

Risk Factors for Sporadic *Campylobacter* Infection in the United States: A Case-Control Study in FoodNet Sites

Cindy R. Friedman,^{1,4} Robert M. Hoekstra,² Michael Samuel,⁵ Ruthanne Marcus,⁶ Jeffrey Bender,⁷ Beletshachew Shiferaw,⁸ Sudha Reddy,¹ Shama Desai Ahuja,³ Debra L. Helfrick,² Felicia Hardnett,² Michael Carter,⁹ Bridget Anderson,¹⁰ and Robert V. Tauxe,¹ for the Emerging Infections Program FoodNet Working Group

¹Foodborne and Diarrheal Diseases Branch and ²Biostatistics and Information Branch, Division of Bacterial and Mycotic Diseases, National Center for Infectious Diseases, Centers for Disease Control and Prevention, and ³Georgia Emerging Infections Program, Atlanta, Georgia; ⁴National Centers for Infectious Diseases, Centers for Disease Control and Prevention, Washington, D.C.; ⁵California Emerging Infections Program, San Francisco; ⁶Connecticut Emerging Infections Program, New Haven; ⁷Minnesota Department of Health, Minneapolis; ⁸Oregon Department of Human Services, Portland; ⁹Maryland Department of Health and Mental Hygiene, Baltimore; and ¹⁰New York State Department of Health, Albany

Campylobacter is a common cause of gastroenteritis in the United States. We conducted a population-based case-control study to determine risk factors for sporadic *Campylobacter* infection. During a 12-month study, we enrolled 1316 patients with culture-confirmed *Campylobacter* infections from 7 states, collecting demographic, clinical, and exposure data using a standardized questionnaire. We interviewed 1 matched control subject for each case patient. Thirteen percent of patients had traveled abroad. In multivariate analysis of persons who had not traveled, the largest population attributable fraction (PAF) of 24% was related to consumption of chicken prepared at a restaurant. The PAF for consumption of nonpoultry meat that was prepared at a restaurant was also large (21%); smaller proportions of illness were associated with other food and nonfood exposures. Efforts to reduce contamination of poultry with *Campylobacter* should benefit public health. Restaurants should improve food-handling practices, ensure adequate cooking of meat and poultry, and consider purchasing poultry that has been treated to reduce *Campylobacter* contamination.

Since the late 1970s, *Campylobacter* has been recognized as the most common cause of bacterial gastroenteritis in many countries [1]. Each year, there are an estimated 2.4 million *Campylobacter* infections in the United States, associated with an estimated 124 deaths [2]. In addition, the sequelae of *Campylobacter* infection can cause considerable morbidity [3], including reactive arthritis and Guillian-Barré syndrome [4, 5].

The majority of *Campylobacter* infections are sporadic. Epidemiological investigations to determine risk factors for sporadic *Campylobacter* infections have been conducted in the United States and in other developed nations. Although these studies differed in location, technique, and sample size, they consistently indicated several dominant sources of infection, including contact with and consumption of poultry, transmission from pets and other animals, consumption of raw milk, and contaminated drinking water [6–13]. Despite the identification of these risk factors for infection, the incidence of *Campylobacter* infection continues to increase in many industrialized nations [14]. In the United States, the incidence, as measured through the Foodborne Disease Active Surveillance Network (FoodNet), decreased by 27% between 1996 and 2001 but remains above the national objective for 2010 of 12.3 cases per 100,000 [15].

Financial support: Centers for Disease Control and Prevention's National Center for Infectious Diseases, US Department of Agriculture Food Safety Inspection Service, and US Food and Drug Administration Center for Food Safety and Applied Nutrition.

Reprints or correspondence: Dr. Cindy R. Friedman, National Center for Infectious Diseases, Centers for Disease Control and Prevention, 746 G Hubert Humphrey Bldg., 200 Independence Ave. SW, Washington, D.C. 20201 (ccf6@cdc.gov).

Clinical Infectious Diseases 2004;38(Suppl 3):S285–96

This article is in the public domain, and no copyright is claimed.
1058-4838/2004/3808S3-0023

Prevention measures for human *Campylobacter* infection are needed to reduce the associated morbidity and economic loss and to attenuate the emergence of antimicrobial-resistant strains, particularly strains resistant to fluoroquinolones, a class of antibiotic used to treat severe campylobacteriosis. Better understanding of the risk factors for *Campylobacter* transmission may provide a means to prevent infection with antimicrobial-resistant strains as well as susceptible strains.

To address this ongoing public health problem, we conducted a large nationwide case-control study of patients with sporadic *Campylobacter* infections in FoodNet. FoodNet is a collaboration between the Centers for Disease Control and Prevention (CDC), selected state health departments, the United States Department of Agriculture, and the US Food and Drug Administration and was conducted as part of the CDC Emerging Infections Program. In 1998, FoodNet participants accounted for 8% of the United States population. The objectives of the study were to better describe the burden of illness and to identify specific risk factors associated with sporadic *Campylobacter* infection in the United States to guide prevention efforts. Analysis of infections caused by fluoroquinolone-resistant *Campylobacter* will be reported separately [16].

METHODS

Study design and population. We conducted a 12-month, population-based, case-control study at 7 FoodNet sites located in Connecticut, Georgia, Minnesota, and Oregon and in selected counties in California, Maryland, and New York in 1998 and 1999. A case was defined as diarrhea and a culture-confirmed *Campylobacter* infection in a patient who was not part of an outbreak during the study period. We attempted to enroll ≥ 200 *Campylobacter* case patients per site randomly distributed throughout the year. The protocol was approved by the CDC and FoodNet site Institutional Review Boards. We obtained informed consent from participants and conducted research in accordance with guidelines for human experimentation as specified by the US Department of Health and Human Services. After obtaining informed consent, patients and controls ≥ 12 years of age were interviewed by telephone. Parents or guardian's permission was obtained before interviewing any patient or control 12–18 years of age. For patients or controls < 12 years of age, a parent or guardian (whoever was most familiar with the eating habits and activities of the patient or control) was interviewed.

Patients were excluded from the study if (1) their primary residence was outside the FoodNet catchment area, (2) they were not reached after ≥ 15 telephone attempts, (3) no telephone number was available for their primary residence, (4) they could not speak English or they were unable to answer questions, (5) they were unable to give an estimated onset date

for their diarrhea, or (6) the onset of their diarrhea was ≥ 10 days before the date that the specimen which yielded *Campylobacter* was obtained.

Patients were interviewed within 21 days after the specimen collection date. If more than 1 patient had a culture-confirmed case in a household, only the patient with the earliest onset of diarrhea was included in the case-control study.

