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Quantification of *Campylobacter* on the Surface and in the Muscle of Chicken Legs at Retail

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ABSTRACT

The objective of this study was to determine the prevalence and numbers of *Campylobacter* on the skin and in the muscle of chicken legs at retail to examine the external and internal contamination for an exposure assessment. Furthermore, the study assessed seasonal influence on *Campylobacter* contamination in chicken legs. Of the 140 examined skin samples, 66% were positive, and the internal contamination of 115 sampled chicken legs was 27%. The enumeration of *Campylobacter* on the surface of positive chicken legs revealed a median of 2.4 log CFU/g of skin, and the quantification of *Campylobacter* in the muscle gave results mainly under the detection limit of the most-probable-number method (<0.3 MPN *Campylobacter* per g). The external contamination was significantly higher than the internal. In both skin and muscle samples, *Campylobacter jejuni* had a much higher incidence than *Campylobacter coli*. However, with regard to the specification of *Campylobacter* on the surface of chicken legs, *C. coli* was isolated at higher colony counts than *C. jejuni*. During the 1-year study, two peaks of *Campylobacter* contamination occurred, one in the early springtime (February and March, 100 and 90%, respectively) and the second during the warmer months in the summer (July and August, both 90%). Furthermore, a positive correlation between prevalence and numbers of *Campylobacter* on chicken legs was observed.

Campylobacter species are the second most important cause of acute bacterial enteritis reported in Germany. The notification system according to the Act for Protection against Infectious Diseases confirmed 55,745 campylobacteriosis cases with an incidence of 67.5 cases per 100,000 inhabitants for 2004 *(30)*.

Human campylobacteriosis is mainly foodborne, and several case-control studies conducted in Norway, Sweden, The Netherlands, the United States, and Denmark reported that undercooked or contaminated poultry and poultry products are the most important vehicles for human infections. The high prevalence of mainly *Campylobacter jejuni* in poultry and poultry products (e.g., 58.1% of poultry meat in Germany is positive) *(12)*, but also the high quantitative contamination of fresh poultry meat *(5, 33)*, emphasizes the importance of poultry as a major risk factor for *Campylobacter* infections *(21, 27)*.

For exposure assessment as part of risk assessment, it is essential to focus on two different routes of exposure for the consumer: (i) the classic route of exposure such as that which occurs through consumption of undercooked chicken, assuming presence of *Campylobacter* in the muscle and their survival throughout the cooking process and (ii) acquiring infection via cross contamination of *Campylobacter* to hands, kitchen utensils, or ready-to-eat food during the improper preparation of contaminated poultry or poultry products. For the exposure via the second route, the contamination of the chicken skin is decisive. Because of the suitable conditions on chicken skin for the survival of *Campylobacter*, a large number of these pathogens on the surface of chicken can be assumed.

Several outbreaks associated with the consumption of undercooked chicken have been reported *(7, 8)*, whereas in other cases cross contamination of another food with *Campylobacter* from raw chicken was suspected *(16)*.

It is important to determine the prevalence and number of *Campylobacter* in chicken to gain an insight into the risk of infection with these pathogens and to prevent food poisoning after consumption of a contaminated product.

The object of the present study was to determine the prevalence and number of *Campylobacter* on the surface and in the muscle of chicken legs at retail and to investigate seasonal influence on the prevalence and number of these pathogens.

MATERIALS AND METHODS

Sample collection. From November 2003 to December 2004, 140 packages (10 each month) of fresh chicken legs were bought randomly from a variety of retail outlets located in Berlin. Each package contained of at least two fresh legs. The external qualitative and quantitative contamination was examined in 140 legs; internal contamination was examined in 115 chicken legs from February to December 2004.

Sample preparation. To determine the prevalence and the quantitative load of *Campylobacter* on the surface of chicken parts, one leg from each package was examined by removing 25 g of skin (nearly all the skin of a leg) with a flame-sterilized scalpel. The skin sample was placed in a stomacher bag containing 225 ml of Preston broth (CM 67 plus SR 117 plus SR 84 plus

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SR 48, Oxoid, Wesel, Germany) and homogenized in a Lab-Blender 400 stomacher (Seward, Calworth, UK) for 120 s (first dilution). Decimal dilutions were made in 0.1% buffered peptone water. The remaining Preston broth was poured into a flask and used for enrichment of *Campylobacter*. The same leg was used for examination of the internal contamination. After removal of the skin, the underlying surface was flamed and a 10-g muscle sample from a depth of approximately 1 cm was prepared after the superficial layers of muscles were removed aseptically with a flame-sterilized scalpel and forceps. Muscle samples were placed in a stomacher bag containing 90 ml of Preston broth, homogenized, and decimal diluted as described above.

