

An outbreak of gastrointestinal illness associated with the consumption of escolar fish

Keflemariam Yohannes,^{1,2} Craig B Dalton,² Linda Halliday,¹ Leanne E Unicomb,² Martyn Kirk³

Abstract

An outbreak of gastrointestinal illness occurred amongst attendees of a conference lunch in the Hunter area, New South Wales, in October 2001. A distinctive symptom reported by many ill persons was the presence of oily diarrhoea. The Hunter Public Health Unit investigated the outbreak by conducting a telephone interview of the cohort of conference attendees using a standard questionnaire. Twenty persons out of 44 attendees (46%) became ill following the conference. The median incubation period was 2.5 hours (range 1–90 hours). The most common symptoms reported were; diarrhoea (80%) — 38 per cent of these reported oily diarrhoea; abdominal cramps (50%); nausea (45%); headache (35%) and vomiting (25%). For analyses, a case was defined as a person who developed oily diarrhea, or diarrhoea within 48 hours, or had at least two other symptoms of gastroenteritis within 6 hours, of the conference lunch. Seventeen persons had symptoms that met the case definition. None of the foods or beverages consumed were significantly associated with illness, however, all cases had consumed fish and none of those who did not eat fish (4 persons) became ill. Moreover, only 'fish' or 'potato chips' could explain a significant proportion of the illness. Analysis of the oil composition of the fish consumed was consistent with the known profile of the species marketed as 'escolar'. Among those who consumed fish the following potential risk factors did not have a significant association with the illness: Body Mass Index, age, health status and the amount of fish consumed. We concluded that consumption of fish within the marketing group escolar can cause severe abdominal cramping, nausea and vomiting, in addition to incontinent diarrhoea. *Commun Dis Intell* 2002;26:441-445.

Keywords: fish, outbreak, diarrhoea, Australia, escolar, rudderfish, wax ester

Introduction

Purgative properties are reported for members of the escolar (*Lepidocybium flavobrunneum*, *Ruvettus pretiosus*) and rudderfish (*Centrolophus niger* and *Tubia* species) marketing groups.¹ Escolar are commonly sold in the domestic market mislabeled as 'rudderfish' or 'butterfish'. Their oil profiles have been found to be very distinctive from each other and other fish species.² Studies have found that both escolar and rudderfish have higher oil composition in proportion to their wet mass (2–25%) than most seafood, but it is the high wax ester content in escolar oil (>90%) that explains the purgative property.^{2,3} In humans, wax esters accumulate in the rectum causing oily diarrhoea.³ In October 2001, the Hunter Public Health Unit received a report of diarrhoea from a person who had attended a conference lunch at a local catering centre. Further investigation identified a number of conference attendees who had developed gastrointestinal illness after attending the conference

lunch where the main meal was reported to be rudderfish. The Public Health Unit investigated the outbreak with the aim of preventing further cases of gastrointestinal illness and identifying the causative agent.

Methods

Epidemiologic investigation

The Public Health Unit conducted a cohort study of all conference attendees who attended the lunch. A list of conference attendees was obtained from the conference organisers and an effort was made to contact the entire cohort by telephone. A standard questionnaire was used to obtain information on the type and quantity of food and beverages consumed. Detailed information on clinical symptoms and duration of symptoms were also collected. In addition to the standard questions the study incorporated questions related to the use of medication, health status, height and weight and description of build that could be used to examine

1. Master of Applied Epidemiology Program (MAE), National Centre of Epidemiology and Population Health, Australian National University, ACT.

2. Hunter Public Health Unit, Wallsend, NSW.

4. OzFoodNet, c/o National Public Health Partnership, Melbourne VIC.

Corresponding author: Mr Keflemariam Yohannes, Hunter Public Health Unit, PO Box 119, Wallsend, NSW 2287, Australia. Telephone: +61 2 4924 6477. Facsimile: +61 2 4924 6490. E-mail: hunkyoha@doh.health.nsw.gov.au.

the impact of other factors on illness. Body mass index (BMI) was calculated for each interviewee (weight/height²).