Controls. Each control was matched to the corresponding patient on the basis of age group and telephone exchange. The age groups were: 0 to < 6 months of age, 6 to < 24 months of age, 2 to < 6 years of age, 6 to < 12 years of age, 12 to < 18 years of age, 18 to < 40 years of age, 40 to < 60 years of age, and ≥ 60 years of age. Patients were interviewed about exposures in the 7 days before diarrhea onset, and controls were asked about exposures during the corresponding 7-day period. Controls were excluded if (1) their primary residence was outside the FoodNet catchment area, (2) they reported having diarrhea within 28 days of the onset date of diarrhea of the matched case patient, (3) they reported the recent occurrence of a culture-confirmed *Campylobacter* infection (with an onset of diarrhea within 28 days of the onset date of diarrhea of the matched case patient) in a person in their household, or (4) they could not speak English or they were unable to answer questions. To reduce recall bias, controls were interviewed no later than 7 days after the interview of the matched case patient. Controls were sought using progressive and sequential telephone digit dialing. We telephoned each number only once; if there was no answer, if the person answering the phone was not cooperative, or if the person answering the call identified the number as a business, we terminated that call and telephoned the next number in the sequence.

Questionnaire. Using a standardized questionnaire, we gathered detailed information about foreign and domestic travel, dining locations, kitchen and food handling practices, demographic characteristics, food, water, and animal exposures. In the questionnaire, a commercial food establishment was defined as anywhere food is prepared and sold ready to eat or reheat (e.g., restaurant, caterer, supermarket, deli, or salad bar). A fast-food restaurant was defined as a restaurant where the patron paid before eating the meal. A large social gathering was defined as an eating venue that is not a commercial food establishment, such as a church supper, club event, or community sports event. The categories of "other" chicken and "other" nonpoultry meat referred to open-ended questions regarding any type of chicken or nonpoultry meat consumed that did not fit into one of the categories listed in the questionnaire.

Statistical analysis. In addition to a descriptive analysis, we performed univariate and multivariate risk factor analysis using SAS software, version 8.1 (SAS Institute). We excluded children under 2 years of age from univariate and multivariate risk factor analysis because of the large number of missing values in that

Table 1. *Campylobacter*-infected patients identified by surveillance and enrolled in the case-control study, Foodborne Disease Active Surveillance Network (FoodNet), 1998–1999.

FoodNet site	No. of patients enrolled in case-control study (% of total enrolled)	No. of patients identified by surveillance (% of total identified)	Percentage of patients identified by surveillance enrolled in case-control study
California	188 (14)	761 (19)	25
Connecticut	280 (21)	600 (15)	47
Georgia	164 (13)	480 (12)	34
Maryland	120 (9)	240 (6)	50
Minnesota	242 (18)	1001 (25)	24
New York	108 (9)	240 (6)	45
Oregon	214 (16)	681 (17)	31
Total	1316	4003	33

age group; however, children under 2 years of age were included in the descriptive analysis. A conditional logistic model was used for univariate analysis, and exposures with P values of $\leq .05$ were considered significant. For both univariate and multivariate analysis, continuous variables were dichotomized, and the median value was chosen as the breakpoint. Variables were constructed by combining multiple items from the questionnaire—for example, the construction of the variable “ate any chicken” was derived by grouping ate fried chicken, ate roast chicken, ate baked chicken, et cetera. Other constructed variables included non-poultry meat (defined as beef, lamb, pork, or veal) and farm animals (defined as chickens, turkeys, sheep, goats, lamb, cattle, calves, horses, and swine).

We performed multivariate analysis with those variables that showed an association with disease status with $P < .2$ in a univariate conditional logistic model, variables of significant association reported in the literature, and variables that were biologically plausible in the context of the study. We considered the effect of age by examining risk factors in children 2 to <12 years of age and in those aged ≥ 12 years. We pursued a variety of model selection strategies, including forward, backward, and best subset selection based on F statistic and χ^2 score criteria, as well as manual strategies based on examining changes in the regression parameter vector and model fit criteria. We considered model stability with respect to missing values via simple imputation and subsequent model fit comparison. The above strategy yielded a set of candidate models that were examined for possible interactions with matching factors and model component variables. We then chose a representative model based on criteria of adequacy of description, stability of estimated parameters, interpretability, and parsimony. We considered selected interactions between risk factors and both demographic and exposure variables, some of which were sufficiently robust and were included in the final model.

The population attributable fractions (PAFs) were calculated for all risk factors in the final model using the logistic model case-control method described by Bruzzi et al. [17]. Protective factors were not included in our calculation of PAFs. Ninety-five percent confidence intervals for PAFs were calculated using jackknife estimates [18].

RESULTS

Descriptive Analysis

Study population. FoodNet active surveillance in the 7 sites identified 4025 patients with culture-confirmed *Campylobacter* infections during the 12-month study period, for an incidence of 19.4 infections per 100,000 population. The incidence varied from 36.8 cases per 100,000 in California to 10.1 cases per 100,000 in Maryland. Using selection sampling criteria, we contacted 2093 patients (52%) for the case-control study. Of these, 1316 (63%) were enrolled as case patients; 777 patients were excluded from the case-control study, either because they refused to participate or because they met 1 of the exclusion criteria. The number of enrolled patients ranged from 108 in New York to 280 in Connecticut. It took a mean of 3.5 telephone calls to reach patients; over 80% of patients were reached with <5 telephone calls.

Overall, the study patients represented 33% of all *Campylobacter*-infected patients identified through FoodNet surveillance during the study period. The proportion of the surveillance cases enrolled in the case-control study from each site ranged from 24% to 50% (table 1). The proportion of patients enrolled in the case-control study by site was similar to the proportion of patients identified by surveillance at each site during the same time period.

Group comparability and demographic characteristics.

The demographic characteristics of the 777 patients who were excluded from the case-control study were similar to those of the 1316 enrolled patients (table 2). The median ages of enrolled patients and controls were 34 and 35 years, respectively. There was a bimodal age distribution among enrolled patients, with one peak at 4 years of age and a second at 22–52 years (figure 1). Female subjects constituted 46% of patients and 67% of controls (matched OR [mOR], 0.4; 95% CI, 0.4–0.5; $P < .01$).

Patients and controls were generally similar in racial and ethnic makeup and location of residence, but more patients were white (mOR, 1.5; 95% CI, 1.1–1.9; $P < .01$) and more patients lived on farms (mOR, 1.7; 95% CI, 1.1–2.4; $P = .01$) than did controls. The median income range and level of education were similar among patients and controls, but more patients earned above the median income bracket of \$30,000–\$59,999 than did controls (mOR, 1.8; 95% CI, 1.4–2.3, $P < .01$).

