AN OUTBREAK OF *CAMPYLOBACTER JEJUNI* ASSOCIATED WITH CONSUMPTION OF CHICKEN, COPENHAGEN, 2005

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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.

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Introduction

Campylobacter species, particularly *C. jejuni* and *C. coli*, are important causes of acute bacterial gastroenteritis of varying severity. Symptoms include (occasionally bloody) diarrhoea, abdominal pain, fever, nausea and vomiting. In rare instances the infection is complicated by Guillian-Barré syndrome. Although the infectious dose required for infection is low, most cases are sporadic [1]. In Denmark, *Campylobacter* is the most frequent cause of bacterial diarrhoea and with an incidence of 69 per 100 000 population in 2004, *Campylobacter* accounted for more than twice the number of *Salmonella* episodes [2,3]. Despite this, and in contrast to *Salmonella*, only one large Danish outbreak has previously been described, a waterborne outbreak that occurred in the mid-1990s [4]. Foodborne *Campylobacter* outbreaks that have been registered to date in Denmark have been few and included relatively small numbers of people [5-8].

On 6 June 2005, a general practitioner reported a case of *Campylobacter* gastroenteritis on suspicion of a possible foodborne outbreak. The patient was employed in a company in Copenhagen and had mentioned that other employees had similar illness. On 7 June, the regional public health office in Copenhagen received the notification and alerted the Regional Food Control Authority (RFCA). Initial enquiries revealed that the company canteen received food from a catering kitchen that catered for eight companies, and that diarrhoeal illness had been reported among staff in seven of these eight companies and that many employees had fallen ill around 28-29 May. It was therefore likely that canteen food was implicated in disease transmission, and an outbreak investigation was launched by the RFCA and the Statens Serum Institut (SSI) to identify the vehicle of the outbreak in order to remove the source and to prevent future spread.

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Methods

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

Table 1

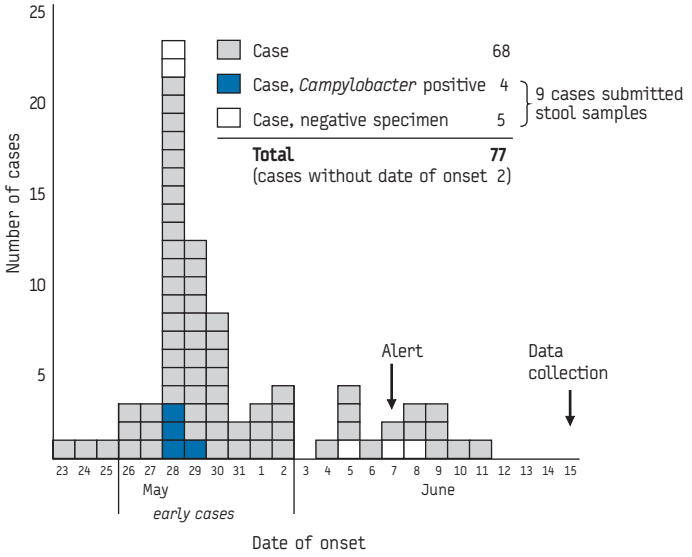
Attack rates (AR), and relative risks (RR) among employees, Copenhagen, Denmark, May-June 2005

	Cases/Total	AR%	RR	95% CI
All cases	**79/247**	**32**		
Company A-Total	50/149	34	1.2	0.7-2.0
Company B	21/76	28	Reference	
Company C	8/22	36	1.3	0.6-2.9
Sex				
Male	35/131	27		
Female	44/116	38	1.4	0.98-2.01
Age group* (years)				
20-34	26/75	35	2.5	1.0-6.2
35-49	47/122	39	2.8	1.2-6.6
50-64	6/44	14	Reference	

* chi^2= 9.3, p = 0.01, information on age was not available for 6 employees

Figure 1

Cases of acute gastroenteritis, companies A, B and C, by day of illness onset and laboratory result, Copenhagen, May-June 2005



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 onset of illness.

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 eaten chicken salad on 25 May. 25 May was the only day during the week of 23 – 27 May when chicken salad was served.

To separate potential outbreak cases from background cases, the case definition was refined. Cases with onset of illness between 26 May and 3 June were defined as 'early' cases (n=58), and cases with onset of illness after 3 June were defined as 'late' cases (n=18).

