

Outbreak of group A streptococcal throat infection: don't forget to ask about food

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SUMMARY

We report a large foodborne outbreak due to group A streptococci (GAS), which caused acute tonsillo-pharyngitis in 200–250 patrons of a company canteen in Copenhagen, Denmark, in June 2006. A retrospective cohort study of canteen users showed that consumption of cold pasta was associated with an increased risk of illness (attack rate 68%, risk ratio 4·1, $P < 0\cdot0001$). Indistinguishable GAS strains (*emm*89, T-type 3/13/B3264) were cultured from three cases and a cook, who had prepared the pasta. To our knowledge, pasta has previously only twice been incriminated as the source of a GAS outbreak. Only six foodborne GAS outbreaks have been reported in Europe since 1970, four of them in Sweden or Denmark. This geographical clustering suggests that foodborne GAS outbreaks are probably under-recognized elsewhere.

INTRODUCTION

Group A streptococcus (GAS), also called *Streptococcus pyogenes*, is the most frequent bacterial cause of acute tonsillo-pharyngitis ('sore throat'), primarily in school-aged children, although all age groups can be affected. Rare but severe post-infectious complications are acute rheumatic fever and glomerulonephritis [1]. Over 120 serotypes of GAS can be identified based on variations in M- and T-proteins on the cell surface [2]. Transmission is usually from person-to-person by respiratory droplets or by skin contact. Foodborne transmission of GAS and other haemolytic streptococci through raw milk, often originating from cows with streptococcal mastitis, used to be common up to the 1940s [3, 4], but has

declined sharply since the introduction of pasteurization. Nowadays, foodborne GAS outbreaks reported from industrialized countries are caused by contamination of food items during the final preparation. Food handlers identified as the source of contamination had sore throat, infected skin lesions on their hands/arms, or were asymptomatic carriers [5, 6]. Most outbreaks occurred in summer, suggesting multiplication of GAS in insufficiently refrigerated foods. We report here a large foodborne GAS outbreak, which affected 200–250 patrons of a canteen in Denmark in summer 2006. The vehicle of transmission was probably cold pasta, contaminated by a cook who was probably an asymptomatic carrier.

METHODS

Outbreak setting

On 30 June 2006, the regional food inspection authority for Copenhagen area was contacted by the

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management of a company located in a large office building. The management reported that many employees had fallen ill with acute sore throat over the previous 2 days and that some had had a positive GAS rapid test by their general practitioner. Simultaneously, several employees of other companies in the same building reported acute sore throat. The only apparent common exposure to employees of these different companies was eating in the only canteen of the building. We investigated the outbreak in order to test the hypothesis that consumption of canteen food was a risk factor and to determine if any control measures were necessary.

Epidemiological investigation

All companies having offices in the building were telephoned in order to determine the magnitude of the outbreak and possible common exposures. Following explorative interviews with several ill people, we conducted two retrospective cohort studies among the employees of the two largest companies. The aim of the first study was to test the hypothesis that illness was associated with eating in the canteen. We e-mailed a questionnaire to all employees with questions about type, onset time and duration of symptoms, sick leave taken, and results of diagnostic tests for GAS. We asked if they had eaten in the canteen on the days preceding the outbreak and what type of meal they had eaten (hot meat meal, hot vegetarian meal, cold board, salad bar). Based on these results, we sent a second questionnaire listing specific food items to canteen users to identify if any specific item was associated with an increased risk of illness.

We defined a 'primary case' as an employee of either of the two companies who reported onset of acute sore throat between 27 June noon and 30 June 2006. Questionnaire data were entered using EpiData version 3.1 [7] and statistical analysis was performed with STATA version 8.0 [8].

Microbiological and environmental investigation

The canteen was inspected and details of food preparation obtained. Food samples and swabs from kitchen utensils and surfaces were cultured for haemolytic streptococci. We interviewed all members of the canteen staff about preceding sore throat or skin infections, inspected their hands and forearms and took throat swabs from all and wound swabs where

applicable. Throat swabs were also taken from ill office employees on a voluntary basis on 12 July.