Clinical illness. By definition, 100% of the 1316 patients reported diarrhea; the median duration was 6 days (mean, 7 days; range, 1–31 days), and 45% reported having bloody diarrhea. Over 80% of patients reported cramps and fever. There were no known deaths among patients; 79% reported visiting a physician for their illness, 37% went to an emergency de-

partment, and 12% were hospitalized for a median of 3 days (mean, 3 days; range, 1–21 days). The source of the specimen that yielded *Campylobacter* was reported for 1291 (98%) of the 1316 patients; 1285 (99%) were from stool, and 6 (<1%) were from blood. Among the 766 isolates for which the *Campylobacter* species was reported, 727 (95%) were *Campylobacter jejuni*, 28 (4%) were *Campylobacter coli*, 10 (1%) were *Campylobacter lari*, and 1 was *Campylobacter mucosalis*.

Risk Factor Analysis

Thirteen percent of patients reported international travel in the 7 days before illness, compared with 1.5% of controls (mOR, 10.0; 95% CI, 6.0–16.7; $P < .01$). The most common international destinations among patients were Europe (31%), Mexico (21%), Asia (20%), Central and South America (10%), and Canada (6%). Because of their potentially unique exposures and because they were not matched with controls who traveled, these 164 patients were excluded from further analysis.

Of the more than 450 variables assessed, 110 were associated with *Campylobacter* infection in univariate analysis (table 3). Consuming meals prepared in a restaurant was associated with

Table 2. Demographic characteristics of 1316 patients and 1316 controls who participated in a case-control study of *Campylobacter* infection and of 777 patients who refused to participate or were excluded from the case-control study.

Characteristic	Patients	Controls	Patients not participating in or excluded from case-control study ^a
Female sex	659 (46)	857 (67)	337 (45)
Age, median years (range)	34 (<1–96)	35 (<1–85)	33 (<1–100)
Race/ethnicity			
White	1083 (83)	1035 (79)	558 (73)
Black/African American	67 (5)	114 (9)	61 (8)
Hispanic/Latino	80 (6)	78 (6)	63 (8)
Asian	53 (4)	38 (3)	60 (8)
American Indian or Alaska native	8 (0.6)	8 (0.6)	4 (0.5)
Other	18 (1.4)	30 (2.4)	23 (3)
Location of residence			
Urban	455 (35)	482 (38)	319 (41)
Suburban	494 (37)	477 (36)	275 (36)
Town	167 (13)	173 (13)	81 (11)
Rural, not a farm	114 (9)	121 (9)	65 (8)
Farm	79 (6)	57 (4)	31 (4)
Median household income	\$30,000–\$59,999	\$30,000–\$59,999	\$30,000–\$59,999
Median highest level of education	Some college	Some college	High school diploma

NOTE. Data are no. (%) of persons, unless otherwise indicated.

^a Reasons for exclusion included (1) primary residence was outside catchment area, (2) not reached after ≥ 15 telephone attempts, (3) no telephone number available for primary residence, (4) could not speak English or unable to answer questions, (5) unable to give an estimated onset date for their diarrhea, and (6) onset of diarrhea was ≥ 10 days before the date that the specimen which yielded *Campylobacter* was collected.

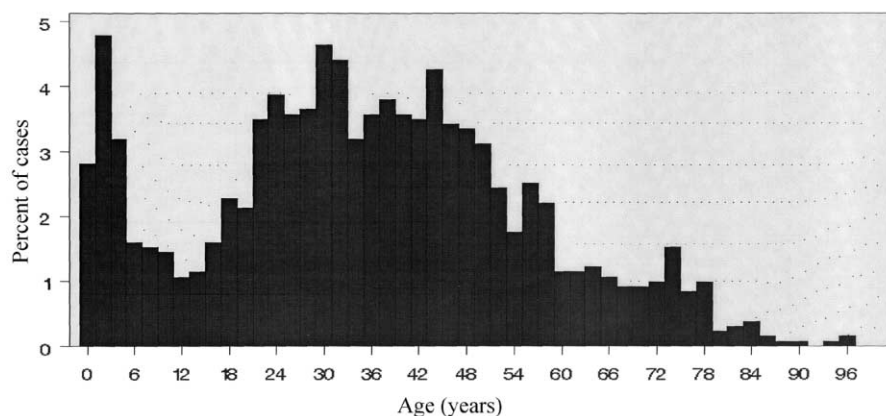


Figure 1. Age distribution of patients with *Campylobacter* infections, by 2-year intervals, FoodNet case-control study ($n = 1316$).

illness; 51% of patients had ≥ 3 meals at a restaurant in the 7 days before illness onset, compared with 44% of controls (mOR, 1.4; 95% CI, 1.1–1.6). Eating poultry and meat items was associated with a higher risk of illness if these foods were prepared at a restaurant, whereas eating these items when prepared at home was associated with a lower risk of disease. In particular, eating chicken (mOR, 2.4; 95% CI, 1.9–2.9), turkey (mOR, 2.5; 95% CI, 1.57–4.0), or nonpoultry meat (mOR, 2.1; 95% CI, 1.7–2.5) at a restaurant increased risk of campylobacteriosis. Food items that were associated with illness regardless of where they were prepared included undercooked or pink chicken, raw seafood, raw oysters, and unpasteurized milk. Food items that were protective regardless of where they were prepared included fresh lettuce, berries, chicken cooked as a whole (i.e., not cut into pieces), and chicken grilled outdoors. Some home food preparation practices that prevent cross-contamination, such as washing the cutting board after use with raw chicken (mOR, 0.5; 95% CI, 0.3–0.7) and washing hands after handling raw chicken (mOR, 0.5; 95% CI, 0.4–0.6), were associated with a reduced risk of campylobacteriosis. Several animal or farm exposures associated with infection included living on a farm, visiting a farm, having contact with farm animals, puppies, and adult dogs, particularly with their stool or with animals who had diarrhea. Visiting a petting zoo and being exposed to cats or kittens were not associated with illness. Drinking untreated water from a lake, river, or stream was associated with illness (mOR, 2.9; 95% CI, 1.6–5.3). No association was found between antibiotic use in the 4 weeks before illness and *Campylobacter* infection (mOR, 0.9; 95% CI, 0.6–1.3). Having a household member with diarrhea in the 4 weeks before illness onset was associated with a decreased risk of campylobacteriosis (mOR, 0.6; 95% CI, 0.4–0.8).