Relative risks (RR) with 95 per cent confidence intervals (95% CI) were calculated to estimate measures of association between exposure and illness. To further investigate factors associated with being a case, logistic regression analysis was performed, using BMI, age, health status and the amount of fish consumed as covariates. For the logistic regression analysis BMI and age were both categorised into 2 groups with the mean as the cut off. Statistical analysis was performed using Epi Info version 6.04c and SPSS version 11.0.

For the purpose of the analysis a case was defined as a person who developed oily diarrhoea, or diarrhoea within 48 hours, or suffered at least 2 symptoms that included; nausea, abdominal cramps, vomiting or headache within 6 hours of eating at the conference lunch. Diarrhoea was defined as three or more loose stools in 24 hours.

Environmental and laboratory investigations

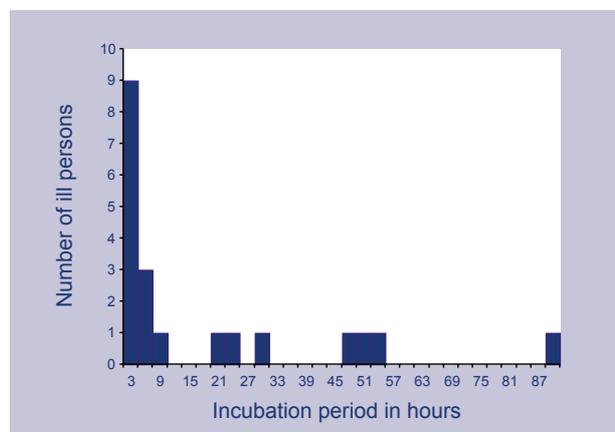
Hunter Public Health Unit Food Surveillance Officers inspected the fish market and lunch venue and reviewed food preparation and handling procedures. Two remaining pieces of fish from lunch and a sample of the oil in which it was cooked were sent to the CSIRO Marine Research Laboratory, Hobart, Tasmania, for oil content and composition analysis and possible identification of the species. The methods of analysis involved the extraction of oil with solvents and the determination of individual oil classes using an Iatroscan thin-layer chromatography-flame-ionization detector (TLC-FID) analyzer, and is reported elsewhere.²

Results

Public Health Unit officers interviewed 94 per cent (44/47) of persons who ate at the conference lunch. Of these, 46 per cent (20/44) reported gastrointestinal symptoms (Table 1). The male to female ratio for persons reporting gastrointestinal symptoms was 1.2:1. The median duration of illness was 22 hours (range 5–78 hours). The most frequent symptom reported was diarrhoea (16/20), which was also reported as the most severe symptom by 89 per cent of ill persons. Thirty-eight per cent of persons with diarrhoea described the diarrhoea as oily.

The median time between lunch and onset of illness was 2.5 hours (range 1–90 hours) (Figure). Symptoms of abdominal cramping, vomiting, nausea and headache, generally preceded diarrhoea (Table 1). Fifty-six per cent (9/16) of persons with diarrhoea reported additional symptoms, which included abdominal cramping (8/9), nausea (6/9), vomiting (3/9) or headache (4/9). Thirty-five per cent of ill persons could not perform their normal activities for a median of 2 days (range 0.5–5 days), however no one sought medical attention.

Figure. Incubation period of illness reported by persons who attended a conference lunch, Hunter, New South Wales, 2001



Seventeen ill persons (17/20) met the case definition and were included in the analysis. Of the 3 ill persons who did not meet the case definition, two had watery diarrhoea more than 48 hours after the lunch and one did not have diarrhoea but had other symptoms more than 6 hours after lunch. Food specific attack rates for cases showed that consumption of 'fish' or 'potato chips' could explain a significant proportion of the illness (Table 2). There were no cases who did not eat the fish and everyone consumed approximately the same amount of fish. No other foods or beverages showed a statistical association with illness.