All swabs were cultured on 5% horse-blood agar and colonies showing β -haemolysis were investigated for the presence of GAS by standard methods. GAS isolates were further characterized by determination of T-type using antisera from Sevapharma (Prague, Czech Republic), sequencing of the *emm* gene coding for M-protein [9] and by pulsed-field gel electrophoresis (PFGE) using *Sma*I. *Emm* sequences were compared with the reference 'Streptococcus pyogenes *emm* sequence database' at the United States Centers for Disease Control and Prevention [10].

Literature search

We aimed to identify reports of foodborne GAS outbreaks in the European Union. We searched PubMed by using various combinations of the terms 'outbreak', 'epidemic', 'food*', 'streptococ*', 'tonsil*', 'pharyn*'. Additional reports, including those written in other European Union languages were retrieved from the references of these articles.

RESULTS

Epidemiological findings

About 1000 people worked in 16 different companies in the building. Telephone interviews with all companies showed that at least 140 persons, employed by 13 different companies, had become ill with acute sore throat in the week starting 26 June 2006. Many employees of all companies usually eat lunch at the canteen. No other shared exposures were reported. For logistical reasons, we restricted our further epidemiological investigation to two large companies with about 540 and 100 employees, respectively.

In our first study, we sent the questionnaire to all 640 employees, of whom 191 (30%) replied. Sixty-four (34%) reported acute sore throat. Frequently reported concomitant symptoms were tiredness (81%), headache (75%) and fever (72%). Forty-three cases (67%) reported at least one gastrointestinal symptom (nausea, abdominal pain, vomiting or diarrhoea), 16 (25%) had diarrhoea and seven (11%) vomiting. Fifteen persons reported a positive GAS rapid test performed by their general practitioner. The median duration of illness was 4 days (range 1–10) and 40 persons took sick leave for a median of 2 days (range 1–10), totalling 86 sick days. Only six

Table 1. Risk of illness among employees by different categories of exposure to canteen food

Exposure	Exposed			Not exposed			RR (95% CI)	Prevalence of exposure among cases
	Cases*	Total	AR (%)	Cases*	Total	AR (%)		
Eaten in canteen on								
26 June	38	116	33	2	49	4	8.0 (2.1–32.0)	95%
27 June	37	114	32	3	48	6	5.2 (1.7–16.0)	93%
Categories of food eaten by canteen patrons on 26 June								
Salad bar	31	85	36	3	18	14	2.6 (0.9–7.6)	91%
Hot vegetarian dish	9	17	53	22	83	27	2.0 (1.1–3.5)	29%
Hot meat dish	19	57	33	14	47	30	1.1 (0.6–2.0)	58%
Cold board	11	46	24	21	55	38	0.6 (0.3–1.2)	34%
Categories of food eaten by canteen patrons on 27 June								
Salad bar	31	83	37	4	21	19	2.0 (0.8–4.9)	89%
Hot vegetarian dish	9	15	60	22	83	27	2.3 (1.3–3.9)	29%
Hot meat dish	16	44	36	16	53	30	1.2 (0.7–2.1)	50%
Cold board	11	42	26	20	54	37	0.7 (0.4–1.3)	35%

Totals do not add up to the same numbers in each line due to missing data.

AR, Attack rate; RR, risk ratio; CI, confidence interval.

* Cases = people who had onset of acute sore throat between 27 June noon and 30 June 2006, referred to as ‘primary cases’ in the text. Two of 42 primary cases did not provide any information about canteen use.