In the final multivariate model, 6 food exposures were independently associated with illness. In order of descending adjusted OR (AOR), they were as follows: drinking raw milk (AOR, 4.3), eating turkey prepared at a restaurant (AOR, 2.5),

eating chicken prepared at a restaurant (AOR, 2.2), eating undercooked or pink chicken (AOR, 2.1), eating raw seafood (AOR, 1.9), and eating nonpoultry meat prepared at a restaurant (AOR, 1.7) (table 4). Eating at a restaurant was a risk factor in univariate analysis, but it did not remain significant in the multivariate model. Of the nonfood exposures, the following factors were independently associated with illness: having a pet puppy (AOR, 3.4); drinking untreated water from a lake, river, or stream (AOR, 3.3); and having contact with animal stool (AOR, 1.4). Independent protective factors were eating turkey prepared at home (AOR, 0.5), eating chicken prepared at home (AOR, 0.7), eating nonpoultry meat prepared at home (AOR, 0.7), eating fried chicken (AOR, 0.5), eating fresh berries (AOR, 0.6), and female sex (AOR, 0.5). Adding a variable for the number of telephone calls necessary to reach patients to the final multivariate model did not change the point estimates of the AORs for the above exposures.

The model permitted comparison of risk and protective factors in age groups 2 to <12 years and ≥ 12 years. In general, these factors remained similar across age, except for exposure to farm animals, which was more likely to occur and had a higher OR among those aged 2 to <12 years (22% exposed; AOR, 21.0; 95% CI, 2.5–178) than among those aged ≥ 12 years (13% exposed; AOR, 2.0; 95% CI, 1.2–3.6). Comparison of risk and protective factors across gender in the final model also remained similar (data not shown).

PAFs

Among all patients, the proportion whose illness could be attributed to foreign travel was 12%. In the multivariate analysis of persons who had not traveled abroad, the largest PAF of 24% (95% CI, 17%–30%) was derived from consumption of chicken prepared at a restaurant (table 4). The PAF for consumption of nonpoultry meat that was prepared at a restaurant was also large (21%; 95% CI, 13%–30%). The other PAFs

Table 3. Univariate analysis of 1152 nontravelers participating in the Foodborne Disease Active Surveillance Network (FoodNet) *Campylobacter* case-control study, 1998–1999

Exposure in the 7 days before illness	Percentage of patients exposed	Percentage of controls exposed	mOR (95% CI)	P
Dining location				
Had ≥3 meals at any restaurant	51	44	1.4 (1.1–1.6)	<.01
Had >1 meal at a fast-food restaurant	46	40	1.2 (1.0–1.5)	.03
Had >1 meal at a table-service restaurant	60	58	1.2 (1.0–1.5)	.04
Had >14 meals prepared at home	45	53	0.7 (0.6–0.8)	<.01
Had a meal prepared on an outdoor grill at a small function	26	31	0.7 (0.6–0.9)	<.01
Food exposure				
Ate hamburger prepared at a large social gathering	2	0.4	8.0 (1.8–34.8)	.01
Ate pork roast cooked at a restaurant	1	0.5	6.5 (1.5–28.8)	.01
Ate chicken prepared on outdoor grill at a large social gathering	1	0.3	5.5 (1.2–24.8)	.03
Ate nonpoultry meat prepared at a large social gathering	4	0.7	4.9 (2.3–10.4)	<.01
Drank raw or unpasteurized milk	2	0.6	3.8 (1.4–10.2)	.01
Ate sausage cooked at restaurant	7	2	3.2 (2.0–5.4)	<.01
Ate raw oysters	1	0.6	3.0 (1.1–8.3)	.03
Ate pork chops prepared at a restaurant	2	0.8	2.8 (1.1–7.2)	.03
Ate stir-fried chicken prepared at a restaurant	5	2	2.8 (1.6–4.8)	<.01
Ate turkey prepared at a restaurant	6	3	2.5 (1.5–4.0)	<.01
Ate oven-roasted turkey prepared at a restaurant	3	1	2.5 (1.3–5.1)	.01
Ate bacon cooked at a restaurant	11	4	2.5 (1.8–3.7)	<.01
Ate chicken prepared at a restaurant	44	26	2.4 (1.9–2.9)	<.01
Ate broiled chicken prepared at a restaurant	3	2	2.3 (1.2–4.4)	.01
Ate steak prepared at someone else's home	2	1	2.3 (1.1–5.1)	.03
Ate steak prepared at a restaurant	9	5	2.2 (1.4–3.4)	<.01
Ate nonpoultry meat prepared at a restaurant	52	35	2.1 (1.7–2.5)	<.01
Ate oven-roasted chicken prepared at a restaurant	9	4	2.0 (1.4–3.0)	<.01
Ate chicken wings prepared at a restaurant	8	5	2.0 (1.3–3.0)	<.01
Ate roast beef prepared at a restaurant	7	4	2.0 (1.3–3.1)	<.01
Ate any other type of chicken	14	9	1.9 (1.4–2.5)	<.01
Ate undercooked or pink chicken	6	4	1.9 (1.2–2.8)	<.01
Ate any other type of meat	11	8	1.8 (1.3–2.4)	<.01
Ate raw seafood	6	4	1.8 (1.2–2.8)	.01
Ate rotisserie chicken prepared at a restaurant	6	4	1.7 (1.1–2.7)	.01
Ate chicken fingers, nuggets, or patties prepared at a restaurant	12	9	1.7 (1.2–2.3)	<.01
Ate ham prepared at a restaurant	13	9	1.6 (1.2–2.2)	<.01
Ate hamburger prepared a restaurant	29	21	1.6 (1.3–2.0)	<.01
Ate other ground beef prepared at a restaurant	6	4	1.6 (1.1–2.6)	.03
Ate fried chicken prepared at a restaurant	10	7	1.5 (1.1–2.0)	.02
Ate chicken salad	7	5	1.5 (1.0–2.1)	.03
Ate fresh lettuce	52	61	0.8 (0.6–0.9)	<.01
Ate fried chicken	18	23	0.7 (0.6–0.9)	<.01
Ate bacon prepared at home	18	25	0.7 (0.5–0.9)	<.01
Ate chicken cooked whole (not cut up during preparation)	9	13	0.7 (0.6–1.0)	.03
Ate chicken prepared on an outdoor grill	15	19	0.7 (0.6–0.9)	<.01
Ate ground beef prepared at home	14	21	0.7 (0.5–0.9)	<.01
Ate oven-roasted chicken prepared at home	23	32	0.7 (0.6–0.9)	<.01
Ate ham prepared at home	13	17	0.7 (0.5–0.9)	.01
Ate pork chops	20	26	0.7 (0.6–0.9)	<.01
Ate steak prepared at home	25	32	0.7 (0.5–0.8)	<.01
Ate stir-fried chicken	10	13	0.7 (0.5–1.0)	.03
Ate turkey prepared at home	9	13	0.6 (0.5–0.8)	<.01
Ate oven-roasted or baked turkey prepared at home	3	5	0.6 (0.4–0.9)	.02

(continued)

Table 3. (Continued.)

