *Campylobacter coli* isolated in France. Eur J Clin Microbiol 1987; **6**: 641–5.