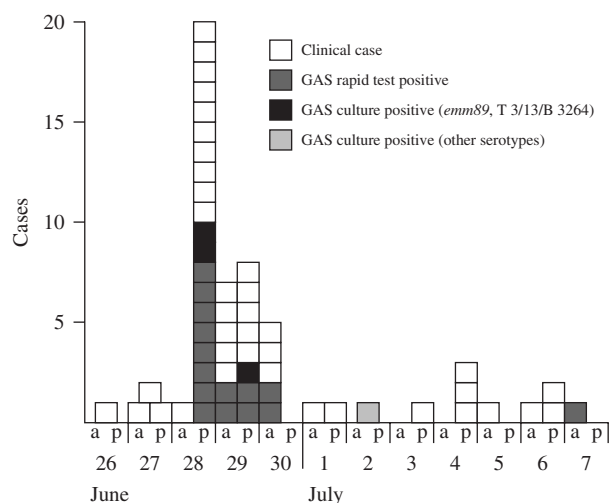


Fig. Foodborne outbreak of group A streptococcal (GAS) sore throat, Copenhagen, 2006, persons with known date of symptom onset. a=00:01–12:00 hours; p=12:01–24:00 hours.

secondary cases among household members were reported.

Based on the shape of the epidemic curve (Fig.), we decided to calculate attack rates (AR) and risk ratios (RR) for specific exposures by comparing the 42 ‘primary cases’ (symptom onset between 27 June noon and 30 June) with people who were not ill or became ill after 30 June (Table 1). The AR in people

who had eaten in the canteen on 26 or 27 June was 33% (32%), compared to 4% (6%) in those who had not. The RR was 8.0 (5.2), $P < 0.001$. Of all primary cases 95% (93%) reported eating in the canteen on 26 or 27 June.

Eating from the salad bar or the hot vegetarian dish on either day was associated with similarly increased risk of illness (RR 2.0–2.6). The proportion of exposed cases was 91% (89%) for salad bar on 26 (27) June, and 29% for the vegetarian dish on either day. Of the ten cases who reported eating the vegetarian warm dish on either day, nine also reported eating from the salad bar.

In the second analytical study, we asked the 122 patrons who had eaten in the canteen on 26 or 27 June about consumption of 13 specific food items. Sixty-four (52%) responded (Table 2). The AR and RR were highest for those who ate cold pasta from the salad bar (AR 68%, RR 4.1, 95% CI 1.9–8.8, $P < 0.0001$), and 76% of primary cases reported eating this item. In a multivariate logistic regression, it was the only exposure significantly associated with illness (odds ratio 8.9, $P = 0.001$).

Microbiological and environmental findings

The canteen was inspected on 4 July. It operated as a self-service buffet and was frequented by several

Table 2. Food specific attack rates among employees eating in the canteen on 26 or 27 June 2007

Exposure	Exposed			Not exposed			RR (95% CI)	Prevalence of exposure among cases
	Cases	Total	AR (%)	Cases	Total	AR (%)		
Food items from salad bar								
Cold pasta	19	28	68	6	36	17	4.1 (1.9–8.8)	76%
Iceberg salad	20	43	47	5	21	24	2.0 (0.9–4.5)	80%
Cucumber	20	43	47	5	21	24	2.0 (0.9–4.5)	80%
Broccoli	10	17	59	15	47	32	1.8 (1.0–3.3)	40%
Green peppers	15	37	41	10	27	37	1.1 (0.6–2.1)	60%
Egg salad	5	12	42	20	52	38	1.1 (0.5–2.3)	20%
Other food items								
Cold lamb sausage	3	5	60	22	59	37	1.6 (0.7–3.5)	12%
Cold ox tongue	2	9	22	23	55	42	0.5 (0.2–1.9)	8%
Hot pasta	12	30	40	13	34	38	1.0 (0.6–1.9)	48%
Hot chicken dish	11	34	32	14	30	47	0.7 (0.4–1.3)	44%

Only one or no cases occurred among people who reported eating marinated herring, liver paté or trout salad. AR, Attack rate; RR, risk ratio; CI, confidence interval.

hundred clients per day. Most dishes were prepared in the adjacent kitchen. The general hygiene standards were good, but the refrigeration capacity appeared insufficient. Food items inside the refrigerators including boiled pasta were stored in tightly packed large plastic containers, thereby delaying cool-down. Left-over food from before the outbreak was not available. Nine swabs from kitchen utensils and surfaces and 19 samples of currently served food including raw milk cheese were culture-negative for haemolytic streptococci.