Exposure in the 7 days before illness	Percentage of patients exposed	Percentage of controls exposed	mOR (95% CI)	P
Ate chicken prepared on an outdoor grill at a small function	12	18	0.6 (0.5–0.8)	<.01
Ate pork chops prepared at home	17	24	0.6 (0.5–0.8)	<.01
Ate berries that were bought in a store	18	25	0.6 (0.5–0.8)	<.01
Ate nonpoultry meat prepared at home	64	72	0.6 (0.5–0.8)	<.01
Ate chicken prepared on an outdoor grill at home	11	16	0.6 (0.4–0.8)	<.01
Ate roast beef prepared at home	6	10	0.6 (0.4–0.8)	<.01
Ate broiled chicken prepared at home	5	10	0.5 (0.4–0.7)	<.01
Ate chicken prepared at home	45	64	0.5 (0.4–0.6)	<.01
Ate hamburger prepared at home	30	42	0.5 (0.4–0.7)	<.01
Ate lamb prepared at home	1	3	0.5 (0.3–0.9)	.03
Ate chicken wings prepared at home	4	7	0.5 (0.3–0.8)	<.01
Ate unpasteurized cheese	1	2	0.5 (0.2–0.9)	.03
Ate chicken fingers, nuggets, or patties prepared at home	3	6	0.4 (0.2–0.6)	<.01
Ate fried chicken prepared at home	7	15	0.4 (0.3–0.5)	<.01
Ate stir-fried chicken prepared at home	4	11	0.4 (0.2–0.5)	<.01
Ate rotisserie chicken prepared at home	0.5	2.0	0.3 (0.1–0.9)	.03
Kitchen and food-handling practices				
Had raw chicken in home refrigerator	21	28	0.7 (0.5–0.8)	<.01
Person in household bought raw chicken	37	45	0.7 (0.6–0.9)	<.01
Prepared >12 meals	43	55	0.6 (0.5–0.7)	<.01
Purchased raw chicken that was separated from other groceries with a plastic bag	12	18	0.6 (0.4–0.8)	<.01
Touched raw chicken	27	41	0.6 (0.5–0.7)	<.01
Had raw chicken at home that required thawing	29	42	0.6 (0.5–0.7)	<.01
Prepared raw chicken at home	48	61	0.6 (0.5–0.7)	<.01
Marinated raw chicken that was prepared at home	11	17	0.6 (0.5–0.8)	<.01
Used marinade for raw chicken and then discarded it	8	12	0.6 (0.5–0.8)	<.01
Used cutting board for cutting up raw chicken	7	14	0.5 (0.4–0.7)	<.01
Washed cutting board after use with raw chicken	7	14	0.5 (0.3–0.7)	<.01
Washed hands with soap and water after touching raw chicken	21	37	0.5 (0.4–0.6)	<.01
Cut up chicken that was prepared at home while it was raw	9	17	0.5 (0.4–0.7)	<.01
Demographic characteristics				
Annual household income more than \$30,000–\$59,999	40	32	1.8 (1.4–2.3)	<.01
White race	84	80	1.5 (1.1–1.9)	<.01
Black race	5	8	0.5 (0.4–0.8)	<.01
Female sex	46	66	0.4 (0.4–0.5)	<.01
Animal exposure				
Had contact with cow, bull, or steer stool	2	0.4	11.5 (2.7–48.8)	<.01
Had contact with a cow, bull, or steer with diarrhea	1	0.1	11.0 (1.4–85.2)	.02
Had contact with any farm animal ^a with diarrhea	3	0.3	11.0 (2.6–46.8)	<.01
Had contact with a puppy with diarrhea	3	0.3	9.3 (2.8–30.7)	<.01
Had contact with a calf with diarrhea	1	0.2	7.0 (1.6–30.7)	.01
Had contact with calf stool	2	0.3	4.0 (1.3–12.0)	.01
Had contact with chicken stool	2	0.7	4.0 (1.5–10.7)	.01
Had contact with farm animal ^a stool	5	2	3.8 (2.0–7.0)	<.01
Had contact with a calf	3	1	3.4 (1.6–7.2)	<.01
Had a pet puppy	7	2	3.4 (2.1–5.7)	<.01
Had contact with puppy stool	4	1	3.2 (1.7–6.2)	<.01
Had contact with any animal with diarrhea	9	4	2.9 (2.0–4.3)	<.01
Had calf (or calves) on farm where lived	2	0.7	2.6 (1.1–6.2)	.03
Had contact with a cow, bull, or steer	5	3	2.4 (1.4–4.2)	<.01

(continued)

Table 3. (Continued.)

Exposure in the 7 days before illness	Percentage of patients exposed	Percentage of controls exposed	mOR (95% CI)	P
Had contact with a live chicken	4	2	2.4 (1.4–4.2)	<.01
Had contact with a farm animal ^a	10	6	2.2 (1.5–3.2)	<.01
Visited a farm	10	5	2.0 (1.4–2.8)	<.01
Visited a farm where there were animals	8	5	2.0 (1.4–2.9)	<.01
Had contact with a puppy	11	6	1.8 (1.3–2.6)	<.01
Lived on a farm	8	5	1.7 (1.1–2.4)	.01
Had contact with animal stool	22	16	1.6 (1.2–2.0)	<.01
Had contact with dog stool	9	6	1.5 (1.1–2.2)	.02
Lived on a farm where there were animals	7	4	1.5 (1.0–2.3)	.04
Had contact with >1 animal	38	30	1.4 (1.2–1.7)	<.01
Had a pet dog	38	33	1.3 (1.1–1.6)	.01
Had >1 pet	58	54	1.2 (1.0–1.4)	.05
Water exposure				
Drank untreated water from a lake, river, or stream	4	2	2.9 (1.6–5.3)	<.01
Other exposure				
Antibiotic use in the 4 weeks before illness	6	6	0.9 (0.6–1.3)	.45
Household member with diarrhea in the 4 weeks before illness	7	11	0.6 (0.4–0.8)	<.01

NOTE. mOR, matched OR.

^a Farm animals were defined as horses, goats, sheep, cattle, chickens, and turkeys.

ranged from 1.5% to 6%. The PAF for persons who had contact with farm animals was 4% (95% CI, 1%–7%) for persons \geq 12 years old and 2% (95% CI, 0.7%–2%) for children 2 to <12 years old. Although it had a large AOR in the final multivariate model, drinking raw milk had few patients exposed and a small PAF (1.5%; 95% CI, 0.4%–3%).