Qualitative detection. After the decimal dilutions were prepared, the Preston broth enrichment containing 25 g of skin and 10 g of muscle, respectively, were used for the enrichment of *Campylobacter* under microaerophilic conditions (approximately 5% O_2, 10% CO_2, and 85% N_2) in a CO_2 incubator (CB 210, Binder) for 24 h at 42°C.

Enumeration of skin samples. *Campylobacter* numbers from skin samples were determined by a direct enumeration method. This was performed by spread plating aliquots of 1 ml from the first dilution step in Preston broth in duplicate onto three Karmali agar plates (CM 935 plus SR 205, Oxoid) and additional aliquots of 0.1 ml from each dilution in duplicate. All plates were incubated under microaerophilic conditions at 42°C for 48 h as described above. Karmali plates were screened for presumptive colonies; these colonies were tested via phase-contrast microscopy for typical morphology and motility, counted, and subcultured onto blood agar (MHB A, CM 405, Oxoid) for further species identification of thermophilic *Campylobacter* on the basis of Gram stain, catalase, and oxidase production, growth at 25 and 43°C, indoxylacetate hydrolysis, hippurate hydrolysis, and susceptibility to nalidixic acid and cephalotin. *C. jejuni* ATCC 33560 and *C. coli* ATCC 33559 were used as control strains.

Quantification of muscle samples. Numbers of *Campylobacter* in muscle samples were estimated by a most-probable-number (MPN) method. For this, aliquots of 1 ml from each dilution (10^{-1} until 10^{-2}) were brought into three MPN tubes containing 9 ml of Preston broth. The MPN tubes were incubated microaerophilically at 42°C for 24 h. Then, 10 μl from each tube was streaked onto Karmali agar and incubated under microaerophilic conditions at 42°C for 48 h as described above. The numbers of *Campylobacter* were estimated using the MPN table of de Man *(10)*. MPN data according to de Man give the number of bacteria present in the largest volume of a series of sample volumes (95% confidence limits). Presumptive colonies were subcultured on blood agar and further characterized as *C. jejuni, C. coli*, and *C. lari* as described above.

Statistical analysis. Statistical analysis was conducted using the software SPSS 12.0. *Campylobacter* counts from skin samples were transformed to log CFU per gram. The limit of detection for *Campylobacter* was 1 log CFU/g of skin by direct enumeration and 0.3 MPN *Campylobacter* per g of muscle by the MPN method. Eighty-five percent of the MPN indices lay in category 1 and 15% in category 2. Ninety-five percent confidence intervals were calculated for prevalence data. The distributions of numbers of *Campylobacter* in positive legs were diagrammed as boxplots giving the median and percentiles. The chi-square test was used to compare the recovery of *Campylobacter* from skin and muscle samples. The numbers of *Campylobacter* in skin and muscle samples were compared by using the Wilcoxon test. Focusing on the distribution of numbers and the differences between the two species *C. jejuni* and *C. coli*, we used the Mann-Whitney U test. The correlation coefficient between detection rate and numbers of *Campylobacter* on chicken legs was calculated with SPSS 12.0.

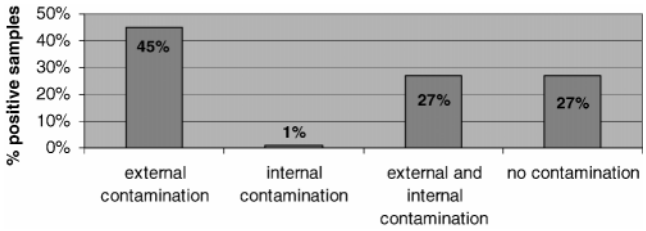

FIGURE 1. *External (direct plating, Karmali agar) and internal (MPN, Preston broth, Karmali agar)* Campylobacter *contamination of chicken legs (*n *= 115).*

