In May/June 2005 an outbreak of diarrhoeal illness occurred among company employees in Copenhagen. Cases were reported from seven of eight companies that received food from the same catering kitchen. Stool specimens from three patients from two companies were positive for Campylobacter jejuni. We performed a retrospective cohort study among employees exposed to canteen food in the three largest companies to identify the source of the outbreak and to prevent further spread. Using self-administered questionnaires we collected information on disease, days of canteen food eaten and food items consumed. The catering kitchen was inspected and food samples were taken. Questionnaires were returned by 295/348 (85%) employees. Of 247 employees who ate canteen food, 79 were cases, and the attack rate (AR) was 32%. Consuming canteen food on 25 May was associated with illness (AR 75/204, RR=3.2, 95%CI 1.3-8.2). Consumption of chicken salad on this day, but not other types of food, was associated with illness (AR=43/97, RR=2.3, 95%CI 1.3-4.1). Interviews with kitchen staff indicated the likelihood of cross-contamination from raw chicken to the chicken salad during storage. This is the first recognised major Campylobacter outbreak associated with contaminated chicken documented in Denmark. It is plausible that food handling practices contributed to transmission, and awareness of safe food handling and storage has since been raised among kitchen staff. The low number of positive specimens accrued in this outbreak suggests a general underascertainment of adult cases in the laboratory reporting system by a factor of 20.

Results

We did a retrospective cohort study among employees exposed to canteen food in the three largest companies affected (known here as A, B and C). Based on the typical incubation period of campylobacteriosis (2-5 days) [9] and reports of peak incidence on 28 and 29 May, exposure was most likely to have occurred between Monday 23 May and Friday 27 May. Self-administered paper questionnaires were distributed to employees on 15 June and information was collected on demographic details, symptoms, time of onset and duration of illness, number of days absent from work, type of healthcare contact, canteen food consumption by day (from 23 May to 3 June) and the individual canteen food items consumed in the canteen on 24 and 25 May. A case was defined as an employee in company A, B or C, who had consumed canteen food between 23 May and 3 June and who developed either diarrhoea (>3 loose stools/day) or abdominal pain and fever after 23 May.

The RFCA inspected the catering kitchen and interviewed kitchen staff about food handling practices and illness. Processed and unprocessed food specimens were collected on 9 and 13 June and examined by the RFCA. Cases were asked to submit stool samples for standard bacteriological and virological analysis. Positive Campylobacter isolates were speciated by PCR and subtyped by automated ribotyping (Riboprinter; Qualicon) using the restriction enzyme HaeIII.

Results

Of the 348 employees in companies A, B and C, 295 (85%) returned questionnaires. Of these, 47 people had not been exposed to canteen food during the study period and were therefore excluded. One questionnaire was excluded because outcome information was missing. Therefore, 247 questionnaires were included in the analysis. The median age in this cohort was 39 years (range 20–64 years), and 131 (53%) were male. Seventy nine employees met the case definition. The overall attack rate was 32%. The company-, gender- and age-specific attack rates are shown in Table 1.

Day of illness onset for 77 cases is shown in Figure 1; information on date of onset was missing for two cases. After a slight increase beginning on 26 May, the number of cases rose sharply to a distinct peak on 28 May and decreased then exponentially during the following two weeks. Nine patients provided stool samples [FIGURE 1]. Four samples (three with illness onset on 28 May, one on 29 May) were culture positive for Campylobacter, three of these samples were from employees of company A and one was from company C. One of the four isolates was discarded immediately after culturing in the diagnostic laboratory, leaving three isolates for further typing. These were all found to be C. jejuni and were found to have identical DNA
profiles by riboprinting. Stool samples from five patients (two fell ill on 28 May, the remainder on 5, 7 and 8 June) [FIGURE 1] were negative for diarrhoeagenic bacteria and viruses. The negative samples from the patients who had fallen ill on 28 May were taken 2-3 weeks after illness onset and laboratory result, Copenhagen, May-June 2005.

The cases’ main symptoms were diarrhoea (95%) and abdominal pain (86%). Nausea (43%) and fever (38%) were less frequent [TABLE 2]. The cases’ main symptoms were diarrhoea (95%) and abdominal pain (86%). Nausea (43%) and fever (38%) were less frequent [TABLE 2]. Duration of illness ranged from <1 day to 18 days, with a median of 4 days. Illness led to sick leave in 47 cases (59%), with a median of two days absent from work (range 1-7). One patient was admitted to hospital.