Logistic regression analysis was performed on data from persons who ate fish. BMI was calculated for 39 interviewees. The mean BMI for cases was 25.8 (SD 3.2) and non-cases was 26.8 (SD 6.5). The results showed that cases and non-cases did not differ by BMI, age or health status.

Table 1. The prevalence of various symptoms among the persons who reported illness after attending a conference lunch, Hunter, New South Wales, 2001

Symptom	Prevalence (%) n=20	Median incubation period
Diarrhoea – watery only	50	4 hours (range 2–90 hours)
Diarrhoea – oily	30	4 hours (range 2–90 hours)
Abdominal cramps	50	2 hours (range 1–53 hours)
Nausea	45	2 hours (range 1–6 hours)
Headache	35	2 hours (range 1–27 hours)
Vomiting	25	2 hours (range 1–2 hours)

Table 2. Food-specific attack rates among persons who attended a conference lunch, Hunter, New South Wales, 2001

Food items	Persons who consumed item		Persons who did not consume item		Relative risk	95% CI
	Total ill	Attack rate (%)	Total ill	Attack rate (%)		
Fish	17/40	43	0/4	0.0	Undefined	Undefined
Potato chips	17/39	44	0/5	0.0	Undefined	Undefined
Apple slice	2/8	25	15/34	44	0.6	0.6–2.0
Coconut slice	2/3	67	15/40	38	1.8	0.7–4.4
Curried egg	3/7	43	13/33	39	1.1	0.4–2.8
Honeydew	1/10	10	16/32	50	0.2	0.0–1.3
Kiwi	2/9	22	15/34	44	0.5	0.1–1.8
Other foods	7/12	58	10/32	31	1.9	0.9–3.8
Pineapple	2/4	50	14/35	40	1.3	0.4–3.6
Rockmelon	6/16	38	11/27	41	0.9	0.4–2.0
Vanilla slice	3/7	43	14/37	38	1.1	0.4–2.9
Watermelon	4/17	24	13/26	50	0.5	0.2–1.2

Table 3. Oil content and composition of fish samples from the outbreak, Hunter, New South Wales 2001, compared with that of escolar and rudderfish reference specimens²

Oil content and composition		Fish samples from outbreak		Reference specimens ²	
		Sample 1	Sample 2	Escolar species	Rudderfish species
Oil content (% of wet body mass)		21.7	22.4	17.8–21.2	1.7–24.8
Oil composition (% in oil)	Wax ester	96.4	97.6	90.1–96.9	n.d. – 1.5
	Triglyceride	1.9	0.3	n.d. – 1.5	0.3–14.9
	Free fatty acids	n.d.	n.d.	n.d. – 0.7	0.6–21.6
	Polar lipids	1.7	2.1	2.1–5.7	1.3–42.1
	Hydrocarbon	n.d.	n.d.	n.d. – 1.1	n.d. – 93.4
	Diacylglyceryl ether	n.d.	n.d.	n.d. – 0.5	2.2–92.5

n.d.= not detected

Environmental and laboratory investigations

No breach of food preparation and handling procedures was detected. The results of the analysis of oil content and composition of the fish showed that the fish samples had oil content of 22 per cent (percentage of weight), which is in excess of the average Australian marine fish oil content of 1 per cent^{1,2} and 97 per cent of the oil content was wax ester (Table 3). These results are consistent with the oil content of members of the escolar marketing group (*Lepidocybium flavobrunneum*, *Ruvettus pretiosus*),^{2,4} and suggest that the fish served at the conference meal were escolar, and not rudderfish, as shown on the sale invoice to the catering venue.

Discussion

The investigation identified an outbreak of gastrointestinal disease caused by the consumption of escolar. Escolar has been described as having a purgative effect due to the high wax ester content in the oil of the fish accumulating in the rectum causing oily diarrhoea. In this outbreak we identified the effects of escolar consumption to involve more severe symptoms of gastrointestinal illness, including diarrhoea, nausea, headaches, abdominal cramps and vomiting. It is unclear why some people who consumed fish became ill and some did not. In this investigation BMI, age, health status and the amount of fish consumed did not affect the outcome.