DISCUSSION

This large case-control study of sporadic *Campylobacter* infections identified 2 major independent food-specific risk factors for *Campylobacter* infection. The most important food-specific risk factor, based on PAF, was consumption of chicken prepared at a commercial food establishment. Combined with consumption of turkey prepared at a commercial food establishment and with consumption of undercooked chicken, which were independent food-specific risk factors, this result indicates that poultry was the dominant food source for *Campylobacter* infection during the study period. Eating other meats prepared at a commercial food establishment was the second most important risk factor (based on PAF) identified, indicating that poultry is not the only food vehicle associated with the risk of *Campylobacter* infection. Further attention to sources of and food-handling practices for poultry and meat in restaurants is needed. Consumption of poultry prepared at home was associated with a reduced risk of disease. Many other epidemiologic investigations of sporadic *Campylobacter* infections have implicated exposure to poultry as a major risk factor [6–13, 19–

24], and eating poultry prepared outside the home was a risk factor in several previous studies [7, 23–25]. Recent microbiological studies of poultry purchased at retail establishments in the United States and in the United Kingdom have shown that 68%–83% of chickens were contaminated with *Campylobacter* species [26–28], but it is not known whether poultry supplied to restaurants is more likely to be contaminated than poultry supplied to grocery stores. It is unclear why eating poultry and nonpoultry meat at home were independent protective factors in our study. Several studies have also reported that eating chicken at home significantly reduced the risk of *Campylobacter* infection [6, 7, 23, 24]. Although efforts at consumer food safety education could, perhaps, account for a reduced risk of illness associated with eating poultry prepared at home, compared with poultry prepared at a restaurant, it does not explain an independent protective effect of eating poultry prepared at home.

Investigators in the United Kingdom postulated that either bias in the selection of controls, the existence of confounding variables, or the immune status of controls could explain the protective effect of eating chicken at home found in their case-control study [6]. In contrast to the UK study, which used patient-nominated controls, we used sequential dialing for control selection so that this type of selection bias was unlikely. As a consequence of our study design, significantly more calls were required to reach case patients than to reach controls. This design may have led to a selection bias in favor of controls who were more likely to be at home and, thus, perhaps more likely

Table 4. Multivariate analysis and derived population attributable fractions (PAFs) from the Food-borne Disease Active Surveillance Network (FoodNet) *Campylobacter* case-control study, 1998–1999

Exposure	AOR (95% CI)	PAF, % (95% CI)
Ate chicken prepared at a restaurant	2.2 (1.7–2.9)	24.0 (17–30)
Ate nonpoultry meat prepared at a restaurant	1.7 (1.3–2.2)	21.0 (13–30)
Had contact with animal stool	1.4 (1.02–1.9)	6.0 (0.9–12)
Had pet puppy	3.4 (1.8–6.5)	5.0 (3–7)
Had contact with farm animals (for persons aged ≥ 12 years)	2.0 (1.2–3.6)	4.0 (1–7)
Ate turkey prepared at a restaurant	2.5 (1.3–4.7)	4.0 (1–6)
Drank untreated water from a lake, river, or stream	3.3 (1.5–7.5)	3.0 (1–4)
Ate undercooked or pink chicken	2.1 (1.2–3.4)	3.0 (1–6)
Ate raw seafood	1.9 (1.1–3.4)	3.0 (0.3–5)
Had contact with farm animals (for persons aged 2 to <12 years)	21.0 (2.5–178)	2.0 (0.7–2)
Drank raw milk	4.3 (1.3–14.2)	1.5 (0.4–3)
Ate nonpoultry meat prepared at home	0.7 (0.5–0.9)	NA
Ate chicken prepared at home	0.7 (0.6–0.9)	NA
Ate fresh berries bought at a store	0.6 (0.5–0.9)	NA
Female sex	0.5 (0.4–0.6)	NA
Ate fried chicken	0.5 (0.3–0.6)	NA
Ate turkey prepared at home	0.5 (0.4–0.8)	NA

NOTE. AOR, adjusted OR; NA, not available.

to eat at home. However, controlling for the number of calls needed to reach patients in our final multivariate model did not significantly change our findings. Furthermore, we did not identify any confounding variables that may have accounted for the association between eating poultry or meat prepared at home and a lower risk of illness. It is possible that the controls who ate chicken at home were protected from *Campylobacter* infection because they have immunity from repeated previous exposures to *Campylobacter* via contaminated poultry eaten at their home. Protective immunity to *Campylobacter* has been demonstrated [29], and protection from infection has been observed in persons who repeatedly drink raw milk, another significant risk factor [30, 31]. Further studies to determine the association of protective immunity and the risk of subsequent *Campylobacter* infection are needed.

Several studies have demonstrated that handling raw chicken in the kitchen and poor kitchen practices were associated with an increased risk of illness caused by *Campylobacter* [12, 13, 19]. We found that eating undercooked chicken, defined as chicken that was pink inside, was a significant risk factor (although it was reported by $<10\%$ of patients). However, we did not find an association with handling or cooking raw chicken at home, suggesting that either food handling at home has improved substantially since earlier studies or that collecting accurate self-reported behaviors via a questionnaire is a challenge. We did find an association between a lower risk of illness and several reported “good” handling habits such as putting packages of poultry into a separate plastic bag in the grocery store, discarding marinade

after use on raw chicken, and washing the cutting board with soap and water after it was used to prepare raw chicken. Although we did not identify frozen chicken as a protective factor, freezing chicken has been reported to lower *Campylobacter* counts by ≥ 2 logs and may represent a useful pathogen reduction measure in some settings [32].

The association of illness with eating specific foods prepared in commercial food establishments suggests that sources or preparation practices for those foods may differ between home kitchens and restaurant kitchens. Few data are available that describe the likelihood of cross-contamination or undercooking in the restaurant setting, although such events may not be rare. One such study of *C. jejuni* in commercial kitchens showed that, when *Campylobacter* was introduced via poultry, surrounding work areas became contaminated [33]. Because the infectious dose of *Campylobacter* is small, haphazard cleaning of surfaces with soap and water may not eliminate the risk of cross-contamination [34]. More detailed study of practices and procedures in commercial kitchens are warranted. This study could be conducted using systematic observation and direct measurement in a sample of restaurants. Restaurants that wish to lower their risk of transmitting *Campylobacter* should consider specifying sources of poultry and meat with low levels of *Campylobacter* contamination, such as irradiated product, frozen product, or product from producers with lower levels of contamination.

Eating nonpoultry meat prepared outside the home was also an important risk factor in this study (PAF, 21%). Cattle and

swine are known to harbor *Campylobacter* species [35], and ground beef and pork have been identified as risk factors in previous studies [8, 10]. Additionally, occupational exposure to raw meat has been associated with disease [6]. However, most previous studies recognize poultry consumption, rather than nonpoultry meat consumption, as the most significant source of sporadic *Campylobacter* infection in the United States. The findings in our study could be attributed to the changing epidemiology of *Campylobacter* infections or to the large sample size of our study.

