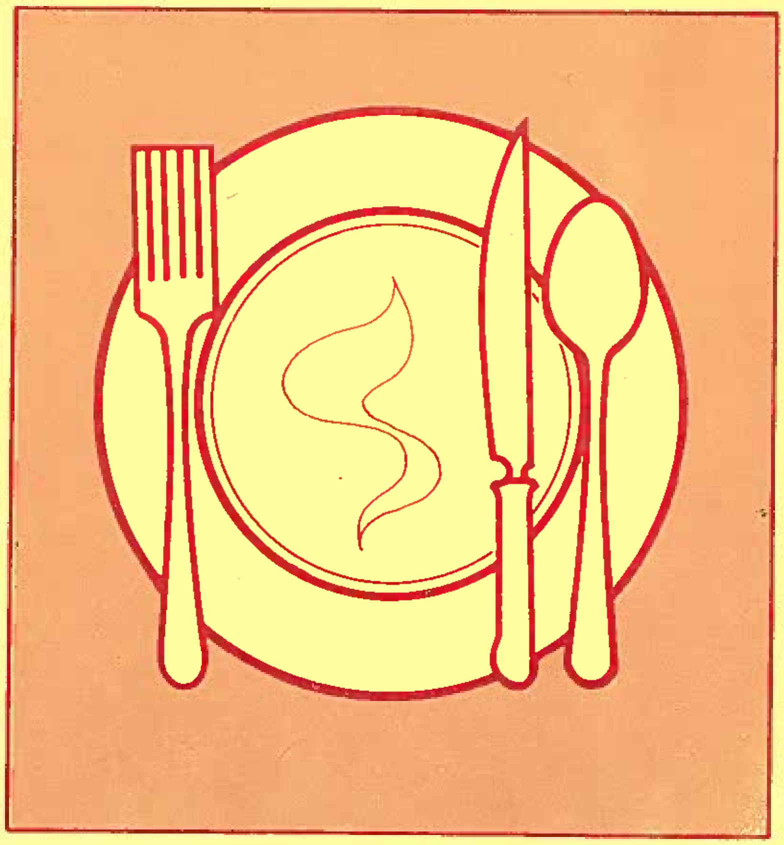
ANNUAL SUMMARY 1981
ISSUED JUNE 1983

CENTERS FOR DISEASE CONTROL

FOODBORNE DISEASE SURVEILLANCE



U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES • Public Health Service

PREFACE

This report summarizes information received from state and local health departments, the Food and Drug Administration, the U.S. Department of Agriculture, and private physicians. The information is preliminary and is intended primarily for use by those with responsibility for disease control activities. Anyone desiring to quote this report should contact the Enteric Diseases Branch for confirmation and further interpretation.

Contributions to the report are most welcome. Please address them to:

Enteric Diseases Branch
Division of Bacterial Diseases
Center for Infectious Diseases
Centers for Disease Control
Atlanta, Georgia 30333

SUGGESTED CITATION

Centers for Disease Control: Foodborne Disease Outbreaks Annual Summary 1981 Issued June 1983

00-4194

TABLE OF CONTENTS

HHS Publication No. (CDC) 84-8185

I. SUMMARY

In 1981 there were 568 outbreaks (14,432 cases) of foodborne disease reported to the Centers for Disease Control. The etiology was confirmed in 44% of the outbreaks. Bacterial pathogens accounted for 185 outbreaks (8,208 cases). The most frequently isolated bacterial pathogen was Salmonella (66 outbreaks), followed by Staphylococcus aureus (44 outbreaks), and Clostridium perfringens (28 outbreaks). Chemical agents were responsible for 51 outbreaks (327 cases), with ciguatera the most common chemical etiology. Food was eaten in a restaurant in 43% of outbreaks, and the most common contributing factor was improper storage (61%).

II. INTRODUCTION

A. History

The reporting of foodborne and waterborne diseases in the United States began over half a century ago when state and territorial health officers, concerned about the high morbidity and mortality caused by typhoid fever and infantile diarrhea, recommended that cases of enteric fever be investigated and reported. The purpose was to obtain information about the role of food, milk, and water in outbreaks of intestinal illness as the basis for sound public health action. Beginning in 1923, the U.S. Public Health Service published summaries of outbreaks of gastrointestinal illness attributed to milk. In 1938, it added summaries of outbreaks caused by all foods. These early surveillance efforts led to the enactment of important public health measures which had a profound influence in decreasing the incidence of enteric diseases, particularly those transmitted by milk and water.

From 1951 through 1960, the National Office of Vital Statistics reviewed reports of outbreaks of foodborne illness and published summaries of them annually in Public Health Reports. In 1961 the Centers for Disease Control (CDC), then the Communicable Disease Center, assumed responsibility for publishing reports on foodborne illness. For the period 1961-65, CDC discontinued publication of annual reviews, but reported pertinent statistics and detailed individual investigations in the Morbidity and Mortality Weekly Report (MMWR).

In 1966 the present system of surveillance of foodborne and waterborne diseases began with the incorporation of all reports of enteric disease outbreaks attributed to microbial or chemical contamination of food or water into an annual summary. Since 1966 the quality of investigative reports has improved primarily as a result of more active participation by state and federal agencies in the investigation of foodborne and waterborne disease outbreaks. Due to increasing interest and activity in waterborne disease surveillance, foodborne and waterborne disease outbreaks have been reported in separate annual summaries since 1978. This report summarizes data from foodborne disease outbreaks reported to CDC for 1981.

B. Objectives

Foodborne disease surveillance has traditionally served 3 objectives:

1. Disease Prevention and Control: Early identification and removal of contaminated products from the commercial market, correction of faulty food preparation practices in food service establishments and in the home, and identification and appropriate treatment of human carriers of foodborne pathogens are the fundamental prevention and control measures resulting from surveillance of foodborne disease.

2. Knowledge of Disease Causation: The responsible pathogen was not identified in over half of the foodborne disease outbreaks reported to CDC in each of the last 5 years. In many of these outbreaks pathogens known to cause foodborne illness may not have been identified because of late or incomplete laboratory investigation. In others, the responsible pathogen may have escaped detection even when a thorough laboratory investigation was carried out because the pathogen may not have been appreciated as a cause of foodborne disease or because the pathogen could not be identified by available laboratory techniques. When more thorough clinical, epidemiologic, and laboratory investigations are employed, perhaps many of these pathogens can be identified, and suitable measures for prevention and control can be instituted.

3. Administrative Guidance: The collection of data from outbreak investigations permits assessment of trends in etiologic agents and food vehicles and focuses on common errors in food handling. By compiling the data in an annual summary, it is hoped that local and state health departments and others involved in the implementation of food protection programs will be kept informed of the factors involved in foodborne disease outbreaks. Comprehensive surveillance would result in a clearer appreciation of priorities in food protection, institution of better training programs, and more effective utilization of available resources.

III. FOODBORNE DISEASE OUTBREAKS

A. Definition of Outbreak

For the purpose of this report, a foodborne disease outbreak is defined as an incident in which (1) 2 or more persons experience a similar illness, usually gastrointestinal, after ingestion of a common food, and (2) epidemiologic analysis implicates the food as the source of the illness. There are a few exceptions; 1 case of botulism or chemical poisoning constitutes an outbreak.

Outbreaks of known etiology are those in which laboratory evidence of a specific etiologic agent is obtained, and specified criteria are met (see Section F). Outbreaks of unknown etiology are those in which epidemiologic evidence implicates a food source, but adequate laboratory confirmation is not obtained. These outbreaks are subdivided into 4 subgroups by incubation period of the illnesses: less than 1 hour (probable chemical poisoning), 1 to 7 hours (probable *Staphylococcus* food poisoning), 8 to 14 hours (probable *C. perfringens* food poisoning), and greater than 14 hours (other infectious or toxic agents).

B. Source of Data

Outbreaks are reported to CDC on a standard reporting form (Section G). Reports come most frequently from state and local health departments; reports may also be received from federal agencies such as the Food and Drug Administration (FDA), U.S. Department of Agriculture (USDA), the U.S. Armed Forces, and occasionally from private physicians. Forms are reviewed at CDC to determine if a specific etiology for the outbreak can be confirmed and, in some instances, questions about an etiologic agent may be referred back to the reporting agency. Data are otherwise accepted as reported on the forms.

C. Interpretation of Data

The limitations on the quantity and quality of data presented here must be appreciated in order to avoid misinterpretation. The number of outbreaks of foodborne disease reported by this surveillance system clearly represents only a small fraction of the total number that occur. The likelihood of an outbreak coming to the attention of health authorities varies considerably depending on consumer and physician awareness, interest, and motivation to report the incident; for example, large outbreaks, interstate outbreaks, restaurant-associated outbreaks, and outbreaks involving serious illness, hospitalizations, or deaths are more likely to come to the attention of health authorities than cases of mild illness following a family cookout.

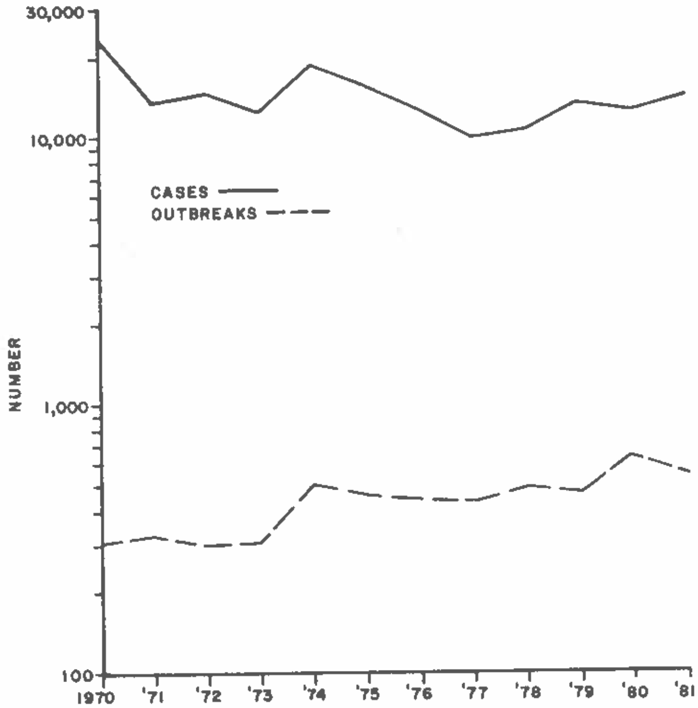
The quality of the data presented here depends upon the commitment to foodborne surveillance by the state or local health departments. The department's interest in foodborne disease and its investigative and laboratory capabilities are central determinants of the quality of the investigation. Furthermore, the likelihood that the findings of the investigation will be reported varies from one locality to another. This report then should not be the basis of firm conclusions about the absolute incidence of foodborne disease, and it should not be used to draw conclusions about the relative incidence of foodborne diseases of various etiologies. For example, foodborne diseases characterized by short incubation periods, such as those of chemical etiology or outbreaks caused by staphylococcal enterotoxin, are more likely to be recognized as common-source foodborne disease outbreaks than those diseases with longer incubation periods, such as hepatitis A, wherein the common-source nature of the cases may be hard to ascertain. Outbreaks involving Bacillus cereus, Escherichia coli, Vibrio parahaemolyticus, Yersinia enterocolitica, or Campylobacter jejuni are less likely to be confirmed because these organisms are often not considered in clinical, epidemiologic, and laboratory investigations. Pathogens which generally cause mild illness will be under-represented, while those causing serious illness, such as Clostridium botulinum, are more likely to be identified. Similarly, restaurant- or commercial product-associated outbreaks have a higher likelihood of being reported.

D. Analysis of Data

In 1981 there were 568 reports of outbreaks (14,432 cases). These reports were received from 48 states, as well as from the U.S. Virgin Islands, Guam, and Puerto Rico (Figure 1). New York reported the largest number of outbreaks, 217, with 156 of these from New York City; California reported the next largest number of outbreaks (44), followed by Washington (38). The total number of outbreaks and cases since 1970 is shown in Figure 2.

Fig.1 OUTBREAKS OF FOODBORNE DISEASE REPORTED TO THE CENTERS FOR DISEASE CONTROL, BY STATE,1981

Fig. 2 NUMBER OF CASES AND OUTBREAKS OF FOODBORNE DISEASE REPORTED TO THE CENTERS FOR DISEASE CONTROL, 1970—1981



In 250 (44%) outbreaks (8,658 cases) an etiology was confirmed (Table 1). Bacterial pathogens accounted for 74% of confirmed outbreaks and 93% of cases. As in the past, Salmonella was the pathogen most frequently responsible for outbreaks followed by S. aureus and C. perfringens.

Chemical etiologies accounted for 20% of the total confirmed outbreaks, but only 4% of the cases. In 1981, ciguatera and mushroom poisoning were the most common chemical etiologies; there were 15 outbreaks of ciguatera and 11 outbreaks of mushroom poisoning. Trichnella spiralis was the most common parasitic foodborne pathogen reported in 1981, accounting for 7 outbreaks and 70 cases; hepatitis A virus was the only viral foodborne pathogen reported, accounting for 6 outbreaks and 128 cases. The breakdown of outbreaks by etiologic category for the period 1977-1981 is shown in Table 2.

In 1981, 39 deaths associated with 20 outbreaks of foodborne diseases were reported; the agents associated with fatalities were Salmonella (21 patients in 8 outbreaks), ciguatera (3 patients in 3 outbreaks), mushroom poisoning (3 patients in 3 outbreaks), C. perfringens (2 patients in 1 outbreak) and C. botulinum (1 patient), S. aureus (1 patient), and T. spiralis (1 patient). In 7 fatal cases (2 outbreaks) the etiologic agent was not determined. Fourteen of the 21 patients who died with salmonellosis were residents of nursing homes; the 2 patients who died with C. perfringens infection and the 1 patient who died with S. aureus infection were also nursing home residents.

No pathogen was identified in 318 (56%) outbreaks (5,638 cases) reported in 1981. The thoroughness of the investigation in these outbreaks varied from those when only minimal laboratory work was performed to others in which extensive investigation failed to reveal a pathogen. Incubation periods, however, were reported for illnesses in 289 of these outbreaks. In 12 outbreaks the incubation period was reported as <1 hour; in 137 outbreaks the incubation period ranged from 1 to 7 hours; in 69 outbreaks the incubation period was 8 to 14 hours; and in 71 outbreaks the incubation period was $\geq$15 hours.

Many different vehicles were implicated in the 1981 outbreaks (Table 3). The most common vehicle was beef, accounting for 34 outbreaks; the most common pathogens associated with beef were Salmonella (13 outbreaks) and C. perfringens (6 outbreaks). Outbreaks involving ham were most often associated with S. aureus (8 of 9 outbreaks), and outbreaks involving milk were most commonly associated with Campylobacter (5 of 9 outbreaks). Most outbreaks associated with fish were due to ciguatera; ciguatera outbreaks were most commonly associated with barracuda (6 outbreaks), grouper (4 outbreaks), or red snapper (4 outbreaks). No vehicle was identified in 56 (22%) of the 250 outbreaks of known etiology; 24 of these outbreaks involved Salmonella (36% of all Salmonella outbreaks). As might be expected, in 268 (84%) of the 318 outbreaks of unknown etiology, no vehicle of transmission was identified.

Two hundred fifty-one outbreaks were associated with a restaurant, cafeteria, or delicatessen, and 131 outbreaks were associated with foods eaten at home (Table 4). Salmonella, S. aureus, Campylobacter, and C. botulinum outbreaks, however, occurred more commonly in homes than in other places. In previous years, outbreaks of foodborne illness occurred most frequently in the spring and fall. In 1981, no overall seasonal trends were seen, although Salmonella-associated outbreaks occurred most frequently in the summer and fall, and S. aureus outbreaks most frequently in the summer (Table 5). In 295 outbreaks, the reporting agency specified a factor or factors which they felt contributed to the outbreak (Table 6). The most common factor in bacterial outbreaks was improper storage, which was cited in 181 (61%) of 295 outbreaks.