There is a paucity of information describing the symptoms associated with escolar consumption and as a result it may be an under-recognised cause of gastrointestinal disease. The health effects of the consumption of escolar are not well described in literature. Berman *et al* (1981)³ distinguished the effects of wax ester from hydroxyoleic acid, which is the purgative element in castor oil. They claimed that consumption of wax ester resulted in a passing of accumulated oil in the rectum, while consumption of the hydroxyoleic acid caused diarrhoea by some irritant effect on the bowel. Therefore, they proposed to label the diarrhoea caused by escolar as keriorrhoea, a Greek word to mean flow of wax. However, this suggestion was based on symptoms described by only two cases. We found that cases suffered not only the inconvenience of incontinent diarrhoea, but also abdominal cramps, nausea, headache and vomiting.

While BMI, age and health status and amount of fish consumed were not associated with illness, there are other factors that could mediate the severity and occurrence of the gastrointestinal effects of reported escolar consumption. These factors include variability in wax ester content of different fillet cut depths and mixing of fillets from different fish species sold as 'rudderfish' at the wholesale or retail levels. These may result in differential exposure in a cohort of consumers.

A limitation of this study was that we did not collect stool samples for microbiological analysis from those who were ill because 4 days had lapsed before the event was reported. In the absence of any stool samples from ill persons, we could not rule out that the illness was caused by an infectious pathogen. Toxicity such as histamine poisoning from fish is not a likely explanation, as the onset is more rapid (45 minutes) than the incubation period observed in this outbreak and symptoms differ as histamine poisoning symptoms usually include fever, flushing and rapid pulse rate.⁵ The lack of illness among those who did not consume the fish did not allow a relative risk to be calculated, however consumption of 'fish' or 'chips' explained the highest proportion of the illness reported. Potato chips are not a plausible cause of the illness. Although glycoalkaloids found in potatoes can cause illness, the oral dose required for such effects is higher than would be expected from a serve of potato chips⁶ and neurological disorder was not reported by members of the cohort. In this study potato chips have a strong correlation with consumption of fish. Dose response could not be assessed from this study, as there was little variation in the amount of fish consumed by each person.

This investigation highlights the need for escolar hazard guidelines to protect both traders and the public. There may be a number of restaurateurs and caterers that are unaware of the potential health effect of escolar. Escolar, a deep-sea fish of the tropical and temperate oceans, is harvested by long line trawlers from southern Queensland, along the south of the continent and up the Northwest Shelf of Western Australia.¹ In New South Wales, more than 60 tonnes of fish is marketed annually under the label of 'rudderfish' at one auction house alone (Sydney Fish Market, Information Sheet, 17 August 2001). In Japan, the Ministry of Health prohibits the sale of the two species of escolar.⁴ In its 1998 hazard guide, the United States of America Federal Drug Administration recommended that *Lepidocybium flavobrunneum* not to be marketed in interstate commerce.⁷ There may be a need for greater education of fish wholesalers and retailers to prevent future outbreaks. Our investigation also highlighted that

selling escolar as 'rudderfish' may indicate a breakdown in quality control in the fish industry. It is important to correctly identify species at the wholesale level to ensure that only species suitable for human consumption are sold. The Department of Agriculture, Fisheries and Forestry – Australia is currently addressing this issue. In April 2002 its committee for seafood marketing names made recommendations for public consultations aimed at resolving existing misidentification and mislabeling of escolar and rudderfish.

Acknowledgments

We would like to thank Ben Mooney, Peter Nichols and Nick Elliott of the CSIRO Marine Research, Hobart, Tasmania for conducting the fish oil analyses and commenting on the manuscript and Rod Thomson and Jodi Booth, Food Surveillance Officers at the Hunter Public Health Unit, New South Wales for investigating the food premises.