SELECTED DATE- AND FOOD-SPECIFIC ATTACK RATES (AR), RISK RATIOS (RR) AND 95% CONFIDENCE INTERVALS (CI) ARE SHOWN IN TABLE 3. The AR (75/204) was higher in those who ate canteen food on 25 May (RR=3.2, 95% CI 1.3-8.2) and on 26 May (AR = 70/194, RR = 1.9, 95% CI 1.0-3.7). Employees who had eaten chicken salad on 25 May had a higher attack rate than employees who had not eaten chicken salad (RR=2.3, 95% CI 1.3-4.1). Of the 54 cases, 43 (80%) recalled having a higher attack rate than employees who had not eaten chicken salad (RR=3.2, 95%CI 1.3-8.2) and on 26 May (AR = 70/194, RR = 1.9, 95% CI 1.0-3.7). Employees who had eaten chicken salad on 25 May had a higher attack rate than employees who had not eaten chicken salad (RR=2.3, 95% CI 1.3-4.1). Of the 54 cases, 43 (80%) recalled having eaten chicken salad on 25 May. Three cases had eaten chicken salad on 25 May, two could not be interviewed and one did not remember whether or not this item had been eaten. No illness was reported in the three people employed at the fifth company.

Interviews with three out of five kitchen workers revealed that raw chicken had been stored in the refrigerator directly on top of the fried chicken that was later used in the chicken salad, with the result that juices from the raw chicken are likely to have dripped onto the fried chicken. The raw chicken fillets used originated from France. Food specimens from the exposure period were no longer available in the catering kitchen at the time of inspection. However, samples were taken from the chicken fillets available in the kitchen at that time, which was a different batch of chicken from the same wholesaler and the same French producer. These chicken breast fillets tested positive for *Campylobacter*, but the isolated strain was of a different ribotype than the one isolated from the cases. Because poultry is frequently contaminated with *Campylobacter* [10], no trace-back was attempted.

**Discussion**

The results suggest that the vehicle of transmission in this outbreak was chicken salad prepared by the catering kitchen and served to employees of company A, B and C on 25 May. The likely infectious agent was *Campylobacter jejuni*. This finding is not surprising, given that consumption and handling of poultry is believed to be the primary source of *Campylobacter* infections in the developed world [11] (a recently published case-control study of sporadic *Campylobacter* infections in Denmark found fresh chicken to be the main risk factor) [12] and given that outbreaks due to cross contamination of cooked food by raw poultry have been described before [1,13]. Considering the high incidence of *Campylobacter* infections and the fact that a substantial proportion of retail chickens are known to be contaminated [2], it is surprising, however, that an outbreak like the one described here had not previously been reported in Denmark.
Our study may be limited by recall bias, as data were collected around three weeks after exposure. It is likely that some participants reported food habits rather than food items actually consumed. Therefore the true RR may be higher than the observed. Information on food items was not collected for all potential days of exposure, but there was no indication that exposure took place on days other than 25 May. No food items from the exposure period were available for testing. Exposure to chicken salad was homogeneously distributed among the age groups and can not explain the lower attack rate in older employees, which does not have a straightforward explanation.

The length of the incubation period, the rarity of secondary Campylobacter infections, the difference in clinical symptoms, and the negative culture results of all cases with late onset of illness that were submitted stool samples suggest that late cases may not be related to the outbreak. In accordance with this the RR for consumption of chicken salad increased after excluding late cases.

Around half of the employees who reported eating chicken salad on 25 May fell ill. It seems plausible that some but not all of the cooked chicken used in the chicken salad may have been cross contaminated by the raw chicken juices in the refrigerator. Therefore, the number of pathogens in the salad may have been low and heterogeneous distributed, which would explain why not all of the exposed fell ill. Immunity to Campylobacter, asymptomatic infections and incorrect recall of exposure may further explain why the attack rate was not higher than observed.

Data from this outbreak may be used to gain a rough estimate of the relationship between the number of Campylobacter cases registered in the Danish laboratory surveillance system and the true number of cases in the community. Three patients decided to see a physician as a result of their illness and had a faecal sample taken for examination, which were subsequently found to be positive for Campylobacter. The remaining five patients who submitted stool samples did so only when asked by the outbreak investigation team. Therefore, only three positive stool samples of 58 (early) cases were detected via the passive routine testing. Exposure to chicken salad was homogeneously distributed, which would explain why not all of the exposed fell ill. Therefore the true RR may be higher than the observed. Information on food habits rather than food items actually consumed.

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## References


