PHR 250, 2007
5/23/07, 6p

FIRST EPIDEMIOLOGICAL PROBLEM SET

DIARRHEA AT A WAKE IN ADELAIDE, AUSTRALIA

Thirteen out of 32 persons from two states who attended a lunch after a funeral in Adelaide on 2nd June 1998 became ill with diarrhea. Most had onset of illness within 3 days, but one case occurred 8 days after, and one 28 days after the lunch. The person whose illness commenced 8 days after was the sister of one of the early onset cases, and it was her boyfriend who became ill after 28 days. The duration of illness ranged from 3 to 10 days (mean=6.2 days) with reported symptoms in addition to diarrhea being abdominal pain (11), vomiting (7) and macroscopic blood in the stool (3).

Only one person had a history of recent travel outside Australia before the funeral. This had been a medically uneventful trip to the Philippines 6 weeks previous. Similarly, in the 2 months before the funeral one person had returned from southern Queensland, one from a trip to Western Australia and the Northern Territory and one from Western Australia only. Of these travelers, only the third had suffered any illness during their travel. Six people came from Melbourne to Adelaide to attend the funeral.

Most of the food for the function was purchased the day before from the refrigerated counter of a retail outlet, transported for 20 minutes in the boot of a car and then refrigerated overnight in the kitchen of the flat where the lunch was served. During that night one item (sliced ham) was removed from the fridge and some of it used. The remainder of the ham was returned to the fridge. The person who handled the ham during the night had recovered earlier that week from a diarrheal illness contracted in the Kimberleys (northwestern Australia — across the continent from Adelaide). The cause of this diarrhea had not been determined.

Pathogens of a single genus, but at least two species, were grown from the stool of five of the cases (onsets 1, 2 (n=2), 8, and 28 days after the funeral). None of the other cases provided a stool specimen.

A cohort study implicated only the sliced ham of the foods served at the funeral lunch as a possible vehicle for this outbreak. Even including a probable secondary case (8 day incubation) who did not eat ham as a primary case and counting one of the early cases (who was not completely certain that she had eaten the ham) as a non-consumer, the relative risk was 2.77 (95% confidence limits 1.05-7.27). With these conservative case definitions the attack rate for ham eaters was 8 out of 13 (62%). The more likely situation with the late onset case as a secondary case and accepting the history that the uncertain persons belief that she probably had eaten ham gave a relative risk of 5.46 (95% confidence limits 1.40-21.27).The attack rate with these definitions was 9 of 14 (64%).

The retailer from whom the ham was purchased is a large supplier which turns over multiple legs each week. Inspection of this premises 2 weeks after the funeral by an

environmental health officer of the Adelaide City Council revealed no poor food handling practices. Laboratory cultures of ham collected at that time did not yield a pathogen. If there had been a problem at or before the retail stage we would also have expected more metropolitan cases notified unrelated to the funeral.

The suspicion is that the person recently recovered from diarrhea acquired in the north west of Australia, who handled the ham the night before the funeral, contaminated it. This person also consumed the ham but did not suffer further illness. Some person-to-person transmission at the lunch was also possible. The meal was served to a large group of people in a very small flat and one person reported that the hand towel in the bathroom became sodden from hand wiping during the afternoon. The multiple-species isolates, especially in a metropolitan outbreak, are surprising but it seems unlikely that there would be multiple sources. Nevertheless the sole isolate of an alternate species in the genus came from a person who did not eat ham and who had a flu-like illness on the day of the funeral but developed prolonged (10 days) of diarrhea the day after.