E. Comments

There are limitations in the quantity and quality of the data presented in this report. The variability in reporting can be seen by looking at the distribution of outbreaks by state. A few states, such as New York, Washington, and California, reported a disproportionately large number of outbreaks. For example, New York State and New York City reported 217 outbreaks, over one-third of those reported for the entire United States. While it is possible that states such as New York, Washington, and California have an increased rate of foodborne disease, it is more likely that these differences simply represent differences in reporting. The same variability in reporting can be seen when looking at outbreaks by pathogen. Our data show that C. botulinum is as common a foodborne pathogen as Shigella and B. cereus, a conclusion that can only be explained by more complete reporting for botulism than for some other pathogens.

The number of outbreaks of foodborne disease of confirmed etiology reported to CDC over the last 5 years has increased. The distribution of cases by etiology has also remained fairly constant. Etiologies typically have been confirmed in about 40% of outbreaks. When the etiologies have been confirmed, bacterial pathogens have consistently accounted for approximately two-thirds of outbreaks, with chemical etiologies accounting for an additional 20%-25%.

An unusually large number of deaths due to foodborne disease occurred in 1981; this cannot be explained by the small increase in reported cases. Over half of these deaths were related to salmonellosis (21/39), and about half (17/39) occurred in nursing home patients. These data suggest that elderly patients and patients with underlying chronic illnesses are at an increased risk of death due to foodborne disease.

Many factors contribute to foodborne disease. In 1981, the 5 most common factors, in order of frequency of occurrence, included: 1) improper holding temperature, 2) poor personal hygiene, 3) contaminated equipment, 4) inadequate cooking, and 5) food from an unsafe source. In most of the outbreaks with a bacterial etiology, the food had been stored at improper holding temperatures. In outbreaks of botulism or trichinosis, the food had usually been inadequately cooked. In outbreaks of ciguatera and mushroom poisoning, the food itself was unsafe, and illness was not in any sense related to improper handling or preparation.

The 1 outbreak classified as "Other Bacterial" was attributed to Citrobacter freundii, an organism whose role as a pathogen in the gastrointestinal tract is unclear. In this outbreak involving 48 cases, the predominant symptoms were diarrhea, cramps, nausea, and less frequently, headache and myalgia; the incubation period was about 40 hours. The implicated vehicle was chicken served at a restaurant banquet. Cultures of the chicken were positive for C. freundii with 10^7 organisms per gram. Three of 3 patients cultured had stool cultures positive for C. freundii, and no recognized bacterial enteric pathogen was isolated. Two of 2 well individuals who attended the banquet and 0 of 2 well individuals who were members of the same organization but did not attend the banquet had stool cultures positive for C. freundii. Because data on asymptomatic carriage of C. freundii are unavailable, it was difficult to interpret these findings. Other bacterial or viral agents could have caused this outbreak. Additional studies of the virulence of C. freundii and its possible role as a foodborne pathogen are needed.

The large number of outbreaks in which no pathogen was identified serves as a challenge to improve investigative skills so that known pathogens can be identified more frequently and new and as yet unidentified pathogens may be recognized.

Table 1 Confirmed Foodborne Disease Outbreaks, Cases, and Deaths, by Etiology, United States, 1981

Etiology	Outbreaks No.	Outbreaks (%)	Cases No.	Cases (%)	Deaths No.	Deaths (%)
BACTERIAL						
Bacillus cereus	8	(3.2)	74	(0.9)	0	(0.0)
Campylobacter jejuni	10	(4.0)	487	(5.6)	0	(0.0)
Clostridium botulinum	11	(4.4)	22	(0.3)	1	(3.1)
Clostridium perfringens	28	(11.2)	1,162	(13.4)	2	(6.3)
Salmonella	66	(26.4)	2,456	(26.8)	21	(65.6)
Shigella	9	(3.6)	351	(4.1)	0	(0.0)
Staphylococcus aureus	44	(17.6)	2,934	(33.9)	1	(3.1)
Streptococcus Group A	2	(0.8)	307	(3.5)	0	(0.0)
Streptococcus Group D	1	(0.4)	24	(0.3)	0	(0.0)
Vibrio cholerae non-01	1	(0.4)	4	(<0.1)	0	(0.0)
Vibrio parahaemolyticus	2	(0.8)	13	(0.2)	0	(0.0)
Yersinia enterocolitica	2	(0.8)	326	(3.8)	0	(0.0)
Other	1	(0.4)	48	(0.6)	0	(0.0)
Total	185	(74.0)	8,208	(93.2)	25	(78.1)
CHEMICAL						
Ciguatoxin	15	(6.0)	152	(1.8)	3	(9.4)
Heavy metals	2	(0.8)	4	(<0.1)	0	(0.0)
Monosodium glutamate	2	(0.8)	4	(<0.1)	0	(0.0)
Mushroom poisoning	11	(4.4)	25	(0.3)	3	(9.4)
Scombrotoxin	7	(2.8)	67	(0.8)	0	(0.0)
Other	14	(5.6)	75	(0.9)	0	(0.0)
Total	51	(20.4)	327	(3.8)	6	(18.8)
PARASITIC						
Giardia lamblia	1	(0.4)	61	(0.7)	0	(0.0)
Trichinella spiralis	7	(2.8)	70	(0.8)	1	(3.1)
Total	8	(3.2)	131	(1.5)	1	(3.1)
VIRAL						
Hepatitis A	6	(2.4)	128	(1.5)	0	(0.0)
Total	6	(2.4)	128	(1.5)	0	(0.0)
CONFIRMED TOTAL	250	(100.0)	8,794	(100.0)	32	(100.0)

Table 2 Confirmed Foodborne Disease Outbreaks, by Etiology, United States, 1977-1981

Etiology	1977 No.	1977 (%)	1978 No.	1978 (%)	1979 No.	1979 (%)	1980 No.	1980 (%)	1981 No.	1981 (%)
BACTERIAL										
A. hinshawii	1	(0.6)	–	–	–	–	–	–	–	–
B. cereus	–	–	6	(3.9)	–	–	9	(4.1)	8	(3.2)
Brucella	–	–	–	–	2	(1.3)	–	–	–	–
C. jejuni	–	–	–	–	–	–	5	(2.3)	10	(4.0)
C. botulinum	20	(12.7)	12	(7.8)	7	(4.0)	14	(6.3)	11	(4.4)
C. perfringens	6	(3.8)	9	(5.9)	20	(11.6)	25	(11.3)	28	(11.2)
E. cloacae	–	–	–	–	1	(0.6)	–	–	–	–
E. coli	–	–	1	(0.7)	–	–	1	(0.5)	–	–
Salmonella	41	(26.1)	45	(29.3)	44	(25.5)	39	(17.7)	66	(26.4)
Shigella	5	(3.2)	4	(2.6)	7	(4.0)	11	(5.0)	9	(3.6)
S. aureus	25	(15.9)	23	(14.9)	34	(19.7)	27	(12.2)	44	(17.6)
Streptococcus Group A	–	–	–	–	–	–	–	–	2	(0.8)
Streptococcus Group D	–	–	1	(0.7)	–	–	–	–	1	(0.4)
Streptococcus Group G	–	–	–	–	1	(0.6)	–	–	–	–
V. cholerae 01	–	–	1	(0.7)	–	–	–	–	–	–
V. cholerae non-01	1	(0.6)	–	–	1	(0.6)	–	–	1	(0.4)
V. parahaemolyticus	2	(1.3)	2	(1.3)	2	(1.3)	4	(1.8)	2	(0.8)
Y. enterocolitica	–	–	–	–	–	–	–	–	2	(0.8)
Other	–	–	1	(0.7)	–	–	1	(0.5)	1	(0.4)
Total	101	(64.2)	105	(68.5)	119	(69.2)	136	(61.7)	185	(74.0)
CHEMICAL										
Ciguatoxin	3	(1.9)	19	(12.3)	18	(10.4)	15	(6.8)	15	(6.0)
Heavy metals	8	(5.1)	1	(0.7)	1	(0.6)	1	(0.5)	2	(0.8)
Monosodium glutamate	2	(1.3)	–	–	–	–	–	–	2	(0.8)
Mushroom poisoning	5	(3.2)	1	(0.6)	1	(0.6)	–	–	11	(4.4)
Paralytic shellfish	–	–	4	(2.6)	–	–	5	(2.2)	–	–
Scombrotoxin	13	(8.3)	7	(4.5)	12	(7.0)	29	(13.0)	7	(2.8)
Other	6	(3.8)	5	(3.2)	4	(2.3)	16	(7.2)	14	(5.6)
Total	37	(23.6)	37	(23.9)	36	(20.9)	66	(29.7)	51	(20.4)
PARASITIC										
Anisakidae	1	(0.6)	–	–	–	–	–	–	–	–
Trichinella spiralis	14	(8.9)	7	(4.5)	11	(6.4)	5	(2.3)	7	(2.8)
Other	–	–	–	–	–	–	2	(0.9)	1	(0.4)
Total	15	(9.5)	7	(4.5)	11	(6.4)	7	(3.2)	8	(3.2)
VIRAL										
Hepatitis A	4	(2.5)	5	(3.2)	5	(2.9)	10	(4.5)	6	(2.4)
Other	–	–	–	–	1	(0.6)	2	(0.9)	–	–
Total	4	(2.5)	5	(3.2)	6	(3.5)	12	(5.4)	6	(2.4)
CONFIRMED TOTAL	157	(100.0)	154	(100.0)	172	(100.0)	221	(100.0)	250	(100.0)

Table 3 Foodborne Outbreaks by Specific Etiology and Vehicle of Transmission, United States, 1981

Etiology	Beef	Venison	Ham	Pork	Sausage	Chicken	Turkey	Other Meat	Shell Fish	Tuna	Other Fish	Milk	Cheese	Egg Nog
BACTERIAL														
B. cereus	-	-	-	-	-	-	-	-	1	-	-	-	-	-
C. jejuni	-	-	-	-	-	-	-	-	-	-	-	5	-	-
C. botulinum	-	-	-	-	-	-	-	1	-	-	2	-	-	-
C. perfringens	6	-	-	2	1	4	4	1	-	-	-	-	-	-
Salmonella	13	-	-	1	-	4	5	-	2	-	-	3	1	1
Shigella	-	-	-	-	-	1	-	-	-	-	-	-	-	-
S. aureus	3	1	8	2	1	3	4	-	-	-	1	1	1	-
Streptococcus Group A	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Streptococcus Group D	-	-	-	-	-	-	-	-	-	-	-	-	-	-
V. cholerae non-01	-	-	-	-	-	-	-	-	1	-	-	-	-	-
V. parahaemolyticus	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Y. enterocolitica	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Other	-	-	-	-	-	1	-	-	-	-	-	-	-	-
Total	22	1	8	5	2	13	13	2	4	-	3	9	2	1
CHEMICAL														
Ciguatoxin	-	-	-	-	-	-	-	-	-	1	14	-	-	-
Heavy metals	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Monosodium glutamate	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Mushroom poisoning	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Scombrotoxin	-	-	-	-	-	-	-	-	-	4	3	-	-	-
Other	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Total	-	-	-	-	-	-	-	-	-	5	17	-	-	-
PARASITIC														
Giardia lamblia	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Trichinella spiralis	1	-	-	2	3	-	-	-	-	-	-	-	-	-
Total	1	-	-	2	3	-	-	-	-	-	-	-	-	-
VIRAL														
Hepatitis A	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Total	-	-	-	-	-	-	-	-	-	-	-	-	-	-
CONFIRMED TOTAL	23	1	8	7	5	13	13	2	4	5	20	9	2	1
UNKNOWN	11	-	1	-	1	5	3	-	5	2	1	-	1	-
TOTAL 1981	34	1	9	7	6	18	16	2	9	7	21	9	3	1

Table 3 Foodborne Disease Outbreaks by Specific Etiology and Vehicle of Transmission, United States, 1981 (Cont'd)

Ice Cream	Baked Foods	Fruits & Vegetable	Potato Salad	Poultry, Fish, Egg Salad	Other Salad	Fried Rice	Chinese Food	Mexican Food	Carbonated Bev	Non-Dairy Bev	Multiple Foods	Mushrooms	Other Foods	Unknown	Total
-	-	-	-	-	-	4	-	-	-	-	-	-	1	2	8
-	-	-	-	-	-	-	-	-	-	-	-	-	-	5	10
-	-	5	-	-	-	-	-	-	-	-	1	-	1	1	11
-	-	-	-	-	1	-	-	2	-	-	1	-	-	6	28
1	1	-	-	-	2	-	-	2	-	-	6	-	-	24	66
-	-	-	2	-	1	-	-	-	-	-	-	-	-	5	9
1	7	1	-	2	2	-	-	-	-	-	1	-	-	5	44
-	-	-	-	-	-	-	-	-	-	-	-	-	-	2	2
-	-	1	-	-	-	-	-	-	-	-	-	-	-	-	1
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
-	-	-	-	-	-	-	-	-	-	-	-	-	-	2	2
-	-	1	-	-	-	-	-	-	-	-	1	-	-	-	2
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
2	8	8	2	2	6	4	-	4	-	-	10	-	2	52	185
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	15
-	-	-	-	-	-	-	-	-	2	-	-	-	-	-	2
-	-	-	-	-	-	1	1	-	-	-	-	-	-	-	2
-	-	-	-	-	-	-	-	-	-	-	-	11	-	-	11
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	7
2	4	1	-	-	1	-	-	-	-	1	-	-	5	-	14
2	4	1	-	-	1	1	-	-	2	1	-	11	5	-	51
-	-	-	-	-	1	-	-	-	-	-	-	-	-	-	1
-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	7
-	-	-	-	-	1	-	-	-	-	-	-	-	-	1	8
-	-	-	-	-	-	-	-	-	-	-	3	-	-	3	6
-	-	-	-	-	-	-	-	-	-	-	3	-	-	3	6
4	12	9	2	2	8	5	1	4	2	1	13	11	7	56	250
1	-	2	2	-	7	2	-	1	1	-	3	-	1	268	318
5	12	11	4	2	15	7	1	5	3	1	16	11	8	324	568

Table 4 Foodborne Disease Outbreaks, by Specific Etiology and Place Where Food Was Eaten, United States, 1981

Etiology	Home	Delicatessen, Cafeteria, or Restaurant	School	Picnic	Church	Camp	Other	Un-known	Total
BACTERIAL									
B. cereus	2	5	-	-	-	-	1	-	8
C. jejuni	5	1	2	-	-	1	1	-	10
C. botulinum	10	-	-	-	-	-	1	-	11
C. perfringens	4	11	3	1	1	-	8	-	28
Salmonella	18	15	7	2	2	2	19	1	66
Shigella	1	3	2	-	1	-	2	-	9
S. aureus	17	6	9	3	2	-	7	-	44
Streptococcus Group A	-	1	-	-	-	-	1	-	2
Streptococcus Group D	-	-	-	-	-	-	1	-	1
V. cholerae non-01	-	-	-	-	-	-	1	-	1
V. parahaemolyticus	-	1	-	-	-	-	1	-	2
Y. enterocolitica	1	-	-	-	-	1	-	-	2
Other	-	1	-	-	-	-	-	-	1
Total	58	44	23	6	6	4	43	1	185
CHEMICAL									
Ciguatoxin	3	2	-	-	-	-	3	7	15
Heavy metals	-	1	-	-	-	-	1	-	2
Monosodium glutamate	-	2	-	-	-	-	-	-	2
Mushroom poisoning	8	-	-	-	-	-	3	-	11
Scombrotoxin	3	2	-	-	-	1	1	-	7
Other	4	3	2	-	1	-	3	1	14
Total	18	10	2	-	1	1	11	8	51
PARASITIC									
Giardia lamblia	-	-	-	-	-	-	1	-	1
Trichinella spiralis	6	1	-	-	-	-	-	-	7
Total	6	1	-	-	-	-	1	-	8
VIRAL									
Hepatitis A	-	3	-	-	-	-	2	1	6
Total	-	3	-	-	-	-	2	1	6
CONFIRMED TOTAL	82	58	25	6	7	5	57	10	250
UNKNOWN	49	193	16	-	7	2	50	1	318
TOTAL 1981	131	251	41	6	14	7	107	11	568