None of the 15 staff members working in the canteen and kitchen reported sore throat or skin lesions before the outbreak. One of the cooks (cook A) had a purulent wound on his left thumb, resulting from a burn wound which he claimed to have sustained on 30 June, 3 days after the start of the outbreak. GAS were cultured from swabs of his wound and his throat. None of the other staff members had visible skin lesions on their hands or forearms, and their throat swabs were negative for haemolytic streptococci.

Cook A reported that in the week beginning 26 June he had boiled pasta for the salad bar. He admitted that before wounding his thumb, he used to stir the pasta with his bare hands when flushing it in a sink with cold water after boiling. He was not involved in the preparation of other foodstuffs for the salad bar.

Of the throat swabs taken from 29 cases on 12 July, five were positive for GAS, and one each for group C and group G streptococci. Three of these GAS isolates and those from cook A's throat and wound were

indistinguishable. They were *emm89* (corresponding to M-type 89), T-type 3/13/B3264 and showed identical PFGE patterns. The other two GAS isolates were of two distinct types (*emm118*, T-type 3/13/B3264 and *emm22*, T-type 12, respectively) and showed different PFGE patterns. All isolates were fully sensitive to penicillin.

DISCUSSION

Our results suggest that this outbreak was caused by GAS-contaminated cold pasta served in the canteen of a large office building in Copenhagen. Our first cohort study showed that almost all primary cases (95%) had eaten in the canteen. Eating from the salad bar or consuming the hot vegetarian dish were associated with a similarly increased risk of illness, the former explaining a larger proportion of cases (~90%). In a second cohort study, where we asked canteen users about consumption of particular food-stuffs from the salad bar, cold pasta was the only food item significantly associated with illness.

The isolation of indistinguishable GAS strains from three outbreak cases and from cook A supports the hypothesis that cold pasta was the outbreak vehicle. Although speculative, a plausible sequence of events was as follows: On Monday morning, 26 June, cook A – either being an asymptomatic GAS carrier or already having an infected hand wound – contaminated the pasta when stirring it with his bare hands after boiling. A too large bulk of still warm pasta was placed in the refrigerator, which delayed cool-down

Table 3. Reported foodborne outbreaks of group A streptococcal (GAS) tonsillo-pharyngitis in Europe, 1970–2006

Date of outbreak	Location	Setting	Number of cases	Incubation period*	Presumed food vehicle	Status of food handler	Ref.
July 1986	Venice, Italy	Restaurant	179	42 (8–104)	Seafood (prawn, squills), possibly custard cake	Asymptomatic GAS carrier	[14]
May 1990	Skaraborg County, Sweden	Church party	121	42 (15–115)	Hard-boiled eggs	Asymptomatic GAS carrier	[15]
June 1990	Växjö County, Sweden	Catering firm	~125	<48	Hard-boiled eggs	Asymptomatic GAS carrier	[16]
June 1995	Copenhagen County, Denmark	School party	164	~48	Pasta salad	Not reported	[11]
December 1997	Thessaloniki, Greece	Boarding college	154	?	Mayonnaise made from raw eggs	Asymptomatic, GAS status not reported	[17]
April/May 2003	Västra Götaland, Sweden	Catering firm	~200	<48	Savoury sandwich layer cake	GAS-infected skin lesion on hand	[18]
June 2006	Copenhagen, Denmark	Company canteen	~200–250	33 (?)*†	Cold pasta	Asymptomatic GAS carrier, possibly GAS-infected skin lesion on hand	‡

* Median (range) in hours.

† Time of transmission uncertain, see Discussion.