Communicable Disease -- Australia Vol. 22; No. 13, Dec. 24/98 (FSnet 12/31/98)

1. What is the likely pathogen in this outbreak?

2. What things might have been done to prevent the outbreak?

SEVERE ILLNESS FROM RAW OYSTERS — FLORIDA, 1981–1992

From 1981 through 1992, 125 persons with similar, often oyster-associated infections, of whom 44 (35%) died, were reported to the Florida Department of Health and Rehabilitative Services (HRS). The infections generally occurred each year from March through December and peaked from May through October. Seventy-two persons (58%) had primary septicemia, 35 (28%) had wound infections, and 18 (14%) had gastroenteritis. In patients with primary septicemia, 58 infections (81%) occurred among persons with a history of raw oyster consumption during the week before onset of illness. The mean age of these persons was 60 years (range: 33-90 years; standard deviation: 12.9 years); 51 (88%) were male. Fourteen (78%) of the patients with gastroenteritis also had raw oyster- associated illness. Their mean age was 49 years (range: 19-89 years; standard deviation: 25.7 years); seven (50%) were male.

Of the 40 deaths caused by septicemia, 35 (88%) were associated with raw oyster consumption. Nine of these deaths occurred in 1992. The case-fatality rate from raw oyster-associated septicemia among patients with pre-existing liver disease was 67% (30 of 45) compared with 38% (5 of 13) among those who were not known to have liver disease.

Results of the 1988 Florida Behavioral Risk Factor Survey (BRFS) were used to estimate the proportions of the Florida population who ate raw oysters, and the proportion of the population who ate raw oysters and who believed they had liver disease (e.g., cirrhosis). BRFS and state population data indicate that approximately 3 million persons in Florida eat raw oysters; of these, 71,000 persons believe they have liver disease. Based on the number of cases reported to the Florida HRS during 1981-1992, the annual rate of illness for adults with liver disease who ate raw oysters was 72 per 1 million adults — 80 times the rate for adults without known liver disease who ate raw oysters (0.9 per 1 million). The annual rate of death from this cause for adults with liver disease who ate raw oysters was 45 per 1 million — more than 200 times greater than the rate for persons without known liver disease who ate raw oysters (0.2 per 1 million).

1. What was the most likely pathogen?

2. What options exist to limit or prevent such illnesses in the future?

GASTROENTERITIS ASSOCIATED WITH CORNED BEEF SERVED AT A ST. PATRICK'S DAY MEAL — VIRGINIA

On March 28, 1993, 115 persons attended a traditional St. Patrick's Day dinner of corned beef and cabbage, potatoes, vegetables, and ice cream. Following the dinner, 86 (76%) of 113 persons interviewed reported onset of illness characterized by diarrhea (98%), abdominal cramps (71%), and vomiting (5%). The median incubation period was 9.5 hours (range: 2-18.5 hours). Duration of illness ranged from 1 hour to 4.5 days; one person was hospitalized.

Corned beef was the only food item associated with illness; cases occurred in 85 (78%) of 109 persons who ate corned beef compared with one of four who did not (relative risk=3.1; 95% confidence interval=0.6-17.1). Cultures of stool specimens from eight symptomatic persons all yielded $\geq 10^6$ colonies of a bacterial pathogen per gram. A refrigerated sample of leftover corned beef yielded $\geq 10^5$ colonies of the same agent per gram.

The corned beef was a frozen, commercially prepared, brined product. Thirteen pieces, weighing approximately 10 pounds each, had been cooked in an oven in four batches during March 27-28. Cooked meat from the first three batches was stored in a home refrigerator; the last batch was taken directly to the event. Approximately 90 minutes before serving began, the meat was sliced and placed under heat lamps.

Following the outbreak, Virginia health officials issued a general recommendation that meat not served immediately after cooking be divided into small quantities and rapidly chilled to $\leq$40°F ($\leq$4.4°C), and that precooked foods be reheated immediately before serving to an internal temperature of $\geq$165°F ($\geq$74°C).

1. What was the most likely pathogen in this outbreak?

2. What was the most likely problem with chilling?

3. Assuming that rapid chilling had not occurred, would the reheating recommendation have prevented this outbreak?

GASTROENTERITIS IN U.S. ARMY TRAINEES — TEXAS, 1998

During August 27-September 1, 1998, 99 (12%) of 835 soldiers in one unit at a U.S. Army training center in El Paso, Texas, were hospitalized for acute gastroenteritis (AGE). Their symptoms included acute onset of vomiting, abdominal pain, diarrhea, and fever. Review of medical center admission records for AGE during the previous year indicated that fewer than five cases occurred each month.