RESULTS

Prevalence. In total, *Campylobacter* was prevalent in 66% (92 of 140) of the skin samples (confidence interval [CI], 58 to 74%) and 27% (31 of 115) of the muscles samples (CI, 19 to 35%). In both skin and muscle samples, *C. jejuni* was isolated more frequently (84 and 97%, respectively) than *C. coli* (16 and 3%, respectively). *C. lari* was not found. Half of all examined chicken legs (52 of 115) had positive skin but negative muscles, 27% (31 of 115) of the tested legs had positive skin and muscle samples, and 1 of 115 legs was contaminated only in the muscle. Twenty-seven percent (31 of 115) of the outside and inside examined chicken legs were *Campylobacter* negative (Fig. 1). The prevalence of *Campylobacter* in skin samples was significantly higher than in muscle samples (chi-square test, $P < 0.001$).

Quantification. Concerning the statistical parameters of skin samples ($n = 140$), a median of 1.8 log CFU/g of skin was obtained; considering only positive skin samples ($n = 92$), the median was 2.4 log CFU/g of skin. The distribution of numbers, the 25th and 75th percentiles, is shown in Figure 2. The median of positive muscle samples was 0.9 MPN *Campylobacter* per g. Prevalence and medians of *Campylobacter* in skin and muscle samples are shown in Table 1. In skin samples, we found *Campylobacter* at a significantly higher level than in the muscle samples ($P < 0.001$). In terms of specification of *Campylobacter*, we detected significantly higher numbers of *C. coli* than *C. jejuni* ($P = 0.044$) (Fig. 3).

Incidence per month. Percentages of *Campylobacter*-positive skin and muscle samples per sampling month are shown in Figure 4. The highest isolation rate of skin samples was detected in February (100%). From August to December (90, 80, 80, 70, and 60%, respectively), we observed a decline in the isolation rate of *Campylobacter* in skin samples. The highest isolation rate in muscle samples was found in September (70%). From February to April, the detection rate in muscle samples decreased but remained constant in May and June (40, 10, 0, 10, and 10%, respectively). A second decline is shown from September through December (70, 50, 30, and 0%, respectively).

We computed the median of skin samples for each

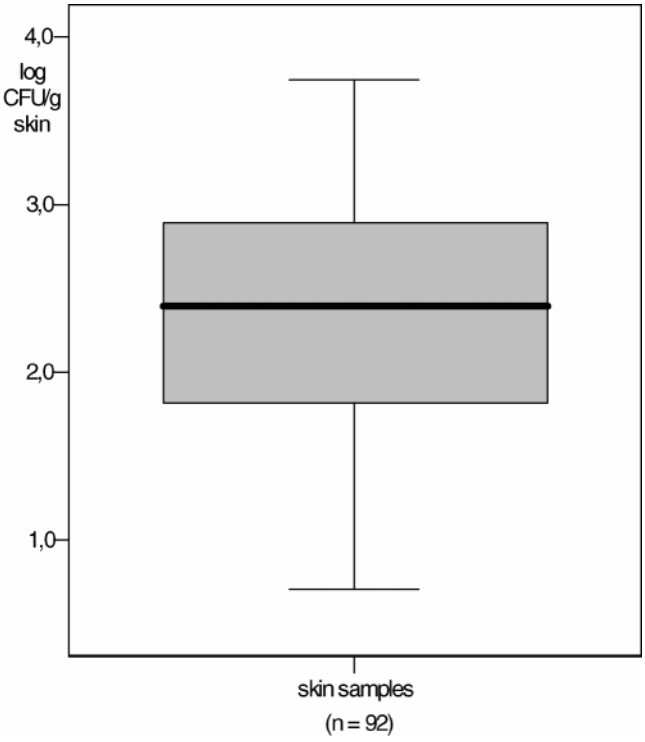

FIGURE 2. *Distribution of numbers of* Campylobacter *in positive skin samples of chicken legs (direct plating, Karmali agar); median, first, and third quartile.*