References

1. Yearsley GK, Last PR, Ward RD. Australian Seafood Handbook: an identification guide to domestic species. CSIRO Marine Research, Australia 1999:365–375.
2. Nichols PD, Mooney BD, Elliott NG. Unusually high levels of non-saponifiable lipids in the fishes escolar and rudderfish identification by gas and thin-layer chromatography. *J Chromatogr A* 2001;936: 183–191.
3. Berman P, Harley EH, Spark AA. Keriorrhoea – the passage of oil per rectum – after ingestion of marine wax ester. *S Afr Med J* 1981;59:791–792.
4. Kawai N, Nakayama Y, Matsuka S, Mori T. Lipid composition of various tissues of *Lepidocybium flavobrunneum*. *Yukagaku (Japan Oil Chemists' Society)*; 1985;34:25–31.
5. Hughes JM, Merson MH. Fish and shellfish poisoning. *N Engl J Med* 1976;295:1117–1120.
6. Jose C. Food toxicology: principles and concepts part A. New York: Marcel Dekker Inc; 1988.
7. USA Food and Drug Administration, Centre for Food Safety and Applied Nutrition. Managing food safety: a HACCP principles guide for operators of food establishments at the retail level. 1998. Available from: <http://www.cfsan.fda.gov/~dms/hret-al.html>. Accessed 14 May 2002.

Gastroenteritis outbreak in a sporting team linked to barbecued chicken

Paul Armstrong,^{1,2} David Peacock,¹ Scott Cameron²

Background

On 25 May 2001, the Centre for Disease Control, Northern Territory Department of Health and Community Services in Darwin, was alerted by local media reports to an apparent outbreak of gastroenteritis that occurred in a visiting interstate sporting team 2 days before. The 16-member team was competing in the Arafura Games, a biennial, international sports competition conducted in Darwin. After corroborating the report by interviews with the team management and by reviewing hospital records, an outbreak investigation was initiated.

Methods

Hypothesis generating interviews were conducted with the team members. Information was collected regarding food consumption history, demographic details, symptomatology, and time of illness onset. From these interviews, a meal organised for team members only and consumed several hours prior to onset of symptoms by affected team members, was identified as the likely source of the outbreak. A retrospective cohort study was conducted to determine any link between illness and eating particular foodstuffs at this meal. The case definition was defined as: 'any member of the team who ate at this team meal (commencing 11pm 23 May) and who became ill with one or more symptoms of vomiting, abdominal pain or diarrhoea, from 11pm, 23 May to 11am, 24 May'. The information was entered into a database using Epi Info Version 6 software. Relative risks were calculated for each food item.

The then Territory Health Services Environmental Health team investigated the food handling practices of the supermarket delicatessen where the food items consumed at the common team meal were purchased. Their aim was to identify potential environmental source(s) of the foodborne illness, and enforce public health legislation where appropriate.

Results

Epidemiological investigations

Descriptive study

On 23 May 2001, after their sporting commitments were completed, the team and their management met at their hotel for a late evening meal consisting of food purchased from a supermarket 6 hours prior to the meal. The foods purchased were 3 hot barbecued chickens, potato salad, coleslaw, bread rolls, fruit juice in small cartons, and confectionary. Soon after they were purchased, one of the team unpacked and handled one of the chickens and placed it on the only plate available (denoted 'plate chicken' in analytical study below). The other chickens were left in their wrappers untouched ('wrapper chicken') and all the chickens, as well as the other food items, were refrigerated until the meal commenced, 5¹/₂ hours later. Between 2¹/₂ and 4 hours after the meal commenced, 6 members of the team (3 male, 3 female; age range 18-26 years) became unwell, initially with malaise (5/6), severe vomiting (5/6) and crampy abdominal pain (4/6), and diarrhoea some hours later (6/6). Five presented to an accident and emergency department and all were discharged after receiving supportive treatment. No samples were obtained for microbiological diagnosis. No other clusters of acute gastrointestinal disease were reported around the time of this outbreak, neither in the hotel where the team were residing nor elsewhere in Darwin.