A U.S. Army Epidemiologic Consultation Service (EPICON) team reviewed data from the inpatient records of 90 ill soldiers. AGE was defined as three or more loose stools and/or vomiting within a 24-hour period in a soldier or employee at the training center during August 26-September 1. Illness was accompanied by a minimally elevated leukocyte count, mild thrombocytopenia, and low-grade fever. The median duration of hospitalization was 24 hours (range: 12-72 hours). Stool samples collected from persons with AGE on hospital admission were negative for bacterial and parasitic pathogens.

Interviews with food handlers in the base's two dining facilities (DF1 and DF2) revealed illness in a confection baker, who had become ill in DF1 while baking crumb cake, pie, and rolls on August 26. One other DF1 employee who was not a food handler also reported self-limited gastrointestinal illness during August 27-29. No worker in DF2 reported illness.

Cultures of food specimens from the ice cream dispenser in DF1 grew nonpathogenic coliform bacteria (*Citrobacter diversus* and *Serratia liquefaciens*); however, the sample was at room temperature before culture. *Enterobacter cloacae* coliform bacteria were cultured from the soda fountain in DF2. Water samples taken from multiple sites in the training compound and from elsewhere on post were all negative for coliform contamination.

A questionnaire about food preferences, based on the previous week's menu, was administered to 86 hospitalized soldiers (84 of whom had eaten in DF1 during the 10 days before answering the questionnaire) and to 237 randomly selected soldiers from the training unit. Of the 237 nonhospitalized soldiers, 41 (17%) did not eat at DF1 during the 10 days before answering the questionnaire; 40 (17%) had illnesses that met the case definition. Thus, cases of AGE were characterized in 126 soldiers (Figure 1).

To determine the point source of the outbreak, cases with onset during August 27-28 (n=98) were analyzed separately for odds ratios (ORs) of selected exposures (Table 1). The univariate OR for illness associated with dining at DF1 during the week before the outbreak was 9.8 (95% confidence interval=2.8-40.2). Two soldiers who ate exclusively at DF2 became ill, and one ill soldier reported not eating at either facility. Food items (crumb cake, pie, cinnamon rolls, and ice cream) and soda fountain dispensers were associated with illness by univariate analysis. Using multivariate analysis, only DF1 and the carbonated beverage dispensers

remained strongly associated with illness (Table 1).

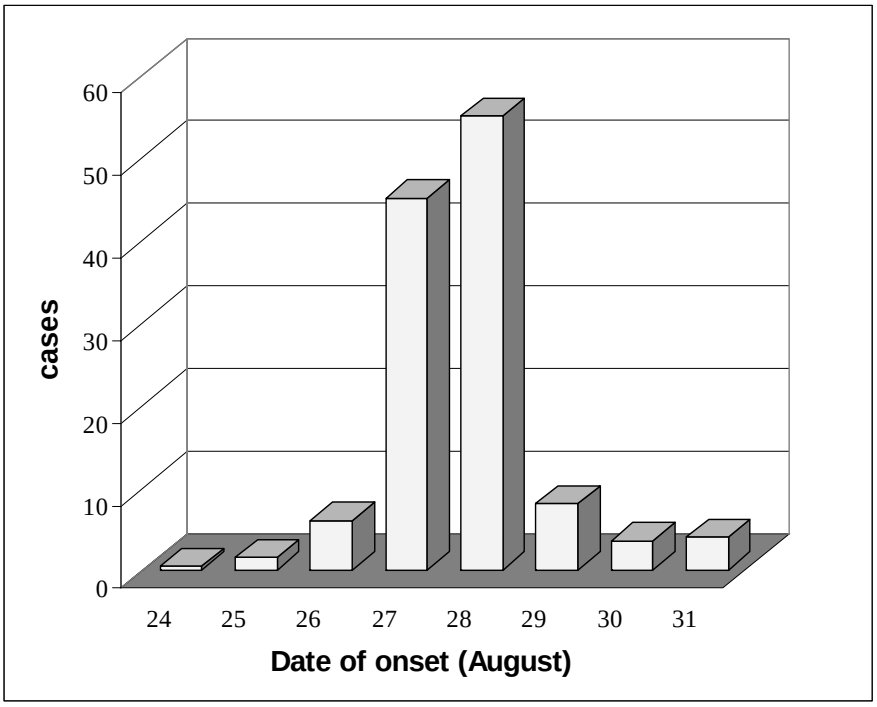

Figure 1. Number of cases of gastroenteritis among U.S. Army trainees, by date of onset — Texas, August, 1998