Table 5 Foodborne Disease Outbreaks by Specific Etiology and Month of Occurrence, United States, 1981

Etiology	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec	Unknown	Total
BACTERIAL														
B. cereus	-	-	-	-	5	-	2	-	1	-	-	-	-	8
C. jejuni	1	-	1	1	2	-	-	-	1	3	1	-	-	10
C. botulinum	2	-	1	-	1	1	-	2	3	-	1	-	-	11
C. perfringens	2	-	4	-	5	4	1	1	5	1	2	3	-	28
Salmonella	4	1	2	4	6	7	8	8	7	8	7	4	-	66
Shigella	-	1	-	-	-	1	-	-	3	3	1	-	-	9
S. aureus	1	2	-	-	5	1	6	12	-	4	5	8	-	44
Streptococcus Group A	-	-	-	-	1	-	1	-	-	-	-	-	-	2
Streptococcus Group D	-	-	-	-	-	-	-	-	1	-	-	-	-	1
V. cholerae non-01	-	-	-	-	-	-	-	-	-	-	1	-	-	1
V. parahaemolyticus	-	2	-	-	-	-	-	-	-	-	-	-	-	2
Y. enterocolitica	-	-	-	-	-	-	1	-	-	-	-	1	-	2
Other	-	-	-	-	-	-	-	1	-	-	-	-	-	1
Total	10	6	8	5	25	14	19	24	21	19	18	16	-	185
CHEMICAL														
Ciguatoxin	-	3	2	1	1	4	-	1	-	1	2	-	-	15
Heavy metals	1	-	-	-	1	-	-	-	-	-	-	-	-	2
Monosodium glutamate	-	-	1	-	-	-	-	-	1	-	-	-	-	2
Mushroom poisoning	-	-	-	-	-	-	1	-	5	-	1	4	-	11
Scombrotoxin	-	-	1	-	-	-	4	1	1	-	-	-	-	7
Other	1	2	-	-	-	2	2	2	1	-	1	2	1	14
Total	2	5	4	1	2	6	7	4	8	1	4	6	1	51
PARASITIC														
Giardia lamblia	-	-	-	-	-	-	1	-	-	-	-	-	-	1
Trichinella spiralis	-	1	1	-	1	1	-	-	-	-	2	1	-	7
Total	-	1	1	-	1	1	1	-	-	-	2	1	-	8
VIRAL														
Hepatitis A	-	1	1	-	-	1	1	-	-	1	1	-	-	6
Total	-	1	1	-	-	1	1	-	-	1	1	-	-	6
CONFIRMED TOTAL	12	13	14	6	28	22	28	28	29	21	25	23	1	250
UNKNOWN	32	26	34	29	30	20	23	23	19	35	20	26	1	318
TOTAL 1981	44	39	48	35	58	42	51	51	48	56	45	49	2	568

Table 6 Foodborne Disease Outbreaks by Etiology and Contributing Factors, United States, 1981

Etiology	Number of Reported Outbreaks	Number of Outbreaks In Which Factors Reported	Improper Holding Temperatures	Inadequate Cooking	Contaminated Equipment	Food From Unsafe Source	Poor Personal Hygiene	Other
BACTERIAL								
B. cereus	8	8	8	2	1	-	2	2
C. jejuni	10	6	1	2	1	3	1	2
C. botulinum	11	-	-	-	-	-	-	-
C. perfringens	28	21	21	6	2	1	1	-
Salmonella	66	43	17	19	12	5	15	11
Shigella	9	6	2	-	1	-	6	-
S. aureus	44	30	28	1	4	4	14	4
Streptococcus Group A	2	2	1	-	-	-	1	-
Streptococcus Group D	1	-	-	-	-	-	-	-
V. cholerae non-01	1	-	-	-	-	-	-	-
V. parahaemolyticus	2	1	-	1	-	1	-	-
Y. enterocolitica	2	2	-	-	-	-	2	-
Other	1	1	1	1	-	-	-	-
Total	185	120	79	32	21	14	42	19
CHEMICAL								
Ciguatoxin	15	8	-	-	-	8	-	-
Heavy metals	2	2	-	-	-	2	-	-
Monosodium glutamate	2	2	-	-	-	-	-	2
Mushroom poisoning	11	7	-	-	-	7	-	-
Scombrotoxin	7	-	-	-	-	-	-	-
Other	14	10	-	-	1	3	-	7
Total	51	29	-	-	1	20	-	9
PARASITIC								
Giardia lamblia	1	-	-	-	-	-	-	-
Trichinella spiralis	7	7	-	7	1	2	-	-
Total	8	7	-	7	1	2	-	-
VIRAL								
Hepatitis A	6	4	-	-	1	-	4	-
Total	6	4	-	-	1	-	4	-
CONFIRMED TOTAL	250	160	79	39	24	36	46	28
UNKNOWN	318	135	102	23	46	4	64	6
TOTAL 1981	568	295	181	62	70	40	110	34

F. Guidelines for Confirmation of Foodborne Disease Outbreak

	Clinical Syndrome	Laboratory, clinical, and/or epidemiologic criteria for confirmation
BACTERIAL		
1. Bacillus cereus	Vomiting toxin: a) incubation period 1-6 hrs. b) vomiting, some cases with diarrhea Diarrheal toxin: a) incubation period 6-24 hrs. b) diarrhea, abdominal cramps, some cases with vomiting	a) isolation of $\geq 10^5$ organisms per gram in epidemiologically incriminated food OR b) isolation of organism from stools of ill persons and not in stools of controls
2. Brucella	a) incubation period several days to several months b) clinical syndrome compatible with brucellosis	a) 4-fold increase in titer OR b) positive blood culture
3. Campylobacter jejuni	a) incubation period 2-10 days, usually 4-7 b) gastrointestinal syndrome--abdominal pain, often severe; bloody diarrhea common	Isolation of organisms from stool/blood of ill individuals
4. Clostridium botulinum	a) incubation 2 hours-8 days, usually 12-48 hours b) clinical syndrome compatible with botulism (see CDC Botulism Manual)	a) detection of botulinal toxin in human sera, feces, or food OR b) isolation of C. botulinum organism from stools OR c) clinical syndrome in persons known to have consumed same food as other individuals with laboratory-proven cases
5. Clostridium perfringens	a) incubation period 9-15 hrs. b) lower intestinal syndrome--majority of cases with diarrhea but little vomiting or fever	a) organisms of same serotype in epidemiologically incriminated food and stool of ill individuals. OR b) isolation of organisms with same serotype in stool of most ill individuals and not in stool of controls OR c) $\geq 10^5$ organisms per gram in epidemiologically incriminated food provided specimen properly handled
6. Escherichia coli	a) incubation period 6-36 hrs. b) gastrointestinal syndrome--majority of cases with diarrhea	a) demonstration of organisms of same serotype in epidemiologically incriminated food and stool of ill individuals and not in stool of controls OR b) isolation from stool of most ill individuals, organisms of the same serotype which have been shown to be enterotoxigenic or invasive by laboratory techniques

		Clinical Syndrome	Laboratory, clinical, and/or epidemiologic criteria for confirmation
7.	Salmonella	a) incubation period 6-48 hrs. b) gastrointestinal syndrome--majority of cases with diarrhea	a) isolation of Salmonella organism from epidemiologically implicated food OR b) isolation of Salmonella organism from stools of ill individuals
8.	Shigella	a) incubation period 12-50 hours b) gastrointestinal syndrome--majority of cases with diarrhea	a) isolation of Shigella organism from epidemiologically implicated food OR b) isolation of Shigella organism from stools of ill individuals
9.	Staphylococcus aureus	a) incubation period 30 min.-8 hours (usually 2-4 hrs.) b) gastrointestinal syndrome--majority of cases with vomiting	a) detection of enterotoxin in epidemiologically implicated food OR b) organisms with same phage type in stools or vomitus of ill individuals; isolation from epidemiologically implicated food and/or skin or nose of food handler is supportive evidence OR c) isolation of $\geq 10^5$ organisms per gram in epidemiologically implicated food
10.	Streptococcus Group A	a) incubation period 1-4 days b) febrile URI snydrome	a) isolation of organisms with same M and T type from implicated food OR b) isolation of organisms with same M and T type from throats of ill individuals
11.	Vibrio cholerae 01	a) incubation period 1-5 days b) gastrointestinal syndrome--majority of cases with diarrhea and without fever	a) isolation of toxigenic V. cholerae 01 from epidemiologically incriminated food OR b) isolation of organisms from stools or vomitus of ill individuals OR c) significant rise in vibriocidal, bacterial agglutinating or antitoxin antibodies in acute and early convalescent sera, or significant fall in vibriocidal antibodies in early and late convalescent sera in persons not recently immunized
	Vibrio cholerae Non-01	a) incubation period up to 3 days b) gastrointestinal syndrome--majority of cases with diarrhea	a) isolation of non-01 V. cholerae of same serotype from stools of ill persons; isolation from epidemiologically implicated food is supportive evidence
12.	Vibrio parahaemolyticus	a) incubation period 4-30 hrs. b) gastrointestinal syndrome--majority of cases with diarrhea	a) isolation of $\geq 10^5$ organisms from epidemiologically implicated food (usually seafood) OR

	Clinical Syndrome	Laboratory, clinical, and/or epidemiologic criteria for confirmation
		b) isolation of Kanagawa-positive organisms from stool of ill individuals
13. Others	clinical data appraised in individual circumstances	laboratory data appraised in individual circumstances
CHEMICAL		
1. Heavy metals Antimony Cadmium Copper Iron Tin	a) incubation period 5 min. to 8 hrs. (usually less than 1 hr) b) clinical syndrome compatible with heavy metal poisoning--usually gastrointestinal syndrome and often metallic taste	demonstration of high concentration of metallic ion in epidemiologically incriminated food or beverage
2. Ichthyosarcotoxin		
Ciguatoxin	a) incubation period 1-48 hrs. (usually 2-8 hrs.) b) usually gastrointestinal symptoms followed by neurologic manifestations, including paresthesia of lips, tongue, throat or extremities, and reversal of hot and cold sensation	a) demonstration of ciguatoxin in epidemiologically incriminated fish OR b) clinical syndrome in person(s) who have eaten a type of fish previously associated with ciguatera fish poisoning (e.g., snapper, grouper)
Puffer fish (tetrodotoxin)	a) incubation period 10 min. to 3 hrs. (usually 10-45 min.) b) paresthesia of lips, tongue, face or extremities often followed by numbness, loss of proprioception or a "floating" sensation	a) demonstration of tetrodotoxin in fish OR b) puffer fish epidemiologically incriminated
Scombrotoxin	a) incubation period 1 min. to 3 hours (usually less than 1 hour) b) flushing, headache, dizziness, burning of mouth and throat, upper and lower gastrointestinal symptoms, urticaria and generalized pruritus	a) demonstration of elevated histamine levels in epidemiologically incriminated fish OR b) clinical syndrome in person(s) known to have eaten a fish of order Scombrodei or type of fish previously associated with scombroid poisoning (e.g., mahi-mahi)
3. Monosodium glutamate	a) incubation period 3 min. to 2 hours (usually less than 1 hour) b) burning sensations in chest, neck, abdomen or extremities, sensations of lightness and pressure over face, or a heavy feeling in the chest	history of large amounts (usually $\geq$1.5 grams) of MSG having been added to epidemiologically incriminated food
4. Mushroom poison Group containing ibotenic acid and muscimol	a) incubation period 1-12 hrs. (usually less than 4 hrs.)	a) demonstration of toxic chemical in epidemiologically incriminated mushrooms OR

	Clinical Syndrome	Laboratory, clinical, and/or epidemiologic criteria for confirmation
	b) clinical syndrome compatible w/mushroom poisoning by this group--often including confusion, delirium, visual disturbances	b) epidemiologically incriminated mushrooms identified as a toxic type
Group containing amanitotoxins and phallotoxins, or gyromitrin	a) incubation period 5-18 hrs. b) characteristic clinical syndrome compatible with mushroom poisoning by this group--upper and lower gastrointestinal symptoms followed by hepatic and/or renal failure	a) demonstration of toxic chemical in epidemiologically incriminated mushrooms OR b) epidemiologically incriminated mushrooms identified as a toxic type
Groups containing muscarine, psilocybin and psilocin, gastrointestinal irritants, disulfiram-like compounds	a) characteristic incubation period b) clinical syndrome compatible with mushroom poisoning by these groups	a) demonstration of toxic chemical in epidemiologically incriminated mushrooms OR b) epidemiologically incriminated mushroom identified as toxic type
5. Paralytic or neurotoxic shellfish poison	a) incubation period 30 min. to 3 hours b) paresthesias of lips, mouth or face, and extremities; weakness, including respiratory difficulty in most severe cases; upper and lower gastrointestinal symptoms in some cases	a) detection of toxin in epidemiologically incriminated mollusks OR b) detection of large numbers of shellfish poisoning-associated species of dinoflagellates in water from which epidemiologically incriminated mollusks gathered
6. Other chemical	clinical data appraised in individual circumstances	laboratory data appraised in individual circumstances
PARASITIC AND VIRAL		
1. *Trichinella spiralis*	a) incubation period 3-30 days b) clinical syndrome compatible with trichinosis--often including fever, high eosinophil count, orbital edema, myalgia	a) muscle biopsy from ill individual OR b) serological tests OR c) demonstration of larvae in incriminated food
2. Hepatitis A	a) incubation period 10-45 days b) clinical syndrome compatible with hepatitis--usually including jaundice, GI symptoms, dark urine	liver function tests compatible with hepatitis in affected persons who consumed the epidemiologically incriminated food
3. Others	clinical evidence appraised in individual circumstances	laboratory evidence appraised in individual circumstances

This report is authorized by law (Public Health Service Act, 42 USC 241). While your response is voluntary, your cooperation is necessary for the understanding and control of the disease.