Analytical study

All 16 team and staff members completed the questionnaire (100%). Two members, who did not participate in the evening team meal and remained well, were excluded from the analysis. Six members of the team had symptoms in keeping with the case definition, giving an overall attack rate of 6/14 (37.5%).

1. Centre for Disease Control, Northern Territory Department of Health and Community Services, NT.

2. National Centre for Epidemiology and Population Health, Australian National University, ACT.

Corresponding author: Dr Paul Armstrong, Centre for Disease Control, Northern Territory Department of Health and Community Services, PO Box 40596, Casuarina NT 0811. Telephone +61 8 8922 8401. Facsimile: +61 8 8922 8310. E-mail: paulk.armstrong@nt.gov.au.

The number of team members who ate the various food items, and the relative risks for becoming ill, are shown in the Table. Two team members who ate chicken could not remember which chicken they ate; one became ill and one did not. These 'unknowns' can be analysed in a number of ways in calculating relative risks for eating the two types of chicken. The most conservative approach, assuming the former ate wrapper chicken and the latter ate plate chicken, yielded a relative risk for eating plate chicken of 5.0 (Table).

Environmental investigation

At the time of the site inspection, acceptable standards of food safety practices were observed and there were no potential sources of the outbreak identified.

Discussion

This small outbreak of an acute gastrointestinal illness has all the hallmarks of food poisoning due to a pre-formed toxin produced by an enterotoxin-producing bacterium, although microbiological proof is lacking. The short incubation period with abrupt onset, the symptomatology, and the short, self-limiting nature of the illness, are all typical of disease caused by either of the 2 pathogens that are commonly implicated in such illnesses, *Staphylococcus aureus* and *Bacillus cereus*.¹ Illness caused by *B. cereus* is usually associated with eating boiled or fried rice that has been cooked and kept warm for an extended period.^{2,3} In this outbreak, *S. aureus* was considered to be the more likely cause, being a commonly recognised

aetiological agent for foodborne outbreaks associated with poultry,^{1,4} the likely vehicle for enterotoxin in this outbreak.^{5,6} High salt foods like commercial barbecued chickens favour the growth of *S. aureus* over other bacteria.

The most conservative estimate of relative risk for eating 'plate chicken' in our analysis was 5.0, making it the most likely food vehicle. The 'plate chicken' may have become contaminated whilst it was in the store, either prior to cooking or during handling by store employees after cooking, or during handling by the purchaser. However, there were no other reports of food poisoning in the region around the time of this outbreak and the conclusion of the environmental investigation was that food handling practices of the store were acceptable. It is more likely that the team member who handled the food was the source of contamination, especially considering the 2 chickens that were not handled by this team member were not associated with illness. The considerable heat load on the team's motel refrigerator when all of the food items were placed within it several hours prior to the meal, could have slowed the rate of cooling of the chickens, thereby allowing enterotoxin to be produced in sufficient quantities to cause disease.

There was a failure of the notification procedure at the beginning of this outbreak which delayed the initiation of the investigation. Gastroenteritis is a notifiable condition in the Northern Territory if it occurs in an institution, in a food handler, or if two or more cases that are apparently related are recognised. The 5 cases who presented to hospital were clearly related yet were not notified because

Table. Association between exposure to a particular food item eaten at the evening meal and symptoms of an acute gastrointestinal illness

Food item	No. ill team members		No. well team members		RR
	Ate item	Did not eat item	Ate item	Did not eat item	
'Plate' chicken	5	1	2	6	5.0
'Wrapper' chicken	3	3	4	2	0.8
Potato salad	4	2	5	3	1.1
Coleslaw	3	3	5	3	0.8
Fruit juice	4	2	6	2	0.8
Bread rolls	5	1	7	1	0.8
Confectionary	4	2	6	2	0.8

the treating team was unaware of the necessity to do so. Immediate remediable action included a presentation to Accident and Emergency staff regarding notification requirements pertaining to diseases likely to be seen in their setting. This information will be incorporated into the regular presentation given by the Centre for Disease Control to Accident and Emergency medical staff given at times of staff turnover.