Other risk factors identified in this case-control study, such as foreign travel, consumption of raw milk, raw seafood, untreated water, and contact with farm animals, animal stool, and pet puppies, have been reported previously [6, 8, 9, 11, 20, 21, 36–38]. Foreign travel was reported by 13% of patients in this study. A similar rate of exposure has been reported in Great Britain [39]. More than 70% of the travel among patients in our study was to Europe, Mexico, and Asia. This finding may reflect a higher risk of *Campylobacter* in these locations or it may simply indicate preferred travel destinations. *Campylobacter* infection is a well-known cause of traveler's diarrhea, and it is likely caused by consumption of contaminated food and water while abroad in both industrialized and developing countries. Because we did not control for travel in our study, we were unable to assess the specific food or water exposures that may have caused illness among travelers. However, the sources of *Campylobacter* infection identified in other developed nations have been similar to those identified in this country [3].

In our study, female subjects were found to be significantly protected from *Campylobacter* infection. The incidence of *Campylobacter* infection in the United States is higher among males than among females in all age groups [40]. Previous studies have also shown that males are at increased risk for *Campylobacter* infection; however, the reason remains unclear [13, 41, 42]. Although we did not identify major sex-specific differences in risk factors, possible explanations include sex-specific differences in kitchen and food-handling practices not identified by our questionnaire, differences in seeking medical care, and biological differences between sexes. Although young adult men may be particularly inept in the kitchen, this does not explain the protective effect of being female, which is present in all age groups. Even if adult men seek medical care more frequently than women, it is doubtful that parents would seek medical care for male children more frequently than for female children. A predominance among males in the incidence of other infectious diseases (e.g., salmonellosis and viral meningitis) has also been shown but has been limited to young children [43]. Sex-specific differences in immunity may exist but have not been documented.

There were several limitations in our study. Because they had

no diarrheal illness in the 28 days before their interview, controls may have been less likely to recall their exposures during the corresponding patients' incubation period. This possibility may have introduced recall bias to our study. The long incubation period and the amount of time between the exposure and the interview may have prompted patients and controls to provide a list of food preferences rather than definitive food exposures. This study included patients and controls from 7 FoodNet sites in several areas of the United States. The distribution of patients was consistent with surveillance data; however, there were some regional differences that may have influenced the results observed in the overall study. Some limitations are inherent in the structure of any case-control study. Respondents can only report what they observed, so many important events cannot be examined. Food-handling procedures in restaurant kitchens, for example, remain unknown. Although persons can be reasonably clear about their food preference, cross-contamination is difficult to assess. Some exposures, such as eating undercooked chicken, may be obvious only when extreme and so may be underreported. Thus, the risk associations and attributable fractions determined in this study are most appropriately understood as relative indicators of risk, rather than as absolute measures of risk associated with specific exposures.

The most recent FoodNet surveillance data indicate that the incidence of *Campylobacter* infection decreased by 27% between 1996 and 2001 [15]. The reason for this decrease may include improvements in slaughter hygiene and sanitation, the introduction of hazard analysis and critical control point management in slaughter plants, and efforts to educate foodhandlers and consumers. However, further efforts will be needed to meet the national health objective of a *Campylobacter* incidence of 12.3 cases per 100,000 population by 2010. The results of this study can be used to guide such efforts.

The findings of this US population-based *Campylobacter* case-control study were similar to those reported in smaller studies conducted over the past 20 years. The largest PAFs were due to consumption of poultry and meat prepared at restaurants. The results of this study indicate that efforts are needed by industry to reduce *Campylobacter* contamination of raw poultry and other meats. Restaurants should take measures to improve food-handling practices and ensure adequate cooking of meat and poultry in their kitchens. Consideration should also be given to irradiation or other pathogen reduction treatments of poultry and meat before sale, particularly to restaurants. Studies specifically focused on restaurants may identify potential sources of cross-contamination and identify ways to minimize the spread of *Campylobacter* in the commercial setting. Travelers are encouraged to observe the same precautions they would take at home when traveling abroad. Enhanced efforts are also needed to improve education and hand-washing

programs for farm and petting zoo visitors, especially children, to reduce the risk of infection after contact with animals [44, 45].