TABLE 1. Odds ratios for selected exposures in an outbreak of gastroenteritis in U.S. Army trainees — Texas, August 1998

Exposure	Univariate analysis		Multivariate analysis	
	Odds ratio	96% CI*	Odds ratio	95% CI
Ever ate at dining facility 1 (DF1) during the week before illness	9.8	2.8–40.2	7.3	2.0–26.4
Ate preferentially at DF1	3.7	2.0–6.9	2.4	1.3–4.5
Ever ate at dining facility 2 during the week before illness	1.1	0.5–2.3	0.6	0.2–1.4
Drank carbonated beverages	3.8	2.0–7.2	2.6	1.3–5.0
Ate crumb cake	2.4	1.2–4.8	1.8	0.8–3.8
Ate ice cream	1.7	1.1–3.0	1.1	0.6–2.0
Ate cinnamon roll	1.7	0.8–3.7	1.3	0.6–3.0
Ate pie	1.5	0.9–2.7	1.1	0.6–2.0
Used ice	1.5	0.8–2.9	1.1	0.6–2.0

*Confidence interval

1. What was the most likely pathogen in this outbreak?

2. What do you think the vehicle was, and how did it get contaminated?

3. If a food handler was the source of the contamination, which of them was the most likely culprit?

4. Why do reports like this tend to come more often from military establishments than from civilian populations?

PHR 250, 2007
5/23/07, 7p

SECOND EPIDEMIOLOGICAL PROBLEM SET

OUTBREAK OF DIARRHEAL ILLNESS ASSOCIATED WITH DRINKING UNPASTEURIZED APPLE CIDER — NEW YORK, OCTOBER 1996

In October 1996, unpasteurized apple cider or juice was associated with three outbreaks of gastrointestinal illness. In the Western United States, an outbreak of *Escherichia coli* O157:H7 infections associated with unpasteurized commercial apple juice caused illness in 66 persons and one death. In addition, one outbreak of apple cider-related *E. coli* O157:H7 infections and another of cider-related agent Z infections occurred in the Northeast. Apple cider is a traditional beverage produced and consumed in the fall. Cider often is manufactured locally at small cider mills where apples are crushed in presses, and the cider frequently is not pasteurized before sale. This report summarizes the clinical and epidemiologic features of the apple cider-related outbreak in New York, which suggests that current practices for producing apple cider may not be adequate to prevent microbial contamination.

New York

During October 10–15, a local hospital laboratory notified the Cortland County Health Department (CCHD) about 10 cases of laboratory-confirmed agent Z illness with recent onset among county residents (1990 population: 48,963). During the same period in 1995, one case of agent Z illness was reported to CCHD. All case-patients had onset of symptoms during September 28–October 10 and reported drinking apple cider produced at a local cider mill (mill A). CCHD, the New York State Department of Health (NYSDOH), and the New York State Department of Agriculture and Markets (NYS A&M) initiated an investigation of this cluster.

A confirmed case was defined as onset of diarrhea during September 28–October 19 in a Cortland County resident and laboratory evidence of agent Z in a stool specimen. A suspected case was defined as onset of diarrhea during the outbreak period in a household member of a person with confirmed agent Z illness. CCHD conducted active surveillance for additional cases by contacting area clinicians, hospitals, and laboratories.