‡ Present study.

and provided an opportunity for GAS to multiply. GAS counts may still have been uncritical by lunch-time on 26 June. It seems probable that a portion of the boiled pasta was kept overnight and served at the salad bar on 27 June, by which time GAS counts were high enough to cause infection. Further bacterial growth may have occurred in the salad bar itself, because its chilling capacity was insufficient at high ambient temperature.

It was impossible to impute the probable day of transmission statistically, because most cases had eaten in the canteen on both 26 and 27 June. Assuming transmission on 27 June, we observed a median incubation period of 33 h (Fig.), which is compatible with reported mean incubation periods for foodborne GAS infection of 32–52 h (range 6–72 h) [5]. The maximum incubation period was difficult to define because cases with late onset may have been due to secondary transmission.

Pasta has to our knowledge only twice previously been reported as the vehicle of a GAS outbreak [11, 12], while the most frequently incriminated vehicles are salads or sandwiches with hard-boiled eggs [5]. Both eggs and pasta appear an excellent sterile ‘growth medium’ for streptococci if contaminated after boiling. The ability of GAS to grow on pasta at 20 °C has been demonstrated experimentally [12].

As the canteen was suspected as the source of the outbreak early on, symptomatic canteen users might have been more likely to return the questionnaire, introducing a selection bias. However, it should not have influenced the observed associations with specific foods, as there had been no rumours about ‘suspicious’ foods. The delay between exposure and the second analytical study was long (18 days), which made it difficult for people to remember what they had eaten. If anything, this may have biased the results towards an underestimation of any statistical association, but not produced a spurious association.

Efforts to determine the magnitude of the outbreak were hampered by the Danish school holidays starting a few days after the start of the outbreak. By the time of our investigation, many of the office employees, including potential cases, had gone on holiday, accounting for the low response rate. Enquiries to the management of all companies in the building revealed 140 cases. Of questionnaire respondents, 34% (64/191) reported acute sore throat, whereas 22% (42/191) met our restrictive case definition of a presumed

primary case. In summary, we estimated that 200–250 people (20–25% of the ~1000 employees in the building) became ill. This makes it one of the largest foodborne GAS outbreaks reported in Europe in the last 30 years, and the first to be associated with a workplace canteen.

The proportion of GAS-positive cultures from throat swabs of cases was low (5/29), probably due to the long delay of 16 days between infection and swabbing. Moreover, half the cases with negative swabs had received penicillin treatment. It is not surprising that apart from the suspected outbreak strain, we found four other streptococcus strains, because streptococcal tonsillitis/pharyngitis and asymptomatic carriage are common conditions. Among Danes aged 15–44 years without a sore throat, 2.3% were carriers of GAS and 3.9% of group C or G streptococci in 1983 [13]. We are not aware of any more recent carriage studies in adults.

Food handlers with acute sore throat or infected skin lesions, and also asymptomatic carriers have been reported as the source of foodborne GAS outbreaks [5, 6]. Banning food handlers with open hand wounds from work will prevent some outbreaks of GAS – and staphylococcal food poisoning. However, strict adherence to time-temperature regulations for storage of prepared foods is equally important.

We found six publications of foodborne GAS outbreaks in the European Union since 1970 (Table 3) [11, 14–18]. One outbreak each was reported from Italy and Greece, three outbreaks occurred in the southern part of Sweden and two (including the present one) in the neighbouring larger Copenhagen area in Denmark. There seems to be a clustering in the south of Scandinavia that is difficult to explain. Are foodborne GAS outbreaks not occurring in most other European countries – or are they not detected? Or are they detected, but not reported?

Foodborne transmission of GAS will only be recognized if actively looked for. Outbreaks of sore throat in families or other groups of people with close personal contact may not be perceived as foodborne. They may not be reported to an appropriate authority, or are missed because an analytical epidemiological study with few cases may lack the power to distinguish between foodborne and person-to-person transmission. In addition, health personnel may not know that GAS can be transmitted by food [15, 18], a problem which also delayed the investigation of the present outbreak. It is even more difficult to

estimate the contribution of foodborne transmission to sporadic GAS cases. Thus, the true public health burden of foodborne GAS transmission is unknown and probably underestimated.