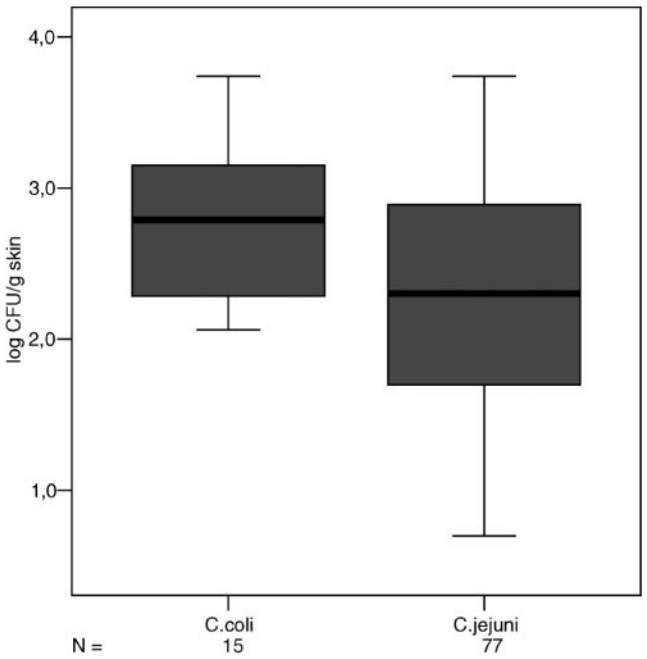

FIGURE 3. *Distribution of numbers of* C. jejuni *and* C. coli *in positive skin samples of chicken legs (direct plating, Karmali agar) (*n = *92); median, first, and third quartile.*

month and transformed it into log CFU per gram of skin. Figure 4 shows the correlation between isolation rate and medians per sampling month. The increase and decrease of *Campylobacter* counts ran parallel with the incidence. We found decreasing numbers of *Campylobacter* from September to December (2.8, 2.6, 2, and 1 log CFU/g, respectively).

The statistic evaluation showed a positive correlation between the incidence and number of *Campylobacter* on chicken legs ($r = 0.85$).

DISCUSSION

It is well known that poultry carcasses become extensively contaminated with *Campylobacter* from intestinal contents during the slaughtering process *(2, 15, 29). Campylobacter* is commonly found in the intestinal tract of chickens; the ceca and the colon especially can harbor these pathogens at a high level *(32).* In a positive flock, *Campylobacter* numbers per gram of cecal content can reach greater than 7.0 log cells per g *(5).* During processing, the intestinal tract can leak or rupture, and contents are transferred to the skin and muscle of broiler carcasses. This contamination has the potential to persist on the carcass through the course of processing *(9).* It is also likely that there is already a large number of *Campylobacter* on skin and feathers when a *Campylobacter*-positive broiler enters the processing plant. Berrang and Dickens *(4)* reported numbers of 5.4 log CFU/g of feathers and 3.8 log CFU/g of skin before scalding. *Campylobacter* has been recovered in high numbers from the carcass after scalding and plucking. Feathers can be contaminated with feces during transport, and *Campylobacter* originally associated with feathers can be transferred to the skin during the plucking process *(3).*

Several studies determined the prevalence and level of *Campylobacter* in chicken and chicken products at retail *(11, 20, 22).* Great variation in their results, depending on the sample type (whole carcass or portions, fresh or frozen), sample preparation (rinsing the carcass or homogenizing the skin), and quantification method (direct count or MPN method), prevents direct comparison of available data.

Prevalence in skin samples at retail as shown in our study are comparable to those detected in similar studies, where 83% of the examined chickens were positive with

TABLE 1. *Prevalence and median of* Campylobacter *in skin and muscle samples of chicken legs*

	Skin samples[a]	Muscle samples[b]
Prevalence	66% *Campylobacter* positive	27% *Campylobacter* positive
C. jejuni	84%	97%
C. coli	16%	3%
Median of positive samples	2.4 log CFU/g of skin	0.9 MPN *Campylobacter*/g of muscle

[a] Direct plating, Karmali agar.
[b] MPN, Preston broth, Karmali agar.

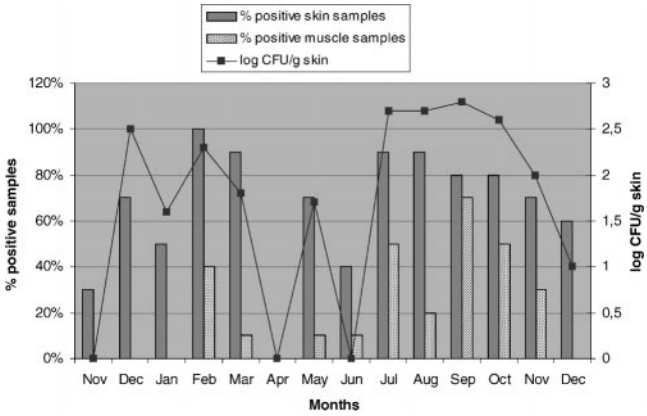

FIGURE 4. *Incidence of* Campylobacter *in skin and muscle samples (%) and medians of skin samples (log CFU per gram of skin) per sampling month.*