A total of 20 confirmed and 11 suspected cases were identified from 19 households. The median age was 27 years (range: 1–62 years), and 17 were female. Symptoms included diarrhea (100%), abdominal cramping (55%), vomiting (39%), fever (36%), and bloody diarrhea (10%). The median duration of symptoms was 6 days (range: 1–21 days). CCHD and NYSDOH conducted a matched case-control study to assess probable sources of the outbreak. One neighborhood-matched control-household was contacted for each household with a laboratory-confirmed case. In each control-household, an adult (age $\geq$18 years) member was asked about history of illness, whether anyone in the household had drank apple cider since September 28, which brand of cider was consumed, and the date the cider was purchased. Eighteen case-households were included in the matched case-control study. A history of drinking cider from mill A was reported for at least one member of the 18 households, compared with only one of the

18 control-households (matched OR = undefined, $p<0.01$). Specifically, cider pressed during September 28–29 (i.e., opening weekend) was associated with illness: 15 of 17 case-households in which the purchase date was known compared with none of the control-households reported drinking cider pressed on opening weekend (matched OR = undefined, $p<0.01$). Mill A purchased all apples for cider pressing from one New York orchard. Local and state health departments and NYS A&M inspected the cider mill and apple orchard. The owner of the orchard reported that only picked apples were sold to the cider mill, and drop apples were sold for use in processed or pasteurized foods. Before pressing, the mill washed and brushed the apples using water from a 45-foot drilled well; preservatives were not added to the cider. Although dairy livestock were not maintained by the orchard, the cider mill was located across the road from a dairy farm. Testing of remaining cider samples from opening weekend, swabs of equipment surfaces, and water obtained on October 21 from the drilled well did not yield agent Z. However, coliform bacteria were detected in four water samples obtained from the well, and *E. coli* was detected in one sample.

1. What is “agent Z”?

2. How did it contaminate the apples or cider?

3. Was the water supply appropriate, and was it involved in the contamination?

4. What might be done to prevent recurrences of this outbreak?

FOODBORNE OUTBREAKS OF DIARRHEA — RHODE ISLAND AND NEW HAMPSHIRE, 1993

Rhode Island

On March 25, the Rhode Island Department of Health was notified of gastrointestinal illness among passengers on an airline flight from Charlotte, North Carolina, to Providence, Rhode Island, on March 21. The flight carried 98 passengers; 47 (64%) of 74 passengers who were interviewed met the case definition of three or more loose stools in 24 hours beginning within 4 days after the flight. Additional symptoms included abdominal cramps (94%), nausea (70%), headache (57%), fever (13%), and vomiting (13%). The only common meal for all ill passengers was dinner served on board the flight. The median incubation period was 41 hours (range: 12-77 hours); two (5%) of 44 persons recovered within 48 hours of onset of illness.

Illness was most strongly associated with eating garden salad made from shredded carrots and iceberg, romaine, and endive lettuce (46 [98%] of 47 ill passengers compared with 6 [22%] of 27 well passengers; relative risk [RR] = 4.4; 95% confidence interval [CI] = 2.2-8.9). Investigators from the Food and Drug Administration (FDA) contacted 18 passengers who had traveled on March 21 on a different flight operated by the airline and who had been served the same meal; 9 passengers reported gastrointestinal illness. On March 21, approximately 4000 portions of salad had been prepared by one catering service for 40 flights operated by the same airline that day. The FDA traceback determined that all of the salad ingredients were of U.S. origin.

Stool specimens obtained from 20 passengers from the index flight were negative on culture for *Salmonella*, *Shigella*, *Campylobacter*, *Yersinia*, and *Vibrio*, and viral particles were not observed in 12 stool specimens examined by electron microscopy at CDC. Samples from 10 ill passengers yielded a common pathogen.

FDA inspection of the caterer's facilities did not identify deficiencies in sanitary conditions. In addition, all food handlers denied gastrointestinal illness or recent travel outside the United States. Samples of food collected for culture on March 27 did not yield the pathogen.