FORM APPROVED
OMB NO. 0920–000

G. INVESTIGATION OF A FOODBORNE OUTBREAK

1. Where did the outbreak occur?

State ________ (1,2) City or Town ________ County ________

2. Date of outbreak: (Date of onset 1st case)

________ (3-8)

3. Indicate actual (a) or estimated (e) numbers:

Persons exposed ________ (9-11)

Persons ill ________ (12-14)

Hospitalized ________ (15-16)

Fatal cases ________ (17)

4. History of Exposed Persons:

No. histories obtained ________ (18-20)

No. persons with symptoms ________ (21-23)

Nausea ________ (24-26) Diarrhea ________ (33-35)

Vomiting ________ (27-29) Fever ________ (36-38)

Cramps ________ (30-32) Other, specify ________

________ (39)

5. Incubation period (hours):

Shortest ____ (40-42) Longest ____ (43-45)

Approx. for majority ________ (46-48)

6. Duration of illness (hours):

Shortest ____ (49-51) Longest ____ (52-54)

Approx. for majority ________ (55-57)

7. **Food-specific attack rates: (58)**

Food Items Served	Number of persons who ATE specified food				Number who did NOT eat specified food			
	Ill	Not Ill	Total	Percent Ill	Ill	Not Ill	Total	Percent Ill

8. Vehicle responsible (food item incriminated by epidemiological evidence): (59,60) ________

9. Manner in which incriminated food was marketed: (Check all applicable)

(a) Food Industry (61)
- Raw ☐ 1
- Processed ☐ 2

Home Produced
- Raw ☐ 3
- Processed ☐ 4

(b) Vending Machine ☐ 1 (62)

(c) Not wrapped ☐ 1 (63)
- Ordinary Wrapping ☐ 2
- Canned ☐ 3
- Canned–Vacuum Sealed ☐ 4
- Other (specify) ☐ 5

____

(d) Room Temperature ☐ 1 (64)
- Refrigerated ☐ 2
- Frozen ☐ 3
- Heated ☐ 4

If a commercial product, indicate brand name and lot number

____

10. Place of Preparation of Contaminated Item: (65)
- Restaurant ☐ 1
- Delicatessen ☐ 2
- Cafeteria ☐ 3
- Private Home ☐ 4
- Caterer ☐ 5
- Institution:
  - School ☐ 6
  - Church ☐ 7
  - Camp ☐ 8
- Other, specify ☐ 9

____

11. Place where eaten: (66)
- Restaurant ☐ 1
- Delicatessen ☐ 2
- Cafeteria ☐ 3
- Private Home ☐ 4
- Picnic ☐ 5
- Institution:
  - School ☐ 6
  - Church ☐ 7
  - Camp ☐ 8
- Other, specify ☐ 9

____

DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE
CENTERS FOR DISEASE CONTROL
ATLANTA, GEORGIA 30333

CDC 4.245
1-74

(Over)

LABORATORY FINDINGS (Include Negative Results)

12. Food specimens examined: (67)

Specify by "X" whether food examined was original (eaten at time of outbreak) or check-up (prepared in similar manner but not involved in outbreak)

Item	Orig.	Check up	Findings Qualitative	Findings Quantitative
Example: beef	X		C. perfringens, Hobbs type 10	$2X10^6$/gm

13. Environmental specimens examined: (68)

Item	Findings
Example: meat grinder	C. perfringens, Hobbs Type 10

14. Specimens from patients examined (stool, vomitus, etc.): (69)

Item	No. Persons	Findings
Example: stool	11	C. perfringens, Hobbs Type 10

15. Specimens from food handlers (stool, lesions, etc.): (70)

Item	Findings
Example: lesion	C. perfringens, Hobbs type 10

16. Factors contributing to outbreak (check all applicable):

	Yes	No	
1. Improper storage or holding temperature	☐ 1	☐ 2	(71)
2. Inadequate cooking	☐ 1	☐ 2	(72)
3. Contaminated equipment or working surfaces	☐ 1	☐ 2	(73)
4. Food obtained from unsafe source	☐ 1	☐ 2	(74)
5. Poor personal hygiene of food handler	☐ 1	☐ 2	(75)
6. Other, specify ______	☐ 1	☐ 2	(76)

17. Etiology: (77, 78)

Pathogen ______

Chemical ______

Other ______

Suspected ☐ 1 (79)

Confirmed ☐ 2

Unknown ☐ 3

18. Remarks: Briefly describe aspects of the investigation not covered above, such as unusual age or sex distribution; unusual circumstances leading to contamination of food, water, epidemic curve, etc. (Attach additional page if necessary)

Name of reporting agency: (80)

Investigating official: | **Date of investigation:**

NOTE: Epidemic and Laboratory Assistance for the investigation of a foodborne outbreak is available upon request by the State Health Department to the Center for Disease Control, Atlanta, Georgia 30333.

To improve national surveillance, please send a copy of this report to: Enteric Diseases Branch
Bacterial Diseases Division
Center for Infectious Diseases
Centers for Disease Control
Atlanta, Georgia 30333

Submitted copies should include as much information as possible, but the completion of every item is not required.

CDC 4.245 (BACK)
1-74

H. LINE LISTING OF FOODBORNE DISEASE OUTBREAKS, 1981

Etiology	State	Number of Cases	Date of Onset	Lab Data: Patient	Lab Data: Vehicle	Lab Data: Food-Handler	Vehicle	Location Where Food Mishandled and Eaten
BACTERIAL								
Bacillus cereus								
	Connecticut	2	5/15	+	+		Fried rice	Restaurant
	Connecticut	14	9/23		+		Unknown	Restaurant
	Georgia	20	5/13				Unknown	Restaurant
	Massachusetts	9	7/21	+	+		Fried rice	Private home
	New York	14	5/10		+		Other food	Restaurant
	New York	9	5/28	+	+		Fried rice	Other
	Virginia	4	5/16		+		Shellfish	Private home
	Washington	2	7/20		+		Fried rice	Restaurant
Campylobacter jejuni								
	Arizona	190	4/24	+			Milk	Private home
	Arizona	14	10/18	+			Milk	Camp
	Kansas	103	3/01	+			Milk	Private home
	Maine	3	10/07	+			Unknown	Private home
	Maine	14	10/20	+			Unknown	Private home
	Minnesota	25	5/14	+			Milk	Other
	New York	19	9/12	+		+	Unknown	School
	New York	10	11/10	+			Unknown	School
	Oregon	106	1/?	+			Milk	Private home
	New York City	3	5/30	+			Unknown	Restaurant
Citrobacter freundii								
	Arizona	48	8/09	+	+		Chicken	Restaurant
Clostridium botulinum								
	Alaska	9	9/2	+			Other meat	Private home
	California	1	3/22	+	+		Fruit or vegetables	Private home
	California	2	9/24		+		Other fish	Private home
	California	2	11/02	+	+		Fruit or vegetables	Private home
	Colorado	1	5/26	+	+		Other food	Private home
	Hawaii	1	8/21	+			Unknown	Private home
	Idaho	1	6/27	+	+		Other fish	Private home
	Montana	1	1/?	+			Multiple vehicles	Other
	Ohio	1	9/17	+			Fruit or vegetables	Private home
	Oregon	2	8/20	+	+		Fruit or vegetables	Private home
	Utah	1	1/7	+	+		Fruit or vegetables	Private home
Clostridium perfringens								
	California	23	1/13		+		Chicken	Private home
	California	19	3/16		+		Pork	Other
	California	19	8/21		+		Chicken	Other
	California	20	9/28		+		Beef	Restaurant
	Colorado	95	12/?	+	+		Beef	Other
	Connecticut	40	6/19	+	+		Beef	Picnic
	Connecticut	24	9/16	+			Unknown	Other
	Connecticut	35	9/30	+			Turkey	School
	Florida	35	5/28	+	+		Turkey	Other
	Georgia	130	3/25	+	+		Turkey	School
	Georgia	185	7/27	+	+		Turkey	Other
	Maryland	50	3/23		+		Multiple vehicles	Restaurant
	New Hampshire	48	11/23	+		+	Beef	School
	New Mexico	14	12/16	+	+		Mexican food	Restaurant
	New York	22	5/02		+		Sausage	Private home
	New York	7	5/25		+		Chicken	Private home
	New York	40	6/27		+		Other salad	Church
	North Carolina	90	6/02	+			Pork	Private home
	Pennsylvania	12	3/01	+	+		Beef	Other

Etiology	State	Number of Cases	Date of Onset	Lab Data: Patient	Lab Data: Vehicle	Lab Data: Food-Handler	Vehicle	Location Where Food Mishandled and Eaten
Clostridium perfringens (Cont'd)								
	Pennsylvania	70	11/21	+	+	+	Beef	Restaurant
	South Carolina	15	10/25	+			Unknown	Restaurant
	Washington	2	1/22	+			Unknown	Restaurant
	Washington	24	5/10	+			Chicken	Restaurant
	Washington	9	5/11	+			Unknown	Restaurant
	Washington	2	9/16	+			Unknown	Restaurant
	Washington	3	12/02	+			Unknown	Restaurant
	Wisconsin	120	6/09	+			Other meat	Other
	Wisconsin	9	9/02	+	+		Mexican food	Restaurant
Salmonella								
S. typhi	Arizona	6	4/17	+		+	Chicken	Private home
S. saint-paul	California	2	1/23	+	+		Milk	Private home
S. alachua	California	9	5/?		+		Mexican food	Restaurant
S. enteritidis	California	77	6/?	+		+	Unknown	Restaurant
S. enteritidis	California	24	8/05	+		+	Unknown	Restaurant
Unknown	California	9	8/12	+			Unknown	Camp
S. haardt	California	26	10/18	+	+		Chicken	Other
S. enteritidis	Colorado	15	7/08	+		+	Beef	Restaurant
S. newport	Colorado	28	10/?	+			Multiple vehicles	Restaurant
S. litchfield	Connecticut	3	4/01	+			Unknown	Other
S. typhimurium	Connecticut	15	6/22	+			Multiple vehicles	Private home
S. typhimurium	Connecticut	14	8/02	+		+	Shellfish	Cafeteria
S. heidelberg	Delaware	37	6/22	+	+	+	Beef	Church
Group B	Delaware	37	7/25	+			Beef	Other
Group B	Florida	6	11/10	+		+	Unknown	Restaurant
Unknown	Florida	6	11/?				Unknown	Unknown
S. typhimurium	Georgia	48	5/28	+	+	+	Turkey	School
S. typhimurium	Georgia	22	5/31	+	+		Ice Cream	Church
S. heidelberg	Georgia	7	10/19	+	+		Multiple vehicles	Private home
S. enteritidis	Illinois	11	6/22	+			Unknown	Private home
S. enteritidis	Illinois	4	10/27	+			Unknown	Private home
S. enteritidis	Illinois	3	10/30	+			Unknown	Private home
S. mbandaka	Louisiana	18	5/13			+	Baked foods	Other
S. montevideo	Michigan	16	9/10	+			Other salad	Other
S. typhi	Michigan	18	10/12	+		+	Unknown	Other
Group B	Minnesota	8	5/22	+			Unknown	Private home
S. johannesburg	Minnesota	55	6/20	+			Turkey	School
S. typhimurium	Minnesota	54	7/12	+		+	Other salad	Other
Unknown	Mississippi	60	7/29	+			Chicken	Picnic
S. montevideo	Nebraska	18	8/11	+			Unknown	Other
Group D	New Hampshire	30	1/24	+		+	Unknown	School
S. typhimurium	New Jersey	47	7/29	+	+		Beef	Other
S. hadar	New Jersey	13	11/08	+			Multiple vehicles	Private home
S. enteritidis	New Jersey	74	12/25	+		+	Egg nog	Other
S. typhimurium	New York	321	1/?	+	+		Cheese	Private home
Unknown	New York	7	3/21	+			Milk	Private home
S. typhimurium	New York	86	4/18	+			Unknown	Restaurant
Group B	New York	19	6/07	+			Beef	Restaurant
Group C2	New York	39	8/01	+			Multiple vehicles	Restaurant
S. chester	New York	4	9/11	+			Beef	Private home
S. chester	New York	4	9/25	+	+		Beef	School
Group B	New York	13	12/06	+		+	Unknown	Other
Group C2	New York	55	12/10	+		+	Unknown	School
S. blockley, infantis	North Carolina	120	10/03	+	+		Pork	Other
S. drypool	Ohio	11	9/30			+	Unknown	Other
S. thompson	Pennsylvania	26	5/28	+	+	+	Shellfish	Restaurant
Groups B and C2	Pennsylvania	48	7/20	+			Beef	Other
S. chester, typhimurium	Pennsylvania	37	7/25	+			Beef	Other
S. saint-paul	Pennsylvania	14	8/29	+			Multiple vehicles	Picnic
S. saint-paul	Pennsylvania	3	9/25	+	+		Beef	Private home
S. saint-paul	Pennsylvania	11	9/27	+			Beef	Other
Group D	Pennsylvania	152	11/27	+	+		Turkey	Restaurant
Group C1	South Carolina	2	2/28	+		+	Unknown	Private home
Group C1	South Carolina	31	3/14	+	+		Unknown	Restaurant
S. hadar	South Dakota	19	6/07	+	+		Turkey	Private home

Etiology	State	Number of Cases	Date of Onset	Lab Data			Vehicle	Location Where Food Mishandled and Eaten
				Patient	Vehicle	Food-Handler		
Salmonella (Cont'd)								
S. typhimurium	Tennessee	181	4/01	+	+		Turkey	School
S. typhi	Texas	78	8/18	+		+	Beef	Private home
S. drypool, ohio	Texas	12	11/02	+		+	Unknown	Private home
S. chester, havana, tennessee	Vermont	47	9/01	+	+	+	Beef	Other
S. anatum	Vermont	30	10/21	+	+		Chicken	Other
S. enteritidis	Virginia	12	7/02	+		+	Unknown	Camp
S. saint-paul	Virginia	211	12/09	+		+	Mexican food	School
S. dublin	Washington	17	1/01	+	+		Milk	Private home
S. anatum	Guam	20	11/23	+			Unknown	Other
Unknown	New York City	2	8/08	+		+	Unknown	Restaurant
S. agona	New York City	4	11/08		+		Unknown	Restaurant
Shigella								
S. flexneri	California	7	6/14	+		+	Unknown	Restaurant
Group D	Colorado	20	11/?	+			Potato salad	Other
S. flexneri	Connecticut	23	10/20	+		+	Other salad	Restaurant
S. sonnei	Florida	119	9/14	+			Unknown	Other
S. sonnei	Georgia	76	10/13	+		+	Unknown	School
S. flexneri	Minnesota	32	10/12	+		+	Chicken	Church
S. sonnei	Virginia	14	2/28	+		+	Unknown	Restaurant
S. sonnei	Virginia	42	9/28	+		+	Potato salad	School
Group D	Washington	18	9/28	+			Unknown	Private home
Staphylococcus aureus								
	Alabama	145	2/26		+		Unknown	School
	Alabama	18	8/16		+		Ice Cream	Picnic
	Alabama	16	8/31	+	+		Multiple vehicles	Picnic
	Arizona	116	5/15		+	+	Turkey	School
	Arkansas	8	10/29	+	+		Beef	Restaurant
	California	1000	11/04	+			Milk	School
	California	40	12/23	+	+		P, F, + Egg salad	School
	Connecticut	28	8/24		+	+	Baked foods	Private home
	Connecticut	2	8/30		+	+	Baked foods	Private home
	Connecticut	20	8/30		+	+	Baked foods	Private home
	Florida	182	5/20	+	+	+	Other salad	School
	Florida	22	10/06			+	Unknown	Restaurant
	Florida	101	12/08		+	+	Turkey	Other
	Georgia	42	8/28		+		Pork	Restaurant
	Illinois	4	11/15		+		Ham	Private home
	Indiana	2	5/06		+		Fruit or vegetables	Private home
	Kansas	100	6/09		+		Unknown	Restaurant
	Maine	11	8/02	+	+		Ham	Private home
	Maryland	3	2/01	+	+		Unknown	Private home
	Montana	5	12/20		+		Vennison	Private home
	Nevada	41	7/12	+	+	+	Ham	Other
	New York	16	5/02	+	+		Cheese	School
	New York	12	7/16		+		Baked foods	Private home
	New York	5	7/17		+		Beef	Restaurant
	New York	9	8/30		+		Baked foods	Private home
	New York	22	8/30		+		Baked foods	Private home
	New York	2	11/25		+		Sausage	Private home
	North Carolina	339	7/25	+	+	+	Ham	Other
	Ohio	13	8/09		+	+	Ham	Picnic
	Ohio	2	10/17		+		Pork	Private home
	Ohio	3	12/03	+	+		Other fish	Private home
	Pennsylvania	11	5/10	+	+		Ham	Private home
	Rhode Island	7	10/09		+		Baked foods	School
	South Carolina	30	11/16		+		Beef	Other
	Tennessee	14	7/13	+	+	+	Ham	Restaurant
	Tennessee	120	11/08	+	+	+	Turkey	Church
	Texas	155	12/17		+	+	Turkey	School
	Texas	33	12/18		+		Ham	School
	Vermont	23	8/05		+		Unknown	Church
	Washington	21	12/22	+	+		Chicken	Private home