Campylobacter (20, 22). Recent raw chicken surveys in the United Kingdom have reported *Campylobacter* isolation rates ranging between 68 and 87% *(18, 23, 24, 26).* Altmeyer et al. *(1)* reported numbers of *Campylobacter* from 53 to 666 CFU/g of skin in fully processed broilers, which are similar to our results for we found a median of 2.4 log CFU/g of skin.

An important consideration is whether the contamination is restricted to the surface or if deeper parts of the carcasses are affected, too. The prevalence and numbers of *Campylobacter* in the muscle were low, and these results correspond with those from related studies where no *Campylobacter* was found in muscle samples of broilers *(1)* or the prevalence was very low at 3% *(2).*

Presence of *Campylobacter* in the muscle could be related to the reported premortal stress causing microbial imbalance in the intestinal flora and leading to translocation of microorganism from the gut to the blood circulation *(31).* Weakening of bacterial defenses resulting from stress may contribute to the survival of bacteria in stressed animals and their distribution to the muscle *(13).* Mengert and Fehlhaber *(25)* reported that even the usual transportation of chicken could cause an internal contamination rate of nearly 50%. They found bacteremia induced by stress in 10% of the transported chickens and contaminated thigh muscles in 23%. The translocation of *Campylobacter* has not been proved yet, but the results of our study indicate that this assumption seems to be very likely. Internal contamination might also occur when a conventional water bath stunner at currents greater than 105 mA per chicken, which increases the incidence of hemorrhaging the breast and leg muscles *(17),* is used. Under these circumstances, *Campylobacter* present in blood may be spread into the muscle.

In slaughtered animals, there is also the possibility that microorganisms are introduced into the blood via contaminated instruments. During bleeding, the knife sectioning the V. jugularis may introduce microorganisms into the blood circulation, which are transferred from the skin to the blade. Such microorganisms may disseminate by the residual blood circulation and contaminate muscle tissue *(28).* As is commonly known, a large number of *Campylobacter* is present on the skin and contamination of blood during bleeding might be possible.

Results of our study did not show any association between contamination of chicken skin and muscle, indicating different routes of contamination. Further investigations are necessary to detect possible routes of internal contamination of chicken with *Campylobacter.*

Because *Campylobacter* is killed at normal cooking temperatures, the significantly higher prevalence and number of *Campylobacter* in skin samples demonstrate that cross contamination of *Campylobacter* between raw and cooked products or contaminated contact surfaces during food preparation represents a higher consumer risk than undercooked chicken meat. The low prevalence and numbers obtained in the muscle samples indicate the lesser importance of internal contamination than external contamination. However, because of the assumed low infective dose in humans *(6),* these *Campylobacter* can cause illness if the chicken is not properly heated during cooking.

The significantly higher numbers of *C. coli* are possibly due to a comparatively higher tenacity of *C. coli* than *C. jejuni.* This advantage might contribute to a better resistance against microbial reducing steps during the slaughtering process, such as scalding, washing, or chilling, and enable *C. coli* to remain in higher numbers on the surface of chicken skin. Fernandez and Pison *(14)* isolated *C. coli* in 78% and *C. jejuni* in 22% of frozen chicken liver samples, suggesting that *C. coli* possibly survives better under environmental stress than *C. jejuni.* The higher quantitative load of chicken legs with *C. coli* has to be considered for quantitative risk assessment studies.

We obtained high recovery rates as well as numbers of *Campylobacter* in skin samples during the period of July to October but also in February and March. The prevalence in muscle samples decreased with the prevalence and numbers in skin samples from September to December and from February to April.