New Hampshire

On April 5, the New Hampshire Division of Public Health Services was notified of gastrointestinal illness in eight persons who ate a buffet dinner served at a mountain lodge on March 31. A total of 202 persons ate the dinner, including 132 guests and 70 lodge employees. A case was defined as diarrhea (three or more loose or watery stools in a 24-hour period) and one other symptom (cramps, fever, headache, nausea, or vomiting) with onset from April 1 through April 7 in a guest or employee who had eaten the dinner. Of the 123 guests and 56 employees

who were interviewed, 96 (78%) and 25 (45%), respectively, had illness that met the case definition. Additional symptoms included cramps (92%), nausea (59%), myalgias (50%), headache (49%), fever (22%), and vomiting (11%). Illness began a median of 38 hours after foods from the buffet were eaten (range: 3-159 hours); 60 (65%) of 93 persons for whom information was available reported continuing illness 4-6 days after symptom onset.

Illness among guests was most strongly associated with consumption of tabouleh salad (cases occurred in 78 {94%} of 83 guests who ate the tabouleh and 18 {53%} of 34 guests who did not [RR = 1.8; 95% C I= 1.3-2.5]). Tabouleh was the only food associated with illness among lodge employees (RR = 6.4; 95% CI = 2.2-18.8). The tabouleh was prepared from onions, carrots, zucchini, peppers, broccoli, mushrooms, green onions, tomatoes, parsley, bulgur wheat, olive oil, lemon juice, and bottled garlic. All of the produce was of U.S. origin. The salad was prepared the evening before the banquet. All food preparers denied gastrointestinal illness or travel outside the United States the week before the banquet.

Cultures of stool specimens obtained from 14 persons were negative for *Salmonella*, *Shigella*, *Campylobacter*, and *Yersinia*; neither ova nor parasites were detected in stool specimens from seven ill persons. However, the same pathogen as that found in the Rhode Island outbreak was isolated from stool specimens from seven of nine ill guests and from one of five well employees. Additional serotypes also were isolated from six specimens.

Follow-up Investigation

Plasmid profiles of the pathogen strains from the outbreaks in New Hampshire and Rhode Island were identical but differed from those of 10 other serotype strains from other sources.

MMWR 43(05);81,87-88, February 11, 1994

1. What is the most likely pathogen in these outbreaks?

2. What was the common food in the two outbreaks?

3. What is the probable source of contamination?

4. Why did the FDA do the investigation in Rhode Island, but not in New Hampshire?

FOOD POISONING AT A BANQUET

An outbreak of food poisoning occurred in Spokane, Washington, following a convention banquet at a large hotel on May 4. Of the 1,000 persons who ate the banquet meal, all 1,000 were questioned and 144 reported being ill. The signs and symptoms were:

Diarrhea	(91%)	Headache	(39%)	Vomiting	(10%)
Cramps	(73%)	Nausea	(37%)	Fever	(7%)

One person was hospitalized; he had bloody diarrhea, along with other signs and symptoms.

Table 1. Food poisoning outbreak, Spokane, Washington.
Hours from banquet to onset of symptoms for 85 cases.

Hours	Number of cases	Hours	Number of cases
1	0	16	5
2	0	17	3
3	2	18	3
4	1	19	4
5	1	20	3
6	0	21	3
7	0	22	2
8	1	23	2
9	0	24	2
10	0	25	0
11	7	26	0
12	13	27	0
13	11	28	1
14	12	29	0
15	8	30	1

Table 2. Food histories, Spokane, Washington, outbreak

Food	Ate			Did not eat		
	Ill	Well	Total	Ill	Well	Total
Crab cocktail	102	478	580	11	193	204
Green salad	92	446	538	21	225	246
Baked potato	103	478	581	10	193	203
Prime rib	116	640	756	0	28	28
Green beans	102	479	581	11	192	203
Bread rolls	86	424	510	27	247	274
Chocolate eclair	90	429	519	23	242	265
Milk	34	123	157	79	548	627
Coffee	87	423	510	26	248	274

All foods were prepared in the same place, but served in three shifts: 7–8 p.m., 8–9 p.m., and 9–10 p.m., in three dining areas. People from Spokane City, who ate in dining room A, had higher attack rates than people from other places.