References

- Blaser MJ, Wells JG, Feldman RA, Pollard RA, Allen JR. *Campylobacter* enteritis in the United States: a multicenter study. *Ann Intern Med* **1983**;98:360–5.
- Mead PS, Slutsker L, Dietz V, et al. Food-related illness and death in the United States. *Emerg Infect Dis* **1999**;5:607–25.
- Friedman CR, Niemann J, Wegener HC, Tauxe RV. Epidemiology of *Campylobacter jejuni* infections in the United States and other industrialized nations. In: Nachamkin I, Blaser MJ, eds. *Campylobacter*. 2nd ed. Washington, DC: ASM Press, **2000**:121–38.
- Peterson MC. Rheumatic manifestations of *Campylobacter jejuni* and *C. fetus* infections in adults. *Scand J Rheumatol* **1994**;23:167–70.
- Mishu B, Ilyas AA, Koski CL, et al. Serologic evidence of previous *Campylobacter jejuni* infection in patients with the Guillain-Barre syndrome. *Ann Intern Med* **1993**;118:947–53.
- Adak GK, Cowden JM, Nicholas S, Evans HS. The Public Health Laboratory Service national case-control study of primary indigenous sporadic cases of campylobacter infection. *Epidemiol Infect* **1995**;115:15–22.
- Eberhart-Phillips J, Walker N, Garrett N, et al. Campylobacteriosis in New Zealand: results of a case-control study. *J Epidemiol Community Health* **1997**;51:686–91.
- Kapperud G, Skjerve E, Bean NH, Ostroff SM, Lassen J. Risk factors for sporadic *Campylobacter* infections: results of a case-control study in southeastern Norway. *J Clin Microbiol* **1992**;30:3117–21.
- Neal KR, Slack RC. Diabetes mellitus, anti-secretory drugs and other risk factors for campylobacter gastro-enteritis in adults: a case-control study. *Epidemiol Infect* **1997**;119:307–11.
- Neimann J, Engberg J, Moelbak K, Wegener HC. Foodborne risk factors associated with sporadic campylobacteriosis in Denmark. *Dansk Veterinaertidsskrift* **1998**;81:702–5.
- Schorr D, Schmid H, Rieder HL, Baumgartner A, Vorkauf H, Burnens A. Risk factors for *Campylobacter* enteritis in Switzerland. *Zentralblatt für Hygiene und Umweltmedizin* **1994**;196:327–37.
- Harris NV, Weiss NS, Nolan CM. The role of poultry and meats in the etiology of *Campylobacter jejuni/coli* enteritis. *Am J Public Health* **1986**;76:407–11.
- 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 [erratum appears in *Am J Epidemiol* **1987**;126:1220].
- World Health Organization (WHO). Statistical information on foodborne disease in Europe—microbial and chemical hazards. In: Program and abstracts of the Pan-European Conference on Food Safety and Quality (Budapest). Geneva: Food and Agriculture Organization/WHO, **2002**.
- Centers for Disease Control and Prevention. Preliminary FoodNet data on the incidence of foodborne illnesses—elected sites, United States, 2001. *MMWR Morb Mortal Wkly Rep* **2002**;51:325–9.
- Kassenborg H, Smith KE, Vugia D, et al. Fluoroquinolone-resistant *Campylobacter* infections: eating poultry outside the home and foreign travel are risk factors. *Clin Infect Dis* **2004**;38(Suppl 3):S271–8 (in this issue).
- Bruzzi P, Green SB, Byar DP, Brinton LA, Schairer C. Estimating the population attributable risk for multiple risk factors using case-control data. *Am J Epidemiol* **1985**;122:904–14.
- Kahn M, O’Fallon W, Sicks J. General population attributable risk estimation: technical report. Report no. 54. Rochester, MN: Mayo Clinic, **2000**.
- Hopkins RS, Scott AS. Handling raw chicken as a source for sporadic *Campylobacter jejuni* infections [letter]. *J Infect Dis* **1983**;148:770.
- Hopkins RS, Olmsted R, Istre GR. Endemic *Campylobacter jejuni* infection in Colorado: identified risk factors. *Am J Public Health* **1984**;74:249–50.
- Oosterom J, den Uyl CH, Banffer JR, Huisman J. Epidemiological investigations on *Campylobacter jejuni* in households with a primary infection. *J Hyg (Lond)* **1984**;93:325–32.
- Norkrans G, Svedhem A. Epidemiological aspects of *Campylobacter jejuni* enteritis. *J Hyg (Lond)* **1982**;89:163–70.
- Ikram R, Chambers S, Mitchell P, Brieseman MA, Ikam OH. A case control study to determine risk factors for campylobacter infection in Christchurch in the summer of 1992–3. *N Z Med J* **1994**;107:430–2.
- Effler P, Ieong MC, Kimura A, et al. Sporadic *Campylobacter jejuni* infections in Hawaii: associations with prior antibiotic use and commercially prepared chicken. *J Infect Dis* **2001**;183:1152–5.
- Rodrigues LC, Cowden JM, Wheeler JG, et al. The study of infectious intestinal disease in England: risk factors for cases of infectious intestinal disease with *Campylobacter jejuni* infection. *Epidemiol Infect* **2001**;127:185–93.
- Harrison WA, Griffith CJ, Tennant D, Peters AC. Incidence of *Campylobacter* and *Salmonella* isolated from retail chicken and associated packaging in South Wales. *Lett Appl Microbiol* **2001**;33:450–4.
- Jorgensen F, Bailey R, Williams S, et al. Prevalence and numbers of *Salmonella* and *Campylobacter* spp. on raw, whole chickens in relation to sampling methods. *Int J Food Microbiol* **2002**;76:151–64.
- Zhao C, Ge B, De Villena J, Sudler R, et al. Prevalence of *Campylobacter* spp., *Escherichia coli*, and *Salmonella* serovars in retail chicken, turkey, pork, and beef from the Greater Washington, D.C., area. *Appl Environ Microbiol* **2001**;67:5431–6.
- Black RE, Levine MM, Clements ML, Hughes TP, Blaser MJ. Experimental *Campylobacter jejuni* infection in humans. *J Infect Dis* **1988**;157:472–9.
- Blaser MJ, Sazie E, Williams LP Jr. The influence of immunity on raw milk—associated *Campylobacter* infection. *JAMA* **1987**;257:43–6.
- Jones DM, Robinson DA, Eldridge J. Serological studies in two outbreaks of *Campylobacter jejuni* infection. *J Hyg (Lond)* **1981**;87:163–70.
- Stern NJ, Green SS, Thaker N, Krout D, Chiu J. Recovery of *Campylobacter jejuni* from fresh and frozen meat and poultry collected at slaughter. *Journal of Food Protection* **1984**;47:372–4.
- Dawkins HC, Bolton FJ, Hutchinson DN. A study of the spread of *Campylobacter jejuni* in four large kitchens. *J Hyg (Lond)* **1984**;92:357–64.
- Cogan TA, Bloomfield SF, Humphrey TJ. The effectiveness of hygiene procedures for the prevention of cross-contamination from chicken carcasses in the domestic kitchen. *Lett Appl Microbiol* **1999**;29:354–8.
- Nielsen EM, Engberg J, Madsen M. Distribution of serotypes of *Campylobacter jejuni* and *C. coli* from Danish patients, poultry, cattle and swine. *FEMS Immunol Med Microbiol* **1997**;19:47–56.
- Salfeld NJ, Pugh EJ. *Campylobacter* enteritis in young children living in households with puppies. *Br Med J (Clin Res Ed)* **1987**;294:21–2.
- Schmid GP, Schaefer RE, Plikaytis BD, et al. A one-year study of endemic campylobacteriosis in a midwestern city: association with consumption of raw milk. *J Infect Dis* **1987**;156:218–22.
- Engleberg NC, Correa-Villasenor A, North CQ, Crow T, Wells JG, Blake PA. *Campylobacter* enteritis on Hopi and Navajo Indian reservations: clinical and epidemiologic features. *West J Med* **1984**;141:53–6.
- Cowden J. *Campylobacter*: epidemiological paradoxes [editorial]. *BMJ* **1992**;305:132–3.
- Centers for Disease Control and Prevention (CDC). 1998 Final FoodNet surveillance report. Atlanta, GA: CDC, **1999**.
- Kapperud G, Aasen S. Descriptive epidemiology of infections due to thermotolerant *Campylobacter* spp. in Norway, 1979–1988. *APMIS* **1992**;100:883–90.

42. Skirrow MB. A demographic survey of campylobacter, salmonella and shigella infections in England: a Public Health Laboratory Service survey. *Epidemiol Infect* **1987**;99:647–57.
43. Green MS. The male predominance in the incidence of infectious diseases in children: a postulated explanation for disparities in the literature. *Int J Epidemiol* **1992**;21:381–6.
44. Friedman CR, Torigian C, Shillam PJ, et al. An outbreak of salmonellosis among children attending a reptile exhibit at a zoo. *J Pediatr* **1998**;132:802–7.
45. Crump JA, Sulka AC, Langer AJ, et al. An outbreak of *Escherichia coli* O157:H7 infections among visitors to a dairy farm. *N Engl J Med* **2002**;347:555–60.