Willis and Murray *(34)* reported that *Campylobacter* shows a seasonal variation, with the highest contamination rate from May through October (87 up to 93%, respectively) and the lowest in December (7%) and January (33%). Similar findings are reported from a study conducted in The Netherlands, where the highest isolation rates were found from June through September (100%) and the lowest in March (50%) *(19).* Distribution of levels and numbers of *Campylobacter* on the carcass could be referred to seasonal variation in the environmental sources for chickens *(33).* Jacobs-Reitsma et al. *(19)* assumed that an elevated temperature during the warmer months of the year may influence the incidence of *Campylobacter* in broiler flocks. This corresponds with our results, as we found high prevalence in skin and muscle samples during the warmer months of the year, but does not explain the high isolation rate and number of *Campylobacter* detected in skin samples during February and March. The highest number in skin samples and the highest detection rate in muscle samples were found in September (2.8 log CFU and 70%, respectively), but the highest isolation rate in skin samples was detected in February (100%). Seasonal variation in the levels of *Campylo-*

bacter has been reported in other studies, showing the highest level during winter (16,000 CFU per carcass) and the lowest level in the spring (4 CFU per carcass) *(32)*. These diverging results indicate that further studies should be conducted to assess the influence of seasonality on detection rate and numbers of *Campylobacter* in chicken. The correlation between prevalence and numbers of *Campylobacter* in chicken legs at retail indicates that in addition to the high exposure risk due to the high incidence of *Campylobacter* in chicken products during the warmer months, there is a high exposure risk for the consumer due to the higher numbers of the agent on the contaminated product.