Crabs were boiled and cracked, and the meat was placed in the refrigerator immediately. Cocktails were prepared within 30 minutes after removal of the meat from the refrigerator and placed back in the refrigerator until served.

The green salad was lettuce with vinegar and oil dressing.

The baked potatoes were served hot from the oven.

Thirty roasts were cooked and sliced. Slices served in room A were not placed in a warmer until served, as were the slices for the other dining rooms.

The green beans were commercially frozen; these were boiled and served hot.

The bread rolls were obtained from the largest city bakery.

The chocolate eclairs were purchased from the same bakery as the bread.

The milk was fresh pasteurized.

The coffee was served hot.

One month after the outbreak, roasts obtained from three slaughterhouses supplying meat regularly to the hotel were found positive for a foodborne pathogen at low levels (<100 cells/g).

1. Calculate the median incubation period.

2. Find the most suspect food from Table 2.

3. What was the most likely cause (disease agent) of the outbreak?

4. How did the food become harmful?

5. Why did the people from Spokane have a higher attack rate?

6. How significant is the finding of the same agent in roasts 1 month later?

7. How could the outbreak have been prevented?

8. What laboratory and epidemiological criteria would confirm your explanation of this outbreak?

PHR 250
Problem #8

ILLNESS FROM A PICNIC

On Sunday, June 10, three families left together in the morning on a picnic in a forested area near a lake. Lunch was served at noon and consisted of: barbecued chicken, vegetable salad, bread, ice cream, coffee, and milk. Supper was served at 6 p.m. and consisted of: shrimp casserole, fresh fruit, milk, and coffee.

That evening, 6 of the 10 people who spent the day together and shared the meals became ill (see Table 1). All recovered within 2 days.

Interviews revealed that the barbecued chicken had been bought ready made at a supermarket Sunday at 9 a.m.; it was kept unrefrigerated in the trunk of a car and served at noon without further preparation. The shrimp casserole was prepared Saturday, June 9, from frozen, already peeled shrimp and fresh vegetables. After cooking, the shrimp casserole was placed in the refrigerator overnight and transported to the picnic ground in an ice chest the following day: it was heated almost to boiling immediately before being served at the Sunday supper. The salad was prepared Sunday morning and kept refrigerated. The ice cream, fresh fruit, and milk were bought at the supermarket Sunday morning. The coffee was freshly made and served hot.

1. What were the most common symptoms?

2. Fill in the attack rate for each food in Table 2.

3. Which food is most suspect?

4. What was the probable causative agent?

5. How did the outbreak most likely occur?

6. Are males more susceptible to the agent than females?

7. What was the median incubation time?

8. What laboratory and epidemiological criteria would confirm your explanation of this outbreak?

Table 1. Data on persons at the picnic

Person	Sex	Age	Lunch	Dinner	Symptoms	Onset
1	M	35	chicken salad bread coffee	shrimp coffee	nausea cramps vomiting diarrhea	8:30 p.m.
2	F	32	chicken salad coffee	fruit milk coffee		
3	M	10	chicken salad bread ice cream	fruit milk coffee		
4	M	8	chicken milk ice cream	shrimp milk	nausea cramps diarrhea	9:30 p.m.
5	M	30	chicken salad ice cream coffee	shrimp fruit coffee	nausea cramps vomiting diarrhea	9 p.m.
6	F	30	salad milk coffee	milk fruit		
7	F	5	ice cream milk	milk	nausea vomiting	midnight
8	M	40	chicken salad bread milk	shrimp fruit milk	nausea cramps vomiting diarrhea	10 p.m.
9	F	35	salad milk coffee	shrimp milk fruit		
10	F	15	salad milk	shrimp milk fruit	nausea cramps vomiting diarrhea	9:30 p.m.

Table 2. Attack rate worksheet

Food	Persons eating food				Persons not eating food			
	Ill	Well	Total	Attack rate	Ill	Well	Total	Attack rate
Chicken								
Salad								
Bread								
Coffee								
Ice cream								
Milk								
Shrimp								
Fruit								