Etiology	State	Number of Cases	Date of Onset	Lab Data: Patient	Lab Data: Vehicle	Lab Data: Food-Handler	Vehicle	Location Where Food Mishandled and Eaten
Staphylococcus aureus (Cont'd)								
	Wisconsin	22	7/12	+	+	+	Chicken	Other
	New York City	146	1/17		+	+	Other salad	Other
	New York City	12	8/02		+		Chicken	Private home
	New York City	11	12/09				P, F, + Egg salad	Other
Streptococcus								
Group A	Maine	7	7/29	+			Unknown	Restaurant
Group A	Oregon	300	5/11	+		+	Unknown	Other
Group D	California	24	9/11		+		Fruit or vegetables	Other
Vibrios								
V. cholerae non-01	Florida	4	11/09	+			Shellfish	Other
V. parahaemolyticus	Arizona	2	2/19	+			Unknown	Restaurant
V. parahaemolyticus	Rhode Island	11	2/07	+			Unknown	Other
Yersinia enterocolitica								
	New York	239	7/02	+	+	+	Multiple vehicles	Camp
	Washington	87	12/16	+	+		Fruit or vegetables	Private home
CHEMICAL								
Ciguatera								
	Florida	3	2/04				Other fish	Restaurant
	Florida	5	2/11				Other fish	Restaurant
	Louisiana	6	10/01				Tuna	Other
	Washington	2	11/17				Other fish	Other
	Virgin Islands	69	2/25		+		Other fish	Other
	Puerto Rico	15	3/27				Other fish	Unknown
	Puerto Rico	15	3/28		+		Other fish	Private home
	Puerto Rico	16	4/04				Other fish	Unknown
	Puerto Rico	4	5/09				Other fish	Unknown
	Puerto Rico	5	6/04				Other fish	Unknown
	Puerto Rico	4	6/14				Other fish	Unknown
	Puerto Rico	2	6/24				Other fish	Unknown
	Puerto Rico	3	6/30				Other fish	Unknown
	Puerto Rico	1	8/13				Other fish	Private home
	Puerto Rico	2	11/05				Other fish	Private home
Heavy metals								
Copper	Florida	3	1/1		+		Carbonated drink	Restaurant
Copper	Georgia	1	5/28		+		Carbonated drink	Other
Monosodium glutamate								
	Washington	2	3/27				Chinese food	Restaurant
	New York City	2	9/11		+		Fried rice	Restaurant
Mushroom poisoning								
Unknown	California	1	7/12				Mushrooms	Other
Russula emetica	California	6	11/30		+		Mushrooms	Private home
Amanita phalloides	California	2	12/27		+		Mushrooms	Private home
Amanita phalloides	California	2	12/?				Mushrooms	Other
Amanita phalloides	California	2	12/?				Mushrooms	Other
Amanita virosa	New Jersey	2	9/30		+		Mushrooms	Private home
Amanita virosa	New York	3	9/19		+		Mushrooms	Private home
Amanita muscaria	New York	1	9/27		+		Mushrooms	Private home

Etiology	State	Number of Cases	Date of Onset	Lab Data: Patient	Lab Data: Vehicle	Lab Data: Food-Handler	Vehicle	Location Where Food Mishandled and Eaten
Mushroom poisoning (Cont'd)								
Clitocybe illudens	New York	1	9/04		+		Mushrooms	Private home
Unknown	New York	2	9/28		+		Mushrooms	Private home
Galerina Sp	Washington	3	12/17		+		Mushrooms	Private home
Scombrotoxin								
	Arizona	3	9/17				Mahi-mahi	Restaurant
	California	2	3/04		+		Mahi-mahi	Private home
	California	13	8/05		+		Other fish	Other
	Maine	38	7/31				Tuna	Camp
	New York	3	7/29		+		Tuna	Private home
	New York	2	7/29		+		Tuna	Private home
	Virginia	6	7/14		+		Tuna	Restaurant
Other chemical								
Marijuana	California	9	7/10		+		Baked foods	Private home
Jimson weed	California	2	?/?				Other food	Unknown
Cleaning agent	Minnesota	6	1/09		+		Other salad	School
Sodium hydroxide	Minnesota	1	2/25		+		Other food	Restaurant
Alkaloids	Minnesota	1	12/10		+		Fruit or vegetables	Private home
Phenolphthalein	New York	6	7/25		+		Baked foods	Other
Calcium chloride	New York	1	8/31		+		Ice cream	Cafeteria
Xylene, acetone	New York	1	11/30		+		Other food	Private home
Detergent	New York	1	12/02				Ice cream	Private home
Possible LSD	Pennsylvania	10	2/26		+		Baked foods	School
Marijuana	Utah	4	6/19		+		Baked foods	Other
Trisodium phosphate	Washington	2	8/14		+		Other food	Restaurant
Unknown	Washington	1	9/21				Nondairy beverage	Other
Antimony	Wyoming	30	6/12	+	+		Other food	Church
PARASITIC								
Giardia lamblia								
	New Jersey	61	7/16	+			Other salad	Other
Trichinella spiralis								
	Connecticut	9	2/03	+	+		Sausage	Private home
	Connecticut	6	3/28	+			Sausage	Private home
	New York	2	5/02	+			Unknown	Restaurant
	Pennsylvania	8	6/29	+			Beef	Private home
	Rhode Island	24	11/11	+			Pork	Private home
	Rhode Island	13	12/08		+		Pork	Private home
	New York City	8	11/04	+	+		Sausage	Private home
VIRAL								
Hepatitis A								
	Florida	9	7/07				Multiple vehicles	Unknown
	New Jersey	55	6/07	+		+	Unknown	Restaurant
	New York	38	10/21	+		+	Multiple vehicles	Cafeteria
	Virginia	10	3/14	+		+	Multiple vehicles	Other
	Washington	13	2/15	+		+	Unknown	Other
	Washington	3	11/26	+		+	Unknown	Restaurant

UNKNOWN

A line listing of outbreaks of unknown etiology may be obtained by writing to the Enteric Diseases Branch, Bacterial Diseases Division, Center for Infectious Diseases, Centers for Disease Control, Atlanta, Georgia 30333.

I. Selected Foodborne Outbreak Articles Taken From Morbidity and Mortality Weekly Report, 1981

Multistate Outbreak of Salmonellosis Caused by Precooked Roast Beef
MMWR 1981;30:391-392

In the first week of August 1981, 3 outbreaks of salmonellosis that affected more than 100 people in 3 northeastern states were reported to CDC. The first 2 outbreaks were traced to precooked roast beef from a Philadelphia meat processing plant, and the third to delicatessen-style sliced sandwich meat served at a hospital cafeteria in Philadelphia. Some of these meat slices were of the precooked roast beef processed in the Pennsylvania plant.

The first outbreak followed a wedding reception held on July 25 at Claymont, Delaware, attended by approximately 150 people, mostly residents of Delaware County, Pennsylvania. Of the 58 persons contacted for interview, 37 had had diarrhea. *Salmonella* group B was isolated from the stools of 13 patients (11 *S. chester*, 2 *S. typhimurium*). Illness was significantly associated with eating precooked roast beef at the reception ($p<0.001$, Chi-square). None of the meat served at the reception was available for culturing.

The second outbreak followed a wedding reception held on July 25 in southern New Jersey, 47 of 92 persons who attended became ill, and illness was again associated with eating precooked roast beef ($p=0.0025$, Fisher exact test, 2-tailed). *Salmonella* was isolated from 18 of 20 stool cultures (17 *S. typhimurium*, 1 *S. newport*). *S. typhimurium* and *S. johannesburg* were isolated from an opened package of precooked roast beef provided by the caterer of the reception. Another unopened package of the same brand from the same caterer contained *S. typhimurium*, *S. newport*, and *S. anatum*.

The third outbreak, which occurred in a Philadelphia hospital, was first recognized on July 24 after 2 patients had severe diarrhea. Subsequent investigation revealed 42 cases of diarrheal illness between July 20 and August 11. Six of the persons involved were patients, and 36 were hospital employees. *Salmonella* group B was isolated from stools from 18 persons (including 4 patients); *Salmonella* group C_2 was isolated from 1 employee. *Salmonella* group B was isolated from 5 of 71 asymptomatic dietary and nursing staff in a stool-culture survey. Preliminary analysis of a case-control study demonstrated an association between illness and eating sandwich-meat slices served at the hospital cafeteria ($p<0.001$, Mantel-Haenszel for variable number of controls per case). The meat slices included the same brand of precooked roast beef involved in the other outbreaks. Some of the infected persons had not eaten the beef; the other meats may have been contaminated by it. The suspected beef samples were not available for culture, but *Salmonella* group B was recovered from meat drippings in a tray containing remnants of meat from the cafeteria delicatessen.

On August 5, the U.S. Department of Agriculture asked the Philadelphia producer to temporarily halt further distribution of the implicated beef. *S. typhimurium* was isolated from 1 of 64 specimens tested by the USDA. Assessment of the internal temperature of these products by the protein coagulase test showed that the core temperature ranged from 130 F-152 F, $\pm 5°$ (54.4 C-66.7 C $\pm 2.8°$). On August 10, the USDA issued a recall order of all precooked roast beef that had been processed by the Philadelphia company before August 6, 1981.

The precooked roast beef from this company is distributed under 6 brand names (Joy, Lapin, Allied Farms, Big Apple, Twin Brothers, Vincent Giordano) to 77 distributors in Philadelphia, Harrisburg, and Chester, Pennsylvania; Rochester and Brooklyn, New York; and Washington, DC.

Editorial Note: This is the first reported multistate outbreak of salmonellosis attributable to commercially produced precooked roast beef in 4 years. Until 1977, this problem had occurred frequently, particularly in the Northeast (1-4). In 1977, when multiple outbreaks of the disease involving several meat-processing companies were reported from Connecticut, Georgia, New York, New Jersey, Pennsylvania, and Virginia, the USDA instituted regulations requiring that raw beef be cooked until heated throughout to at least 145 F (62.8 C) (5).

The outbreaks reported here may have resulted from failure to achieve the required minimum temperature, as indicated by the USDA study. Also, recent evidence shows that under certain conditions even heating raw meat to 145 F (62.8 C) may not produce a completely Salmonella-free product (6). Further studies on the survival of Salmonella in raw beef may be indicated.

References

1. CDC. Food poisoning due to Salmonella chester--Massachusetts. MMWR 1973;22:302.
2. CDC. Salmonella singapore--New Orleans. MMWR 1975;24:397-8.
3. CDC. Salmonella saint-paul in pre-cooked roasts of beef--New Jersey. MMWR 1976;25:34,39.
4. CDC. Multi-state outbreak of Salmonella newport transmitted by precooked roasts of beef. MMWR 1977;26:277-8.
5. Angelotti R. Minimum cooking requirements for cooked beef roast. Federal Register 1977:42:44217-8.
6. Blankenship LC. Survival of a Salmonella typhimurium experimental contaminant during cooking of beef roasts. Appl Environ Microbiol 1978;35:1160-5.

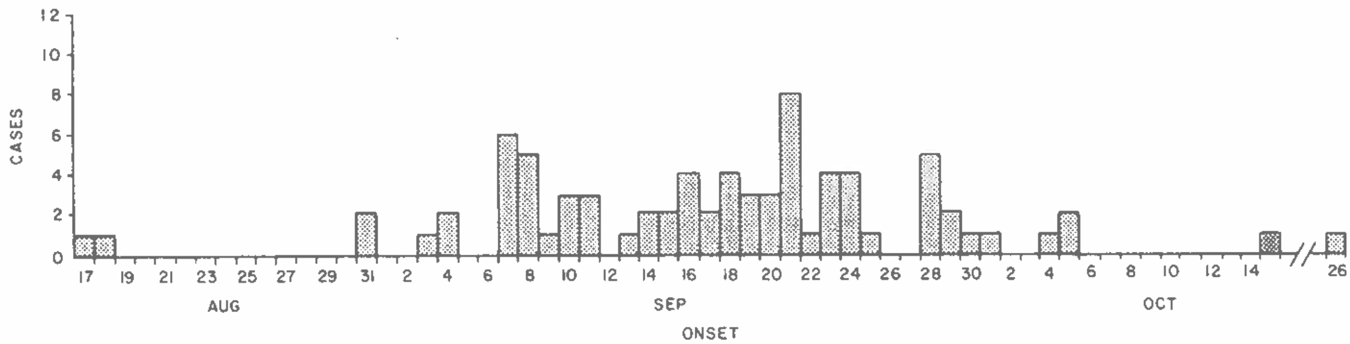
Typhoid Fever -- San Antonio, Texas, 1981

MMWR 1981;30:540,545-6

In the period August 16-October 15, 1981, 72 cases of typhoid fever were reported in the San Antonio Metropolitan Health District. The annual number of such cases in the last 30 years has ranged from 0 to 6 in this city.

The mean age of patients was 19 years (range 1-60); 42 were women and 30 men. Seventy-one were Hispanic and 58 lived on the predominantly Hispanic Westside of San Antonio. An initial investigation revealed no common sources or exposure. All patients had used water only from the municipal system and the city had reported no recent breaks in water lines for the area where most patients lived. Dates of onset ranged over 6 weeks with no single peak (Figure 1). A questionnaire was administered to the first 25 patients in a search for common foods or common-food sources. Analysis revealed 4 potential sources: snowcones from street vendors, food from either of 2 popular Westside fast-food establishments, and food from a specific tortilla molino (mill). A follow-up case-control study was performed. Two controls, matched for age, sex, and neighborhood of residence, were selected for each case. The only variable that demonstrated significance in the case-control study was the consumption of food from the tortilla molino ($p<.00001$). The 2 items purchased most commonly by patients were corn tortillas and barbacoa (a Mexican barbecue made of steamed cow head). The molino was closed to the public on September 27; it was reopened on October 8 after meeting local health department sanitation regulations. Stool specimens from all 31 employees were cultured for Salmonella typhi, and serum specimens were assayed for antibodies to S. typhi. A stool culture from 1 employee yielded S. typhi, and serum specimens from 3 employees (including the culture-positive individual) showed an elevated antibody titer to the Vi antigen of S. typhi. None of the 3 reported a history of typhoid vaccination. Cultures of surface areas and food specimens did not yield S. typhi, although gram-negative bacteria were isolated from barbacoa at all levels of preparation.

Fig. 1 TYPHOID FEVER CASES, BY DATE OF ONSET, SAN ANTONIO, TEXAS, 1981



Barbacoa is salted, unspiced cow head cooked overnight under steam pressure. Meat from the cow head is deboned manually by employees not wearing gloves and is held in a container or a heated grill at 160 F-175 F (71.1 C-79.4 C). It is sold in 1-2 pound portions. Corn tortillas are prepared from corn kernels mixed with a lime slurry (to remove cuticles). The mixture is cooled, washed, and ground into masa (meal), which is shaped manually into tortillas. The tortillas are heated for approximately 2 minutes on rollers warmed by gas jets and are then sorted manually by employees. The employee whose stool culture was positive for S. typhi worked at several locations in the molino including those where barbacoa is deboned and where corn tortillas are shaped and handled. It was not possible without long-term follow-up cultures to determine whether the employee was acutely infected or was a chronic carrier.