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DECLARATION OF INTEREST

None.

REFERENCES

1. **Bisno AL.** *Streptococcus pyogenes*. In: Mandell GA, Bennett JE, Dolin R, eds. *Mandell, Douglas and Bennett's Principles and Practice of Infectious Diseases*, 4th edn. New York, Edinburgh, London: Churchill Livingstone, 1995, pp. 1786–1799.
2. **Johnson DR, et al.** Characterization of group A streptococci (*Streptococcus pyogenes*): correlation of M-protein and emm-gene type with T-protein agglutination pattern and serum opacity factor. *Journal of Medical Microbiology* 2006; **55**: 157–164.
3. **Dublin TD, et al.** Milk-borne outbreaks due to serologically typed hemolytic streptococci. *American Journal of Public Health* 1943; **33**: 157–166.
4. **Evans AC.** Types of streptococci associated with bovine mastitis followed by outbreaks of human disease. *Journal of Infectious Diseases* 1946; **78**: 18–24.
5. **Levy M, Johnson CG, Kraa E.** Tonsillopharyngitis caused by foodborne group A streptococcus: a prison-based outbreak. *Clinical Infectious Diseases* 2003; **36**: 175–182.
6. **Katzenell U, Shemer J, Bar-Dayyan Y.** Streptococcal contamination of food: an unusual cause of epidemic pharyngitis. *Epidemiology and Infection* 2001; **127**: 179–184.
7. **Lauritsen JM, Bruus M. (eds).** EpiData data entry, data management and basic statistical analysis system. Odense Denmark, EpiData Association, 2000–2006 (<http://www.epidata.dk>). Accessed 2 March 2007.
8. **Stata Corporation.** Stata Corporation, College Station, TX 77845 USA (<http://www.stata.com>). Accessed 2 March 2007.
9. **Beall B, Facklam R, Thompson T.** Sequencing *emm*-specific PCR products for routine and accurate typing of group A streptococci. *Journal of Clinical Microbiology* 1996; **34**: 953–958.

10. **Streptococcus pyogenes emm sequence database** (<http://www.cdc.gov/ncidod/biotech/strep/strepblast.htm>). Accessed 19 July 2006.
11. **Jespersen NB, Rasmussen P, Steensberg J.** Foodborne streptococcus epidemic [in Danish]. *Ugeskrift for læger* 1997; **159**: 5368–5371.
12. **Farley TA, et al.** Direct inoculation of food as the cause of an outbreak of group A streptococcal pharyngitis. *Journal of Infectious Diseases* 1993; **167**: 1132–1135.
13. **Hoffmann S.** The throat carrier rate of group A and other beta hemolytic streptococci among patients in general practice. *Acta Pathologica Microbiologica et Immunologica Scandinavica (Section B)* 1985; **93**: 347–351.
14. **Gallo G, et al.** An outbreak of group A food-borne streptococcal pharyngitis. *European Journal of Epidemiology* 1992; **8**: 292–297.
15. **Claesson BE, et al.** A foodborne outbreak of group A streptococcal disease at a birthday party. *Scandinavian Journal of Infectious Diseases* 1992; **24**: 577–586.
16. **Niklasson PM, Bank G, Nyblom R.** Outbreak of food-borne streptococcus tonsillitis [in Swedish]. *Epid aktuell* 1990; **8**: 2–3.
17. **Tsakris A, et al.** Outbreak of rare serotype of group A streptococcus pharyngitis in a boarding college. *Lancet* 1999; **353**: 1585–1586.
18. **Asteberg I, et al.** A food-borne streptococcal sore throat outbreak in a small community. *Scandinavian Journal of Infectious Diseases* 2006; **38**: 988–994.