In the setting of an apparent cluster of related cases, efforts should have been taken to collect specimens for microbiological analysis. In the Accident and Emergency Department, appropriate specimens would have included faeces for microscopy/culture of conventional enteric pathogens, and vomitus and faeces for microscopy, culture and enterotoxin testing for *S. aureus* and *B. cereus* (enterotoxin testing is normally only available at public health laboratories). In suspected foodborne outbreaks caused by *S. aureus* or *B. cereus*, further specimens should ideally be taken during the epidemiological and environmental investigation. These would include hand and nasal swabs from the food handler for culture of *S. aureus*, and samples of the implicated food (if it is still available) for culture and enterotoxin testing for both organisms.¹ With regard to *S. aureus*, valuable epidemiological evidence can potentially be gained from matching phage-types isolated from the food handler, the food items, and the case. Less important is obtaining samples from fomites associated with food preparation, such as the plate that the implicated chicken was stored and served upon, as these are unusual sources of contamination with enterotoxin producing organisms. Because the illness caused by these organisms is a short self-limiting one, and the organism and enterotoxin are cleared relatively quickly, effort should be made to collect the samples within 48 hours after onset of symptoms.

The public health consequence of foodborne outbreaks caused by enterotoxin-producing bacteria is mainly morbidity associated with a short term, often incapacitating illness, but one that rarely leads to death or long term health sequelae. Unlike foodborne outbreaks where the mechanism of spread is waterborne or by the faecal-oral route, food poisoning outbreaks due to preformed enterotoxin ingestion are not self-perpetuating. Apart from physical discomfort experienced by affected team members, and disruption to their sporting program, no other adverse public health consequences eventuated in the outbreak described here.

In summary, this small outbreak of an acute gastrointestinal illness linked to barbecued chicken has features that strongly suggest an enterotoxin-producing bacterium as the causative agent, although microbiological proof is lacking. It is not possible to be definitive about the cause of the contamination of the chicken but the most likely scenario is that the team food-handler was the source. Although mortality and longer-term morbidity are uncommon with food poisoning caused by enterotoxin-producing bacteria, this outbreak highlights its capacity to cause short term, moderately-severe illness in a young and healthy population. It underscores the need for proper food handling practices, both in-store and by the consumer, and reinforces the importance of appropriate microbiological specimen collection from cases of apparent gastroenteritis outbreaks, as well as the public health importance of timely notification of such outbreaks.

Acknowledgements

With thanks to John Bates from Queensland Health Scientific Services, and Agnes Tan from the Microbiology Diagnostic Unit at the University of Melbourne, for advice regarding enterotoxin testing of human samples.

References

1. Tauxe RV, Swerdlow DL, Hughes JM. Foodborne disease. In, Mandell GL, Bennet JE, Dolin R, ed. *Mandell, Bennet and Dolin's Principles and Practice of Infectious Diseases*. Philadelphia; Churchill Livingstone, 2000:1150-1165.
2. Terranova W, Blake PA. *Bacillus cereus* food poisoning. *N Engl J Med* 1978;298:143-144.
3. Raevuori M, Kiutamo T, Niskanen A, Salminen K. An outbreak of *Bacillus cereus* food-poisoning in Finland associated with boiled rice. *J Hyg (Lond)* 1976;76: 319-327.
4. Crerar SK, Dalton CB, Longbottom HM, Kraa E. Foodborne disease: current trends and future surveillance needs in Australia. *Med J Aust* 1996;165:672-675.
5. Wieneke AA, Roberts D, Gilbert RJ. Staphylococcal food poisoning in the United Kingdom, 1969-90. *Epidemiol Infect* 1993;110:519-531.
6. Holmberg SD, Blake PA. Staphylococcal food poisoning in the United States. New facts and old misconceptions. *JAMA* 1984;251:487-489.