All S. typhi isolates in specimens from patients were sensitive to chloramphenicol and ampicillin as assayed by hospital laboratories. CDC identified S. typhi in specimens from patients and from the employee suspected of being the carrier, as phage type degraded Vi approaching B.

Editorial Note: In 1980, about 500 cases of typhoid fever were reported to CDC. The mean number of cases per year over the preceding 5 years was 405 (1). Of the cases reported in detail to CDC between 1977 and 1979, 63% were acquired in foreign countries, and 37% were acquired domestically. During this time period, only 25% of the domestic cases were associated with outbreaks. The last major common-source outbreak in the United States occurred in Dade County, Florida, in 1973, when over 300 of 1,600 exposed workers at a migrant labor camp had typhoid fever (2).

Routine culturing of food handlers is not recommended as previously described (3).

References

1. CDC. Summary--cases of specific notifiable diseases. United States. MMWR 1981;29:632.
2. Hoffman TA, Ruiz CJ, Counts GW, Sachs JM, Nitzkin JL. Waterborne typhoid fever in Dade County, Florida. Clinical and therapeutic evaluation of 105 bacteremic patients. Am J Med 1975;59:481-7.
3. CDC. Health examination of foodhandlers--Europe. MMWR 1981;30:267-73.

Outbreak of Illness due to Clostridium perfringens -- California

MMWR 1981;30:171-2

On January 14, 1981, health department officials were notified of a foodborne outbreak associated with a "Meals on Wheels" program for senior citizens in Victorville, California. Persons who became ill had had dinner delivered to their homes between 11:00 AM and 12:30 PM on January 13. Thirty-nine of the 41 persons who had received the suspected meals were interviewed. Twenty-three (59%) had had symptoms, including diarrhea (91%), abdominal cramps (74%), nausea (35%), vomiting (22%), fever (9%), and headache (4%). The mean incubation period was 8.7 hours, with a range of 1-16 hours. Two patients required hospitalization. All patients recovered within 6 days, with the median duration of illness being 12 hours.

Chicken was implicated as the vehicle of transmission in terms of food-specific attack rates (p=0.037, Fisher exact test, 2-tailed). Food handlers for the catering service reported thinking that the chicken might be spoiled because of its unpleasant odor during heating. An attempt was then made to recall the catering trucks, but 41 of the suspected meals had already been delivered. Further questioning revealed that the chicken had been cooked 6 days earlier, frozen, thawed on January 11, held refrigerated, and reheated on a steam table on January 13. The manner in which the chicken had been prepared and the timing of the onset of symptoms led investigators to suspect *Clostridium perfringens*-associated food poisoning. Laboratory tests of the chicken showed that it contained *C. perfringens* ($\geq 10^5$ organisms/gram).

Editorial Note: Between 1975 and 1979, 57 outbreaks (3,223 cases) of foodborne illness associated with *C. perfringens* were reported to CDC, making *C. perfringens* the third most common bacterial pathogen (after *Salmonella* and *Staphylococcus*) implicated in cases of foodborne disease. The most common vehicles in *C. perfringens*-associated outbreaks were beef and beef products; turkey was the next most common.

C. perfringens is widely distributed in the environment and is frequently isolated from the intestinal tract of animals and humans (1). Some strains produce heat-resistant spores that are not destroyed by cooking but may germinate and multiply if food is held at a warm temperature (optimally in the range of 109-117 F, 42.8-47.2 C) for a period of hours (2). The organism is demanding in its nutritional needs, requiring 13-14 amino acids and 5-6 growth factors for multiplication; these nutritional requirements may account for the frequent association of such outbreaks with high-protein foods.

C. perfringens can be confirmed as the causative agent of an outbreak of foodborne disease by culturing the same serotype from both epidemiologically incriminated food and stool specimens of ill individuals, isolating the same serotype from stool specimens of ill individuals but not of controls, or identifying $\geq 10^5$ organisms per gram from epidemiologically implicated food.

References

1. Collee JG, Knowlden JA, Hobbs BC. Studies on the growth, sporulation and carriage of *Clostridium welchii* with special reference to food poisoning strains. J Appl Bacteriol 1961;24:326-39.
2. Bryan FL. What the sanitarian should know about *Clostridium perfringens* foodborne illness. J Milk Food Technol 1969;32:383-9.

Raw-Milk-Associated Illness -- Oregon, California

MMWR 1981;309:90-92,97

Raw milk has recently been associated with cases of campylobacteriosis in Oregon and salmonellosis in California.

Oregon: In the period December 22, 1980-February 20, 1981, 5 counties in Oregon reported a total of 91 stool isolates of *Campylobacter jejuni* from persons with diarrhea. Of these 91 persons, 52 (57%) gave a history of having consumed raw milk before onset of illness. In the households of these 52 index patients, 76 other household members also drank raw milk; 25 (33%) of these persons developed diarrhea. Of 19 members of these households who did not drink raw milk, none had diarrhea (p=.005).

To test the hypothesis that raw milk was the vehicle of transmission, 2 case-control studies were performed. In the first study, 70 control households in 1 county were selected randomly from a local telephone book. Fifty-seven (81%) of these households were contacted and interviewed. One of these households gave a history of consuming a specific brand of raw milk in the previous 2 months compared with 11 of 15 case households ($p<.0001$). In the control household in which this same brand of raw milk was consumed, the 3 members who drank it became ill but the 3 who did not remained well. No significant association was found between developing *Campylobacter* diarrhea and exposure to sick pets, live poultry, livestock, raw eggs, raw meat, untreated surface water, other individuals with diarrheal illness outside the household, or history of foreign travel in the previous 2 months. In the second case-control study, age-matched controls were selected for 28 cases. In 15 matched pairs, the patient drank the same brand of raw milk as was implicated in the other study, and the control drank no raw milk. In no instance did a patient not drink the implicated brand of raw milk and a control drink it ($p<.001$).

California: On January 23, 1981, the California State Department of Food and Agriculture (DFA) requested that the State Department of Health Services (DHS) order the removal from sale of certified, raw, whole milk from a single California dairy after the DFA Laboratory recovered *Salmonella saint-paul* from bottled milk coded Jan 25C. Independently, the San Diego County Health Department Laboratory recovered the same serotype from an opened milk container of the same code brought in by an ill consumer. That laboratory had also recovered *S. saint-paul* from a 4 1/2-month-old infant who became ill within 2 weeks of starting on this dairy's certified raw milk; the infant had been breast-fed for the previous 4 months. On February 13, the DFA requested that the DHS order the removal from the market of milk coded Feb 17A, 18A, and 19A after both the DFA Laboratory and the Los Angeles County Milk Commission Laboratory--which regulates the dairy--recovered *Salmonella* organisms from milk coded 17A.

The Food and Drug Administration (FDA) cultured the implicated dairy's raw milk, obtained from retail shelves in Nevada, and found *Salmonella* Group B organisms in milk coded Feb 18A.

This dairy's raw milk, which is certified by the American Association of Medical Milk Commissions, was implicated in outbreaks of *S. dublin* in 1958, 1964, and 1971-1975 (3,4). It has also been implicated in sporadic cases of campylobacteriosis (4). Of the 12 different serotypes of *Salmonella* that have been isolated from the dairy's milk (*S. agona*, *S. cerro*, *S. dublin*, *S. infantis*, *S. kentucky*, *S. lille*, *S. meleagridis*, *S. montevideo*, *S. newington*, *S. saint-paul*, *S. typhimurium*, *S. worthington*) only *S. dublin* has been epidemiologically linked to human disease (3).

Editorial Note: An analysis of *S. dublin* cases in the United States in 1979 and 1980 from 17 states (excluding California and Oregon) showed that 11 of 32 patients gave a history of raw milk ingestion. Milk from many different dairies was involved. Unlike tuberculosis and brucellosis, which can be eliminated from dairy herds by adequate precautions, *Salmonella* infections of milking herds continue to occur. Since up to 10% of healthy cattle may carry *S. dublin* (3), *Salmonella* contamination of unpasteurized milk can be a persistant problem, even in dairies that follow the procedures recommended by the American Association of Medical Milk Commissions, a private organization.

The association of *Campylobacter* infections with the use of unpasteurized milk has been documented in the United States and England (4-7). In each outbreak that was investigated, milk was either improperly pasteurized or used in an unpasteurized form. Although up to 60% of healthy cattle excrete *Campylobacter* in their feces, these organisms are eliminated from milk by pasteurization.

Present technology cannot produce raw milk (including that listed as certified) that can be assured to be free of pathogens; only with pasteurization is there this assurance. The U.S. Animal Health Association, the National Association of State Public Health Veterinarians, the Conference of State and Territorial Epidemiologists, the American Academy of Pediatrics, and the House of Delegates of the American Veterinary Medical Association have adopted policy statements that milk for human consumption should be pasteurized.

References

1. CDC. Human Salmonella dublin infection associated with consumption of certified raw milk--California. MMWR 1974;23:175.
2. Werner SB, Humphrey GL, Kamer I. Association between raw milk and human Salmonella dublin infection. Br Med J 1979;2:238-41.
3. Hinton M, Williams BM. Salmonella dublin infection in adult cattle: a slaughterhouse and knackery survey in South West Wales. J Hyg 1977;78:121-7.
4. Taylor PR, Weinstein WM, Bryner JH. Campylobacter fetus infection in human subjects: association with raw milk. Am J Med 1979;66:779-83.
5. Blaser MJ, Cravens J, Powers BW, LaForce FM, Wang W-LL. Campylobacter enteritis associated with unpasteurized milk. Am J Med 1979;67:715-8.
6. Robinson DA, Edgar WJ, Gibson GL, Matcheff AA, Robertson L. Campylobacter enteritis associated with consumption of unpasteurized milk. Br Med J 1979;1:1171-3.
7. Porter IA, Reid TMS. A milk-borne outbreak of Campylobacter infection. J Hyg 1980;84:415-9.

Common-Source Outbreaks of Trichinosis -- New York City, Rhode Island

MMWR 1982;31:161-164

Forty-six cases of trichinosis including 1 death were diagnosed in the period November-December 1981 in 3 common-source outbreaks reported from New York City and Rhode Island. All 3 outbreaks were associated with eating pork from hogs purchased directly from farms and prepared in ethnic dishes calling for raw or partially cooked pork.

New York City: Eight cases of trichinosis were reported to the New York City Department of Health from 2 Brooklyn hospitals on November 16, 1981. The patients were members of 3 related families of Italian heritage. A ninth case was subsequently diagnosed in the course of the investigation. One patient died. The outbreak appeared to be associated with eating dried, homemade pork sausage.

The index patient, a 55-year-old woman, had onset of symptoms on November 4, 1981, 10 days after eating some of the pork sausage. Her initial symptoms included nausea, vomiting, diarrhea, myalgia, and abdominal pain. When she became febrile, she was hospitalized. By the nineteenth day of hospitalization, the patient had developed bronchopneumonia, pulmonary edema, paralytic ileus, and motor paralysis; she died the same day. Autopsy findings revealed bronchopneumonia, pulmonary artery thrombosis, pulmonary edema, mild cardiomegaly and chronic myocarditis, hepatomegaly, and renal vein thrombosis. No Trichinella larvae were identified in the brain, although fresh thromboses were noted in blood vessels. Multiple muscle samples were positive for Trichinella.

The other 8 patients had eaten dried, uncooked, or lightly fried sausage on 2 occasions, November 4 and 11. Onset of symptoms ranged from 2 to 13 days after eating the initial serving of sausage. Of the symptoms commonly associated with clinical trichinosis, all patients had fever (temperatures ranging from 37.9 C to 39.1 C (100.2 F to 102.4 F)), periorbital edema, and muscle pain. Six of 8 patients had abdominal pain; 5 of 8 had diarrhea. Two patients reported headache, and 2 had subconjunctival hemorrhages. Most patients received oral or intravenous steroid therapy; at least 2 also received thiabendazole. Laboratory studies revealed that all of the 8 patients whose blood was examined had eosinophilia (range 4%-57%). Six of 7 patients whose specimens were tested for antibody to Trichinella by the bentonite flocculation (BF) test had titers compatible with recent infection ($\geq$10), and 3 patients from whom additional specimens were tested approximately 7 weeks after onset of symptoms had a $\geq$ 4-fold rise in titer to Trichinella.

Interviews with family members revealed that the sausage had been prepared from a pig purchased and slaughtered at a farm in Green County, New York. Two other pigs kept at the farm over the past year had been slaughtered for consumption by the farm owner. The carcass of the pig that had been purchased was brought to New York City, where it was hung and dried indoors for 2 days; the carcass was then butchered, and sausages were prepared and hung to dry for another 10 days. The sausages were then eaten raw by 8 patients. The ninth patient fried the sausage lightly before eating it. Samples of sausage were examined by the hospital pathologist, who identified multiple encysted larvae of T. spiralis.

Sausage prepared by the farmer with meat from the other 2 pigs was examined at the Animal Parasitology Institute, United States Department of Agriculture (USDA), and found to contain Trichinella. The farmer insisted that the pigs had been fed only grains. However, the farmer went hunting frequently and fed portions of killed game to pet dogs. It was not known whether the pigs had also eaten portions of such meat.

Rhode Island: On November 23, 1981, the Rhode Island Department of Health was notified by the Indochinese Unit of the State Department of Social and Rehabilitative Services of an outbreak in that state. A group of Kampuchean refugees were experiencing swollen eyes, myalgia, and fever; several had been hospitalized. The symptoms plus eosinophilia and elevated creatinine phosphokinase (CPK) levels suggested trichinosis; this diagnosis was confirmed on the basis of findings from a muscle biopsy performed on 1 hospitalized patient. An investigation begun November 24 showed that on October 15, 11 Kampuchean families had shared a meal in the home of 1 family. The main dish was spiced, boiled meat and viscera of a pig that had been purchased the same day from a local farm. Forty-nine family members reported eating the pork either at that meal or subsequently at their homes. Between November 11 and 28, 26 persons became ill with signs and symptoms that included fever and either myalgia or periorbital edema. Trichinosis was confirmed for 24 of the 26 ill persons on the basis of positive BF titers (ranging from 5 to 640) measured in serum specimens obtained between 48 and 75 days after the shared meal. The other 23 asymptomatic family members were negative for Trichinella antibodies. Five of the most severely ill persons were hospitalized; serum CPK values for these patients ranged from 544 to 7,360 International Units (IU). Of the 24 trichinosis patients, who ranged in age from 2 to 66 years, 15 were male. All symptomatic individuals recovered without specific therapy. Staff of the Animal Parasitology Institute, USDA, investigated the farm from which the implicated pig had been obtained. The farmer stated that he only kept a few pigs at a time and that they were not fed-garbage. A land-fill dump was located approximately 2 miles from the farm. Four hogs purchased from the farmer were killed, and the tissues were examined by direct microscopy and digestion of tissues. Blood samples were taken from 5 other pigs for serologic tests. All tissues and serum specimens were negative for Trichinella in these tests. Thirty-two rats trapped and killed on the farm were negative for Trichinella larva.

A second outbreak occurred in Rhode Island in early December 1981. The Rhode Island Department of Health was notified by staff at a hospital emergency room of a group of Laotian refugees with trichinosis-like illness. Investigation revealed that on November 24, 30 members of 4 extended families had shared a meal that included raw pork from a pig purchased at the same farm involved in the first Rhode Island outbreak discussed above. Between December 10 and 29, 13 of these 31 persons became ill with signs and symptoms including fever, periorbital edema, myalgia, eosinophilia (greater than or equal to 10%), and elevated serum CPK levels (>250 IU). Four patients were hospitalized, and T. spiralis larvae were identified in material from muscle biopsies performed on 2 women, ages 22 and 33 years. The patients ranged in age from 7 to 66 years, and 8 of the 13 were male. All recovered. Single or paired-serum specimens obtained from 4 patients and from 4 exposed but asymptomatic persons were sent to CDC for BF testing. All 4 patients and 3 of the 4 asymptomatic persons had single titers of $\geq$ 40 and/or a 4-fold rise in titer between acute- and convalescent-phase specimens. A sample of pork from the implicated meal was examined at CDC in an artificial digestion procedure and revealed a low concentration of *T. spiralis* larvae.