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REFERENCES

1. Altmeyer, M., P. Krabisch, and P. Dorn. 1985. Occurrence and distribution of *Campylobacter jejuni/coli* in broiler production. *Dtsch. Tierarztl. Wochenschr.* 92:456–459.
2. Berndtson, E., M. Tivemo, and A. Engvall. 1992. Distribution and numbers of *Campylobacter* in newly slaughtered broiler chickens and hens. *Int. J. Food Microbiol.* 15:45–50.
3. Berrang, M. E., R. J. Buhr, and J. A. Cason. 2000. *Campylobacter* recovery from external and internal organs of commercial broiler carcass prior to scalding. *Poult. Sci.* 79:286–290.
4. Berrang, M. E., and J. A. Dickens. 2000. Presence and level of *Campylobacter* on broiler carcasses throughout the processing plant. *J. Appl. Poult. Res.* 9:43–47.
5. Berrang, M. E., S. R. Ladely, and R. J. Buhr. 2001. Presence and level of *Campylobacter*, coliforms, *Escherichia coli*, and total aerobic bacteria recovered from broiler parts with and without skin. *J. Food Prot.* 64:184–188.
6. Black, R. E., M. M. Levine, M. L. Clements, T. P. Hughes, and M. J. Blaser. 1988. Experimental *Campylobacter jejuni* infection in humans. *J. Infect. Dis.* 157:472–479.
7. Blaser, M. J. 1982. *Campylobacter jejuni* and food. *Food Technol.* 36:89–92.
8. Brouwer, R., M. J. Mertens, T. H. Siem, and J. Katchaki. 1979. An explosive outbreak of *Campylobacter* enteritis in soldiers. *J. Microbiol. Serol.* 45:517–519.
9. Byrd, J. A., B. M. Hargis, D. E. Corrier, R. L. Brewer, D. J. Caldwell, R. H. Bailey, J. L. McReynolds, K. L. Herron, and L. H. Stanker. 2002. Fluorescent marker for the detection of crop and upper gastrointestinal leakage in poultry processing plants. *Poult. Sci.* 81:70–74.
10. de Man, J. C. 1983. MPN tables, corrected. *Eur. J. Appl. Microbiol.* 17:301–305.
11. Dufrenne, J., W. Ritmeester, E. Delfgou-van Asch, F. van Leusden, and R. de Jonge. 2001. Quantification of the contamination of chicken and chicken products in The Netherlands with *Salmonella* and *Campylobacter*. *J. Food Prot.* 64:538–541.
12. Federal Institute for Risk Assessment. 2004. Annual report on zoonosis in Germany, 2002. National Reference Laboratory of the Epidemiology of Zoonosis, Berlin.
13. Fehlhaber, K. 2003. Microbial risks—from animal farming to the food. *Dtsch. Tierarztl. Wochenschr.* 110:312–315.
14. Fernandez, H., and V. Pison. 1996. Isolation of thermotolerant species of *Campylobacter* from commercial chicken livers. *Int. J. Food Microbiol.* 29:75–80.
15. Genigeorgis, C., M. Hassuneh, and P. Collins. 1986. *Campylobacter jejuni* infection on poultry farms and its effect on poultry meat contamination during slaughtering. *J. Food Prot.* 49:895–903.
16. Graves, T. K., K. K. Bradley, and J. M. Crutcher. 1998. Outbreak of *Campylobacter* enteritis associated with cross-contamination of food—Oklahoma 1996. *Morb. Mortal. Wkly. Rep.* 47:129–130.
17. Gregory, N. G., and L. J. Wilkins. 1989. Effect of stunning current on carcass quality in chickens. *Vet. Rec.* 124:530–532.
18. Harrison, W. A., C. J. Griffith, D. Tennant, and A. C. Peters. 2001. Incidence of *Campylobacter* and *Salmonella* isolated from retail chicken and associated packaging in South Wales. *Lett. Appl. Microbiol.* 33:450–454.
19. Jacobs-Reitsma, W. F., N. M. Bolder, and R. W. Mulder. 1994. Cecal carriage of *Campylobacter* and *Salmonella* in Dutch broiler flocks at slaughter: a one-year study. *Poult. Sci.* 73:1260–1266.
20. Jorgensen, F., R. Bailey, S. Williams, P. Henderson, D. R. Wareing, F. J. Bolton, J. A. Frost, L. Ward, and T. J. Humphrey. 2002. Prevalence and numbers of *Salmonella* and *Campylobacter* on raw, whole chickens in relation to sampling methods. *Int. J. Food Microbiol.* 76:151–164.
21. Kapperud, G., G. Espeland, E. Wahl, A. Walde, H. Herikstad, S. Gustavsen, I. Tveit, O. Natas, L. Bevanger, and A. Digranes. 2003. Factors associated with increased and decreased risk of *Campylobacter* infection: a prospective case-control study in Norway. *Am. J. Epidemiol.* 158:234–242.
22. Kinde, H., C. A. Genigeorgis, and M. Pappaioanou. 1983. Prevalence of *Campylobacter jejuni* in chicken wings. *Appl. Environ. Microbiol.* 45:1116–1118.
23. Kramer, J. M., J. A. Frost, F. J. Bolton, and D. R. Wareing. 2000. *Campylobacter* contamination of raw meat and poultry at retail sale: identification of multiple types and comparison with isolates from human infection. *J. Food Prot.* 63:1654–1659.
24. Meldrum, R. J., D. Tucker, and C. Edwards. 2004. Baseline rates of *Campylobacter* and *Salmonella* in raw chicken in Wales, United Kingdom, in 2002. *J. Food Prot.* 67:1226–1228.
25. Mengert, U., and K. Fehlhaber. 1996. Effect of premortal stress on the internal microbial contamination of broiler carcasses. *Berl. Munch. Tierarztl. Wochenschr.* 109:28–31.
26. Moore, J. E., T. S. Wilson, D. R. Wareing, T. J. Humphrey, and P. G. Murphy. 2002. Prevalence of thermophilic *Campylobacter* in ready-to-eat foods and raw poultry in Northern Ireland. *J. Food Prot.* 65:1326–1328.
27. Neimann, J., J. Engberg, K. Molbak, and H. C. Wegener. 2003. A case-control study of risk factors for sporadic *Campylobacter* infections in Denmark. *Epidemiol. Infect.* 130:353–366.
28. Nickerson, J. T., and A. J. Sinskey. 1972. Microbiology of food processing. Elsevier Publishing Company, New York.
29. Oosterom, J., S. Notermans, H. Karman, and G. B. Engels. 1983. Origin and Prevalence of *Campylobacter jejuni* in poultry processing. *J. Food Prot.* 46:339–344.
30. Robert Koch Institute. 2005. Infection Epidemiology Year Book, 2004. Berlin.
31. Schuppel, H., K. Fehlhaber, and E. Stryczek. 1994. Internal contamination in slaughtered animals (literature review). *Berl. Munch. Tierarztl. Wochenschr.* 107:23–29.
32. Stern, N. J. 1995. Influence of season and refrigerated storage on *Campylobacter* contamination of broiler carcasses. *J. Appl. Poult. Res.* 4:235–238.
33. Stern, N.J., and M. C. Robach. 2003. Enumeration of *Campylobacter* in broiler feces and in corresponding processed carcasses. *J. Food Prot.* 66:1557–1563.
34. Willis, W. L., and C. Murray. 1997. *Campylobacter jejuni* seasonal recovery observations of retail market broilers. *Poult. Sci.* 76:314–317.