Table 2

Symptoms of cases (n = 79), companies A, B and C, Copenhagen, Denmark, May-June 2005

Symptoms*	All cases (n=79)		Early cases (n=58)		Late cases (n=18)	
	number	(%)	number	(%)	number	(%)
Diarrhoea	75	(95)	57	(98)	16	(89)
Abdominal pain	68	(86)	53	(91)	13	(72)
Nausea	34	(43)	25	(43)	8	(44)
Fever	30	(38)	26	(45)	3	(17)
Headache	15	(19)	10	(17)	4	(22)
General body ache	10	(13)	7	(12)	3	(17)
Vomiting	6	(8)	4	(7)	2	(11)
Blood in stool	3	(4)	1	(2)	1	(6)

* Multiple responses possible

Illness was of longer duration in early cases (median 4.5 days) than in late cases (median 2 days); and more early cases (42%) than late cases (5%) presented with the three concurrent symptoms of diarrhoea, abdominal pain and fever.

Individuals who had consumed canteen food on 25 May were 9.8 times (95% CI 1.4-68.3) more likely to be an early case than people who were not exposed to canteen food on that day. The relative risk of being an early case after consumption of chicken salad was 3.6 (95% CI 1.6-8.0) [TABLE 3]. For late cases there was no association between consumption of chicken salad and being ill (AR=5/97, RR = 0.7, 95% CI 0.2-2.6). Furthermore, no specific day of canteen food consumption was significantly associated with being a late case.

Telephone interviews with staff in the five other companies that had served food from the catering kitchen revealed that in four companies, at least 6 of a total of 58 employees developed a gastrointestinal illness compatible with the case definition, all of them either on 28 or 29 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 to the one described here had not previously been reported in Denmark.

Table 3

Selected[1] date- and food-specific attack rates (AR), and risk ratios (RR), of gastroenteritis, companies A, B and C, Copenhagen, 2005

	All cases (n=79)				Early[2] cases (n=58)			
Exposures	AR exposed % (ill/total)	AR non-exposed % (ill/total)	RR	95% CI	AR exposed % (ill/total)	AR non-exposed % (ill/total)	RR	95% CI
Canteen food eaten on:								
Monday 23 May	35 (70/198)	21 (8/39)	1.7	0.9-3.2	26 (51/198)	15 (6/39)	1.67	0.8-3.6
Tuesday 24 May	35 (70/199)	21 (8/39)	1.7	0.9-3.3	25 (50/199)	18 (7/39)	1.4	0.7-2.9
Wednesday 25 May	37 (75/204)	11 (4/35)	3.2	1.3-8.2	28 (57/204)	3 (1/35)	9.8	1.4-68.3
Thursday 26 May	36 (70/194)	19 (8/43)	1.9	1.0-3.7	26 (50/194)	16 (7/43)	1.6	0.8-3.2
Friday 27 May	34 (60/175)	29 (18/62)	1.2	0.8-1.8	25 (43/175)	23 (14/62)	1.1	0.6-1.8
Food items eaten on 25 May								
Chicken salad	44 (43/97)	19 (11/57)	2.3	1.3-4.1	38 (37/97)	10 (6/57)	3.6	1.6-8.0
Carrots with thyme	42 (32/77)	29 (22/74)	1.4	0.9-2.2	29 (22/77)	23 (17/74)	1.2	0.7-2.1
Mackerel	45 (22/49)	33 (36/108)	1.3	0.9-2.0	37 (18/49)	27 (29/108)	1.4	0.8-2.2
Roast beef	38 (33/86)	30 (18/60)	1.3	0.8-2.0	30 (26/86)	25 (15/60)	1.2	0.7-2.1
Rice salad	43 (13/30)	35 (40/115)	1.2	0.8-2.0	37 (11/30)	28 (32/115)	1.3	0.8-2.3
Omelette	39 (32/81)	34 (26/76)	1.2	0.8-1.7	31 (25/81)	26 (20/76)	1.2	0.7-1.9
Salad	39 (48/124)	31 (11/35)	1.2	0.8-2.1	28 (35/124)	26 (9/35)	1.1	0.6-2.1
Carrots	39 (35/89)	32 (22/68)	1.2	0.8-1.9	30 (27/89)	26 (18/68)	1.1	0.7-1.9

1. Only food items eaten on 25 May with a RR>1.1 are shown

2. Cases with date of onset of illness between 26 May and 3 June (2 cases without date of onset were counted as late cases)

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 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 heterogeneously 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 laboratory reporting system. This suggests that the underascertainment in the laboratory surveillance system among adult people is substantial, corresponding to a registration of around 1 in 20 actual cases.

In summary, this is the first recognised major *Campylobacter* outbreak associated with contaminated chicken to be documented in Denmark. The outbreak suggests that 20 actual cases may occur each time one adult case is registered. It is plausible that food handling practices contributed to the transmission, and this outbreak underlines the importance of strict measures to avoid cross contamination when handling poultry in kitchen premises. Following the investigation of the outbreak, the kitchen staff was advised on safe food handling practices.

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