Intensive efforts were made to educate Indochinese families in Rhode Island about the need to cook pork adequately. All state health-care providers and social service agencies were requested to emphasize this recommendation to their refugee populations.

Editorial Note: Trichinosis remains a public health problem in the United States primarily because the infection is enzootic in domestic swine. Surveillance indicates that in 75% of cases for which a probable source is identified, a pork product is incriminated as the source of infection and that ground beef--probably adulterated with pork--accounts for some of the other 25% (1-3). Since 1947, when the Public Health Service began collecting data on cases reported from all the states, the annual incidence of reported human cases has declined from 300-400 cases/year to <150. Since 1966, the annual number of reported cases appears to have stabilized at 100-150, with an average of 1 death/year (1). Factors that have accounted for the decline in the number of humans infected include: 1) state laws that (although directed at preventing other diseases by prohibiting the feeding of raw garbage to swine) have reduced trichinosis in swine; 2) widespread commercial and home freezing of pork, which kills *Trichinae*; 3) consumer awareness of the need to cook pork products adequately; and 4) a national trend to consume more beef than pork.

Trichinosis has occurred most frequently among members of ethnic groups who enjoy eating raw pork (3-4). It has been observed that some outbreaks have occurred among new immigrants who apparently did not understand the need to cook, freeze, or otherwise treat American pork thoroughly in order to kill *Trichinella* larvae. As evidenced by the 2 outbreaks in Rhode Island discussed above, certain groups among the culturally diverse refugees from Southeast Asia must be included in the group at high risk of acquiring trichinosis. Identifying citizens of European ancestry and recent immigrants who belong to ethnic groups that traditionally eat raw pork as "high-risk groups" suggests these groups as targets for special health education as an effort to reduce the potential for further outbreaks.

For approximately two-thirds of the reported cases of pork-associated trichinosis, the incriminated item is a USDA-inspected pork product purchased at a local supermarket or butcher shop (3). The outbreaks reported here are unusual in that the pork was acquired directly from a farm. The most recent data on the prevalence of *Trichinella* in commercially slaughtered swine indicate that approximately 1/1,000 carcasses is infected (5). However, feeding raw garbage to swine, a practice prohibited by law in most states but difficult to enforce, and certain other swine-management practices may result in higher infection rates. Therefore, the rate of infection in hogs purchased directly from farms may be considerably higher than in the 70-80 million hogs that pass through commercial channels each year.

Currently, the Animal Parasitology Institute, USDA, in collaboration with investigators from the Pathobiology Department, University of Pennsylvania, are conducting prevalence studies on hogs in garbage-fed and grain-fed operations in the eastern United States; this study is designed to obtain a better understanding of the roles that wildlife and the feeding of garbage play in *Trichinella* infection in swine.

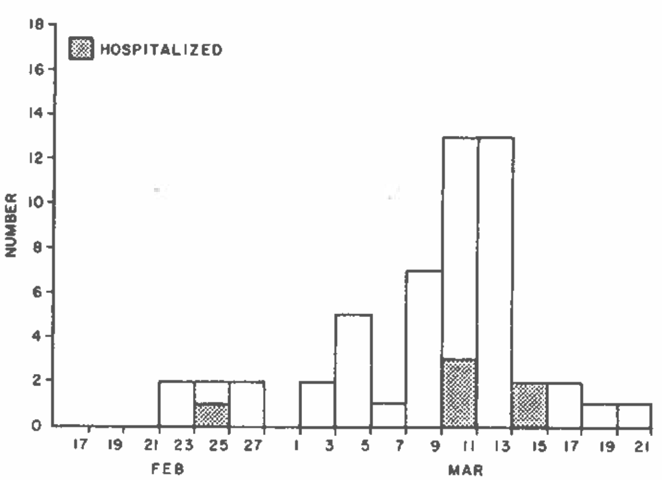
References
1. CDC. Trichinosis surveillance annual summary 1980. Atlanta: CDC, November 1981. (Public Health Service, US Department of Health and Human Services).
2. Juranek DD, Schultz MG. Trichinellosis in humans in the United States: epidemiologic trends. In: Kim CW, Pawlowski ZS, eds. Proceedings of the 4th International Conference in Trichinellosis. Hanover, NH: University Press of New England, 1978:523-8.
3. Schantz PM, Juranek DD, Schultz MG. Trichinosis in the United States 1975: increase in cases attributed to numerous common-source outbreaks. J Infect Dis 1977;136:712-6.
4. Wright WH, Jacobs L, Walton AC. Studies on trichinosis. XVI. Epidemiological considerations based on the examination for Trichinae of 5,313 diaphragms from 189 hospitals in 37 states and the District of Columbia. Public Health Rep 1944;59:669-81.
5. Zimmermann WJ, Zinter DE. The prevalance (sic) of trichiniasis in swine in the United States, 1966-70. HSHMA Health Rep, 1971;86;937-45.

Ciguatera Fish Poisoning -- St. Croix, Virgin Islands of the United States

MMWR 1981;30:138-9

An outbreak of ciguatera (fish poisoning), involving at least 69 persons including 3 tourists, occurred at St. Croix, U.S. Virgin Islands, from February 25 through March 20, 1981. The increase in cases was first noted by the staff at Charles Harwood Memorial Hospital, Christiansted, St. Croix (Figure 1); no increase was noted at the clinic in Frederiksted on the other side of the island.

Fig. 1 NUMBER OF EMERGENCY ROOM PATIENTS DIAGNOSED AS HAVING CIGUATERA FISH POISONING, AND NUMBER OF PATIENTS HOSPITALIZED, CHARLES HARWOOD HOSPITAL, CHRISTIANSTED, ST. CROIX, U.S. VIRGIN ISLANDS, 1981



When the Virgin Islands Department of Health conducted an initial investigation, 20 of 21 patients were found to have eaten red snapper in the 48-hour period before onset of illness. The fish was traced to a fisherman who had landed catches of approximately 1,500 pounds on February 25 and 750 pounds (predominantly snapper) on March 5. The fish from the March 5 catch was distributed to at least 5 restaurants and was also sold by a local retailer from the back of a truck. On March 16, an embargo was placed on the sale of any snapper remaining from this catch.

In a subsequent investigation conducted with the assistance of CDC, a total of 69 individuals were identified who had had ciguatera between February 25 and March 20. Fifty-two (75%) of these 69 individuals had eaten red snapper; 4 had eaten grouper that was traced to the same 2 catches. Symptoms of ill persons who ate red snapper included diarrhea (89% of patients), pain and weakness in the lower extremities (81%), abdominal pain (72%), itching (70%), paresthesias (55%), and vomiting (47%). The mean incubation period was 6.8 hours (range 1/2-48 hours).

Editorial Note: Ciguatera is caused by eating fish containing a toxin thought to be produced by the marine dinoflagellate Gambierdiscus toxicus (1). The toxin is passed up the food chain and concentrated in large predacious fish (such as snapper). The ecologic factors that result in a high percentage of toxic fish in 1 locality or in a single catch are still not well understood.

The diagnosis of ciguatera is clinical, based on the characteristic combination of gastrointestinal and neurologic symptoms (2). The disease is endemic in many areas of the Caribbean and South Pacific; cases are also frequently reported from south Florida (3) and Hawaii. Physicians treating patients who have a syndrome of gastrointestinal symptoms followed by dysesthesias should consider the diagnosis of ciguatera, and should ask about consumption of fish in or from these geographic areas.

References

1. Bagnis R, Chanteau S, Chungue E, Huntel JM, Yasumoto T, Inoue A. Origins of ciguatera fish poisoning. A new dinoflagellate, Gambierdiscus toxicus Adachi and Fukuyo, definitely involved as a causal agent. Toxicon 1980;18:199-208.
2. Hughes JM, Merson MH. Fish and shellfish poisoning. N Engl J Med 1976;295:1117-20.
3. Lawrence DN, Enriguez MB, Lumish RM, Maceo A. Ciguatera fish poisoning in Miami. JAMA 1980;244:254-8.

Mushroom Poisoning Among Laotian Refugees -- 1981

MMWR 1982;31:287-288

In the period December 1-3, 1981 7 Laotian refugees were seen at a Sonoma County California hospital for apparent mushroom poisoning; 6 had nausea, vomiting, diarrhea, dehydration, and elevated liver enzymes. All persons had eaten mushrooms that were gathered and eaten on November 30, although 1 week earlier 20-30 Laotians had eaten mushrooms gathered in the same area without incident. The incubation period was variable, but most patients experienced gastrointestinal distress within 8 hours. Three persons were treated in the intensive care unit, but all recovered and were discharged within 7 days.

Several remaining cooked mushrooms were examined at Sonoma State University; all but 1 were identified as Russula species. The remaining specimen could not be identified, probably because of cooking. The mushrooms examined may not have been representative of those actually consumed.

Laotians customarily gather wild mushrooms in their homeland and attempt to identify poisonous species by boiling the mushrooms with rice; if the rice turns red, the mushrooms are deemed poisonous. Because the Sonoma County mushrooms did not cause a color reaction, it was assumed they were safe to eat.

Editorial Note: Mushroom poisoning can be produced by about 100 of the 2,000 species known. In the United States, mushrooms of the genera Amanita and Galerina produce amanitins and phallotoxins, which are common causes of mushroom poisoning. The most feared fungi are those that produce amanitin, which include the "deathcap" Amanita phalloides. A. phalloides has become increasingly common in the San Francisco bay region in recent years. The odor of fresh A. phalloides is similar to raw potatoes. Symptoms generally begin 6-24 hours after ingestion and may include the explosive onset of violent abdominal pain, vomiting, diarrhea, hematuria, fever, tachycardia, hypotension, rapid volume depletion, fluid and electrolyte imbalance, and extreme thirst. After a short phase of improvement, hepatic, renal, and central nervous system damage may ensue. The mortality rate is 50%, and those who recover do so slowly. Treatment is supportive, and thioctic acid, charcoal hemoperfusion, and vitamin C may be useful (1,2).

Other genera of mushrooms, including the Russula genus implicated in the California outbreak, produce less lethal toxins. The Russula toxin has not been identified, but it results in a shorter incubation period--1-2 hours--followed by minor gastrointestinal and parasympathetic symptoms and hallucinations (1). *Russula emetica* can produce additional toxins, including muscarine.

Most cases of mushroom poisoning occur in late summer and early fall. Early abundant rains and mild temperatures in northern California have produced a profusion of mushrooms, some of which are poisonous. Nontoxic mushrooms may grow in the same area with toxic ones, and even trained mycologists may confuse toxic varieties with edible ones because of the extensive variations among species. There are no simple tests to identify poisonous mushrooms and no safe ways to detoxify the poisonous varieties.

Identification of implicated mushrooms may be difficult if specimens have been prepared and cooked. Since a variety of mushrooms may have been ingested in most poisoning situations, reliance cannot be placed on the initial symptoms. Gastric contents, stool, and mushroom samples may be assayed for toxins by radioimmunoassay (3).

The San Francisco Poison Control Center recommends routine administration of ipecac after ingestion of any wild mushrooms of questionable identification. The Infectious Disease Section offers its assistance in suspected mushroom poisoning, and information is available from local mycologic societies, colleges, and poison control centers.

In the last 5 years, 16 outbreaks involving 44 cases of mushroom poisoning were reported to CDC; 23 cases were from California. In 1981 in California, 1 death in Santa Cruz County and 2 in Marin County were attributed to mushroom poisoning.

References

1. Becker CE, Tong TE, Boerner U, Roe RL, Scott RAT, MacQuarrie MB. Diagnosis and treatment of *Amanita phalloides*-type mushroom poisoning. West J Med 1976;125:100-9.
2. Wauters JP, Rossel C, Farquet JJ. *Amanita phalloides* poisoning treated by early charcoal haemoperfusion. Brit Med J 1978;2:1465.
3. Mushroom poisoning. Lancet 1980;2:351-2.

J. Bibliography

GENERAL

1. Bryan FL. Emerging foodborne diseases. I. Their surveillance and epidemiology. II. Factors that contribute to outbreaks and their control. J Milk Food Technol 1972;35:618-25, 632-8.
2. Bryan FA. Factors that contribute to outbreaks of foodborne disease. J of Food Protection 1978;41:816-27.
3. Bryan FL. Foodborne diseases in the United States associated with meat and poultry. J of Food Protection 1980;43:140-50.
4. Food Research Institute. Annual report 1979, University of Wisconsin, Madison, Wisconsin.
5. Horwitz MA. Specific diagnosis of foodborne disease. Gastroent 1977;73:375-81.
6. Riemann H, ed. Foodborne infections and intoxications. New York: Academic Press, 1969.
7. Sours HE, Smith DG. Outbreaks of foodborne disease in the United States, 1972-1978. J Infect Dis 1980;142:122-5.

BACTERIAL

Bacillus cereus

1. Giannella RA, Brasile L. A hospital foodborne outbreak of diarrhea caused by *Bacillus cereus*: Clinical, epidemiologic, and microbiologic studies. J Infect Dis 1979;139:366-70.
2. Mortimer PR, McCann G. Food poisoning episodes associated with *Bacillus cereus* in fried rice. Lancet 1974;1:1043-5.
3. Terranova W, Blake PA. *Bacillus cereus* food poisoning. N Engl J Med 1978;298:143-4.
4. Turnbull PCB, Kramer JM, Torgensen K, Gilbert RJ, Melling J. Properties and production characteristics of vomiting, diarrheal, and necrotizing toxins of *Bacillus cereus*. Am J Clin Nutr 1979;32:219-28.

Brucella

1. Buchanan TM, et al. Brucellosis in the United States 1960-1972. Med 1974;53:403-39.
2. Fox MD, Kaufman AF. Brucellosis in the United States, 1965-1974. J Infect Dis 1977;136:312-6.
3. Spink WW. The nature of brucellosis. Minneapolis, Lund Press, Inc., 1956.
4. Young EJ, Suvannaparrat U. Brucellosis outbreak attributed to ingestion of unpasteurized goat cheese. Arch Intern Med 1975;135:240-3.

Campylobacter

1. Blaser MJ, et al. *Campylobacter* enteritis. N Engl J Med 1981;305:1444-52.

Clostridium botulinum

1. Center for Disease Control. Botulism in the United States, 1899-1977. Handbook for Epidemiologists, Clinicians, and Laboratory Workers, CDC, Atlanta, May 1979.
2. Cherington M. Botulism. Ten-year experience. Arch Neurol 1974;30:432-7.
3. Dowell VR Jr, McCroskey LM, Hatheway CL, et al. Coproexamination for botulinal toxin and *Clostridium botulinum* - A new procedure for laboratory diagnosis of botulism. JAMA 1977;238:1829-32.

4. Koenig MG, Spichard A, Cardella MA, et al. Clinical and laboratory observations of type E botulism in man. Med 1964;43:517-45.

5. Koenig MG, Drutz DJ, Mushlin AI, et al. Type B botulism in man. Am J Med 1967;42:208-19.

6. Morris JG, Hatheway CL. Botulism in the United States, 1979. J Infect Dis 1980;142:302-5.

Clostridium perfringens

1. Bryan FL. What the sanitarian should know about Clostridium foodborne illness. J Milk Food Technol 1969;32:381-9.

2. Lowenstein MS. Epidemiology of Clostridium perfringens food poisoning. N Engl J Med 1972;286(19):1026-7.

3. Shandera WX, Tacket CO, Blake PA. Food poisoning due to Clostridium perfringens in the United States. J Infect Dis 1983;143:167-170.

4. Stringer MF, Turnbull PCB, Gilbert RJ. Application of serological typing to the investigation of outbreaks of Clostridium perfringens food poisoning, 1970-1978. J Hyg (Camb) 1980;84:443-56.

Escherichia coli

1. Marier R, Wells JG, Swanson RC, Callahan W, Mehlman IJ. An outbreak of enteropathogenic Escherichia coli foodborne disease traced to imported French cheese. Lancet 1973;2:1376-8.

2. Sack RB. Human diarrheal disease caused by enterotoxigenic Escherichia coli. Ann Rev Microbiol 1975;29:333-53.

Salmonella

1. Aserkoff B, Schroeder SA, Brachman PS. Salmonellosis in the United States--A five-year review. Am J Epidemiol 1970;92:13-24.

2. Bryan FL. What the sanitarian should know about salmonellae and staphylococci in non-dairy foods. II. Salmonellae. J Milk Food Technol 1968;31:131-40.

3. Cohen ML, Blake PA. Trends in foodborne salmonellosis outbreaks: 1963-1975. J Food Protection 1977;40:798-800.

Shigella

1. Black RE, Craun GF, Blake PA. Epidemiology of comon-source outbreaks of shigellosis in the United States, 1961-1975. Am J Epidemiol 1978;108:47-52.

2. Donadio JA, Gangarosa EJ. Foodborne shigellosis. J Infect Dis 1969;119:666-8.

Staphylococcus

1. Bryan FL. What the sanitarian should know about salmonellae and staphylococci in non-dairy foods. I. Staphylococci. J Milk Food Technol 1968;31:110-16.

2. Merson MH. The epidemiology of staphylococcal foodborne disease. Proceedings of Staphylococci in Foods Conference, Pennsylvania State University, University Park, Pennsylvania, 1973, pp 20-37.

3. Minor TE, Marth EH. Staphylococcus aureus and staphylococcal food poisoning. J Milk Food Technol 1972;34:21-39, 77-83, 227-241, 1973;35:447-76.

Group A Streptococcus

1. Hill HR, Zimmerman RA, Reid GVK, Wilson E, Kitton RM. Foodborne epidemic of streptococcal pharyngitis at the United States Air Force Academy. N Engl J Med 1969;280:917-21.

2. McCormick JB, Kay D, Hayes M, Feldman RA. Epidemic streptococcal sore throat following a community picnic. JAMA 1976;236:1039-41.

Vibrio cholerae O1

1. Blake PA, Allegra DT, Snyder JD, Barrett TJ, McFarland L, Caraway CT, Feeley JC, Craig JP, Lee JV, Puhr ND, Feldman RA. Cholera--A possible endemic focus in the United States. N Engl J Med 1980;302:305-309.

2. Gangarosa EJ, Mosley WH. Epidemiology and surveillance of cholera. Cholera, edited by Barua D, Burrows W. Philadelphia, London, Toronto, WB Saunders Co., 1974, p 381.

Vibrio cholerae Non-O1

1. Blake PA, Weaver RE, Hollis DG. Diseases of humans (other than cholera) caused by Vibrios. Ann Rev Microbiol 1980;34:341-67.

2. Hughes JM, Hollis DG, Gangarosa EJ, Weaver RE. Non-cholera vibrio infections in the United States--Clinical, epidemiologic, and laboratory features. Ann Intern Med 1978;88:602-6.

3. Morris JG, Wilson R, Davis BR, Wachsmuth IK, Conradine FR, Wathen HG, Pollard RA, Blake PA. Non-O Group 1 Vibrio cholerae gastroenteritis in the United States. Ann Intern Med 1981;94:656-8.

Vibrio parahaemolyticus

1. Barker WH. Vibrio parahaemolyticus outbreaks in the United States. Lancet 1974;1:551-4.

2. Blake PA, Weaver RE, Hollis DG. Diseases of humans (other than cholera) caused by Vibrios. Ann Rev Microbiol 1980;34:341-67.

3. Lawrence DN, Blake PA, Yashuk JC, Wells JG, Creech WB, Hughes JH. Vibrio parahaemolyticus gastroenteritis outbreaks aboard two cruise ships. Am J Epidemiol 1979;10:71-80.

CHEMICAL

Heavy Metals

Cadmium

1. Baker TD, Hafnew WG. Cadmium poisoning from a refrigerator shelf used as an improvised barbecue grill. Public Health Rep 1961;76:543-4.

Copper

1. Hopper SH, Adams HS. Copper poisoning from vending machines. Public Health Rep 1958;73:910-4.

2. Semple AB, Parry WH, Phillips DE. Acute copper poisoning: An outbreak traced to contaminated water from a corroded geyser. Lancet 1960;2:700-1.

Tin

1. Barker WH, Runte V. Tomato juice-associated gastroenteritis. Washington and Oregon, 1969. Am J Epidemiol 1972;96:219-26.

Zinc

1. Brown MA, Thom JV, Orth GL, et al. Food poisoning involving zinc contamination. Arch Environ Health 1964;8:657-60.

Ciguatoxin

1. Bagnis R, Chanteau S, Chungue E, Huntel JM, Yasumoto T, Inoue A. Origins of ciguatera fish poisoning: A new dinoflagellate, Gambierdiscus toxicus Adachi and Fukuyo, definitely involved as a causal agent. Toxicon 1980;18:199-208.

2. Halstead BW. Poisonous and venomous marine animals of the world. Princeton, The Darwin Press, 1978, pp 325-402.

3. Hughes JM, Merson MH. Fish and shellfish poisoning. N Engl J Med 1976;295:1117-20.

4. Lawrence DN, Enriquez MB, Lumish RM, Maceo A. Ciguatera fish poisoning in Miami. JAMA 1980;244:254-8.

5. Engleberg NC, Morris JG, Lewis J, McMillen JP, Pollard RA, Blake PA. Ciguatera fish poisoning: A major common-source outbreak in the U.S. Virgin Islands. Ann Intern Med 1983;19:336-337.

6. Morris JG, Lewin P, Smith CW, Blake PA, Schneider R. Ciguatera fish poisoning: Epidemiology of the disease on St. Thomas, U.S. Virgin Islands. Am J Trop Med Hyg 1982;31:574-8.

7. Morris JG, Lewin P, Hargrett NT, Smith CW, Blake PA, Schneider R. Clinical features of ciguatera fish poisoning: A study of the disease in the US Virgin Islands. Arch Intern Med 1982;142:1090-2.

Puffer Fish (tetrodotoxin)

1. Halstead BW. Poisonous and venomous marine animals of the world. Princeton, The Darwin Press, 1978, pp 437-548.

2. Torda TA, Sinclair E, Ulyatt DB. Puffer fish (tetrodotoxin) poisoning: Clinical record and suggested management. Med J Aust 1973;1:599-602.

Scombrotoxin

1. Arnold SH, Brown WD. Histamine toxicity from fish products. Advances in Food Research 1978;24:113-54.

2. Gilbert RJ, Hobbs G, Murray CK, Cruickshank JG, Young SEJ. Scombrotoxic fish poisoning: Features of the first 50 incidents to be reported in Britain (1976-1979). British Med J 1980;281:71-2.

3. Halstead BW. Poisonous and venomous marine animals of the world. Princeton, The Darwin Press, 1978, pp 417-35.

4. Hughes JM, Merson MH. Fish and shellfish poisoning. N Engl J Med 1976;295:1117-20.

5. Merson MH, Baine WB, Gangarosa EJ, Swanson RC. Scombroid fish poisoning: Outbreak traced to commercially canned tuna fish. JAMA 1974;228:1268-9.

Monosodium Glutamate

1. Schaumburg HH, Byck R, Gerstl R, Mashman JH. Monosodium L-glutamate; its pharmacology and role in the Chinese restaurant syndrome. Science 1969;163:826-8.

Mushroom Poison

1. Becker CE, et al. Diagnosis and treatment of Amanita phalloides type mushroom poisoning - use of thiotic acid. West J Med 1976;125:100-9.

2. Benedict RG. Mushroom toxins other than Amanita, Kadis S, Ciegler A, Ajl SJ: Microbial toxins, Vol 8 Fungal toxins, New York and London, Academic Press, 1972, pp 281-320.

3. Mitchel DH. Amanita mushroom poisoning. Ann Rev Med 1980;31:51-7.

4. Tyler VE. Poisonous mushrooms: Progress in chemical toxicology. Vol 1, edited by Stolman A, New York Academic Press, 1963, pp 339-84.

Paralytic and Neurotoxic Shellfish Poison

1. Halstead BW. Poisonous and venomous marine animals of the world. The Darwin Press, Princeton, 1978, pp 43-78.

2. Hughes JM, Merson MH. Fish and shellfish poisoning. N Engl J Med 1976;295:1117-20.

3. Music SI, Howell JT, Brumback CL. Red tide: its public health implications. J Fla Med Assoc 1973;60:27-9.

PARASITIC

Anisakidae

1. Chitwood MD. Nematodes of medical significance found in market fish. Am J Trop Med Hyg 1970;19:599-602.

Trichinella spiralis

1. Gould SE. Trichinosis in man and animals. Springfield, Ill, Charles C. Thomas, 1970.

2. Zimmerman WJ, Steele JH, Kagan IG. Trichinosis in the U.S. population 1966-1970--Prevalence and epidemiologic factors. Health Services Rep 1973;88:606-23.

Toxoplasma gondii

1. Kean BH, Kimball AC, Christensen WN. An epidemic of acute toxoplasmosis. JAMA 1969;208:1002-4.

VIRAL

Hepatitis A

1. Cliver DO. Implications of foodborne infectious hepatitis. Public Health Rep 1966;81:159-65.

2. Leger RT, Boyer KM, Pattison CP, et al. Hepatitis A: Report of a common-source outbreak with recovery of a possible etiologic agent. I. Epidemiologic studies. J Infect Dis 1975;131:163.

3. Gravelle CR, Hornbeck CL, Maynard JE, et al. Hepatitis A: Report of a common-source outbreak with recovery of a possible etiologic agent. II. Laboratory studies. J Infect Dis 1975;131:167-71.

STATE AND TERRITORIAL EPIDEMIOLOGISTS AND STATE LABORATORY DIRECTORS

The State and Territorial Epidemiologists are the key to all disease surveillance activities, and their contributions to this report are gratefully acknowledged. In addition, valuable contributions are made by State Laboratory Directors.

State	State Epidemiologist	State Laboratory Director
Alabama	Wallace E. Birch, DVM	James L. Holston, Jr., DrPH
Alaska	John P. Middaugh, MD	Harry J. Colvin, PhD
Arizona	Richard L. Coppedge, MD, Acting	Jon M. Counts, DrPH
Arkansas	John Paul Lofgren, MD	Robert L. Horn
California	James Chin, MD	John M. Heslep, PhD
Colorado	Stanley W. Ferguson, PhD	C. D. McGuire, PhD
Connecticut	Vernon D. Loverde, MD	Jesse Tucker, PhD
Delaware	Donald R. Cowan, DDS	Mahadeo P. Verma, PhD
District of Columbia	Martin E. Levy, MD	James B. Thomas, PhD, Acting
Florida	Jeffrey J. Sacks, MD, Acting	Eldert C. Hartwig, Jr., ScD
Georgia	R. Keith Sikes, DVM	Frank M. Rumph, MD
Guam	Robert L. Haddock, DVM	Luis P. Flores
Hawaii	Mona Bomgaars, MD, Acting	Albert I. Oda
Idaho	Charles D. Brokopp, DrPH	D. W. Brock, DrPH
Illinois	Byron J. Francis, MD	Harry C. Bostick
Indiana	Charles L. Barrett, MD	T. L. Eddleman
Iowa	Laverne A. Wintermeyer, MD	W. J. Hausler, Jr., PhD
Kansas	Donald E. Wilcox, MD	Roger H. Carlson, PhD
Kentucky	M. Ward Hinds, MD	B. F. Brown, MD
Louisiana	Charles T. Caraway, DVM	Henry Bradford, PhD
Maine	Kathleen F. Gensheimer, MD, Acting	Philip W. Haines, DrPH
Marshall Islands*	Isao Kisino, MD	Vacant
Maryland	Ebenezer Israel, MD	J. Mehsen Joseph, PhD
Massachusetts	Nicholas J. Fiumara, MD	George F. Grady, MD
Michigan	Kenneth R. Wilcox, Jr., MD	George R. Anderson, DVM
Micronesia*	Eliuel K. Pretrick, MO	Vacant
Minnesota	Andrew G. Dean, MD	C. Dwayne Morse, DrPH
Mississippi	Wm. E. Riecken, Jr., MD	R. H. Andrews, MS
Missouri	H. Denny Donnell, Jr., MD	Elmer Spurrier, DrPH
Montana	John S. Anderson, MD, Acting	Douglas Abbott, PhD
Nebraska	Paul A. Stoesz, MD	John Blosser
Nevada	John H. Carr, MD, Acting	George Reynolds, MD
New Hampshire	John M. Horan, MD	Mrs. Veronica Stukas, Acting
New Jersey	William E. Parkin, DVM	Bernard F. Taylor, PhD
New Mexico	Jonathan M. Mann, MD	Loris Hughes, PhD
New York State	Richard Rothenberg, MD	David O. Carpenter, MD
New York City	Stephen M. Friedman, MD	Bernard Davidow, PhD
North Carolina	Martin P. Hines, DVM	Mildred A. Kerbaugh
North Dakota	James L. Pearson, DrPH, Acting	A. A. Gustafson
Northern Mariana Islands*	Jose T. Villagomez, MO	Vacant
Ohio	Thomas J. Halpin, MD	Gary D. Davidson, DrPH
Oklahoma	Gregory R. Istre, MD, Acting	Garry McKee, PhD
Oregon	John A. Googins, MD	Vacant
Palau*	Anthony H. Polloi, MO, Acting	Vacant
Pennsylvania	Charles W. Hays, MD	Vern Pidcoe, DrPH
Puerto Rico	Antonio Hernandez, MD	Jose L. Villamil
Rhode Island	Jason Weisfeld, MD, Acting	Raymond G. Lundgren, PhD
South Carolina	Richard L. Parker, DVM	Arthur F. DiSalvo, MD
South Dakota	Kenneth A. Senger	A. Richard Melton, DrPH
Tennessee	Robert H. Hutcheson, Jr., MD	Michael W. Kimberly, DrPH
Texas	Charles R. Webb, Jr., MD	Charles E. Sweet, DrPH
Utah	Richard E. Johns, Jr., MD	Francis M. Urry, PhD
Vermont	Richard L. Vogt, MD	Dymitry Pomar, DVM
Virginia	Grayson B. Miller, Jr., MD	Frank W. Lambert, Jr., DrPH
Virgin Islands	John N. Lewis, MD	Norbert Mantor, PhD
Washington	John M. Kobayashi, MD	Jack Allard, PhD
West Virginia	Loretta E. Haddy, MS	John W. Brough, DrPH
Wisconsin	Jeffrey P. Davis, MD	Ronald Laessig, PhD
Wyoming	Harry C. Crawford, MD	Donald T. Lee, PhD

*Formerly Trust Territory of the Pacific Islands

DEPARTMENT OF HEALTH & HUMAN SERVICES

Public Health Service
Centers for Disease Control
Atlanta GA 30333

HHS Publication No. (CDC) 84-8185