BOTULISM

IN THE UNITED STATES

REVIEW OF CASES, 1899-1967
AND HANDBOOK FOR EPIDEMIOLOGISTS,
CLINICIANS, AND LABORATORY WORKERS

U. S. DEPARTMENT
OF HEALTH, EDUCATION, AND WELFARE
PUBLIC HEALTH SERVICE

PREFACE

This report reviews the epidemiology of botulism in the United States since 1899, the problems of clinical and laboratory diagnosis, and current concepts in treatment. It was written in recognition of the need for a comprehensive and up-to-date working manual for epidemiologists, clinicians, and laboratory workers.

A trivalent antitoxin containing anti-A, B, and E has been long sought for the treatment of botulism. Publication of this review is timed to coincide with the licensure of such a preparation, which is now available from the National Communicable Disease Center (NCDC) for use in outbreaks of suspected or proven botulism.

The assistance of Drs. K. F. Meyer, B. Eddie, and M. G. Koenig, in reviewing this manuscript is gratefully acknowledged. Drs. R. W. Armstrong, E. R. Eichner, G. T. Curlin, T. M. Vernon, and W. E. Woodward, who were assigned as Epidemic Intelligence Service Officers to the Enteric Diseases Unit, Epidemiology Program, and Dr. M. P. Magovern, formerly of the Laboratory Program, contributed substantially to the preparation of this review.

The excellent review of Drs. K. F. Meyer and B. Eddie (1950), "Fifty Years of Botulism in the United States," is the source of all statistical information for 1899 - 1949. Data for 1950 - 1967 are based on outbreaks reported to the NCDC.

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STATE EPIDEMIOLOGISTS AND STATE LABORATORY DIRECTORS

Key to all disease surveillance activities are the physicians who serve as State epidemiologists. They are responsible for collecting, interpreting, and transmitting data and epidemiological information from their individual States; their contributions to this report are gratefully acknowledged. In addition, valuable contributions are made by State Laboratory Directors; we are indebted to them for their valuable support.

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I. EMERGENCY ASSISTANCE

Prompt diagnosis and early treatment of botulism are essential to minimize the otherwise great risk of death. The National Communicable Disease Center offers 24-hour diagnostic consultation, epidemic assistance, laboratory testing services, and antitoxin. Therapeutic preparations available are described in Section IV, Therapy.

These services may be obtained by calling the NCDC at any of the numbers listed below:

		Day Phone	Night Phone
1.	NCDC, Atlanta	404-633-3311	404-634-2561
		extensions	404-633-2176
	Dr. Gangarosa	3751	or 404-938-9195*
	Dr. Brachman	3684	404-373-5173*
	Dr. Bennett	3686	404-443-9373*
	Dr. Dowell	3654	404-633-9029*
2.	NCDC, Kansas City	816-374-3989	816-374-3989
		816-374-7000	
3.	Alaska Native Medical Center, Anchorage	907-277-1577	907-277-1577
		FTS-202-967-1221	
		Washington operator	

* Home phones of consultants, subject to change.

II. EPIDEMIOLOGY OF BOTULISM

A. General

Botulism is an intoxication due to the action of a protein neurotoxin elaborated by Clostridium botulinum. Six toxigenic types of C. botulinum are recognized on the basis of antigenically distinct toxins produced by different strains of the organism. Four types--A, B, E, and F--are the principal causes of the disease in man; types C and D are usually associated with botulism in birds and mammals. The disease is rare but often fatal. It is usually due to ingestion of toxin, but it has been caused by infection of a wound with C. botulinum with toxin production in vivo (Petty, 1965).

"Botulism" comes from the Latin botulus, meaning sausage. This derivation, although historically important, has lost its significance, since plant rather than animal products are more common vehicles. Sausage is rarely the cause of botulism in the United States.

Clostridium botulinum is an anaerobic gram-positive bacillus which produces heat-resistant spores. The organism is widely distributed in nature and is frequently found in both terrestrial and marine environments (Meyer and Dubovsky, 1922; Eklund and Poysky, 1966; Ward et al., 1967). Under suitable conditions which will allow germination of spores (e.g., in improperly preserved foods) a heat labile toxin is elaborated which is one of the most poisonous substances known. Toxin production, particularly by type E organisms, can occur at temperatures as low as 38°F and strict anaerobic conditions (i.e. complete absence of oxygen) are not required (Foster and Sugiyama, 1967). After ingestion, the toxin is absorbed, causing symptoms simulating denervation. Paralysis is due to the inhibition of acetylcholine at peripheral nerve endings (Ambache, 1948).

B. Incidence

From 1899 through 1949, there were 477 outbreaks recorded in the United States; from 1950 through 1967 there were an additional 163 reported to the NCDC, for a total of 640 (Table 1). The average number of outbreaks per year during both periods was 9.5.

During the period 1899 - 1949, there were 1283 cases of botulism; in 1950 - 1967, 386 additional cases were reported for a total of 1669. The average number of cases per outbreak was 26 during the first period and 23 during the second period. Since 1899, there have been 948 reported deaths.

This is undoubtedly a conservative measure of the actual incidence, as additional deaths attributed to botulism are found in U.S. Vital Statistics Reports, which were not reported to the NCDC. A line listing of outbreaks reported to the NCDC 1950 - 1967 is included as an appendix to this report.

Of the 640 outbreaks, 21.6 percent were due to the type A strain, 5.3 percent to type B, 2.7 percent to type E, and 0.3 percent to type F; in 70.1 percent, the type was not determined (Table 1). In recent years, however, type E cases have increased in frequency, while cases due to types A and B have declined (Figure 1). The proportion of diagnosed cases in which the type was undetermined remained high; 76 percent of cases in the period 1950 - 1959 were due to unknown causes, compared with 53 percent of cases during 1960 - 1967. During the period 1960 - 1967, type E accounted for most cases reported by specific type, followed by types A, B, and F in that order. The decline in botulism since 1935 is probably due to improved canning methods in industry and in the home.

C. Morbidity and Mortality

During the period 1899 - 1949, the death-to-case ratio remained high, at levels above 60 percent, but since about 1950 there has been a gradual improvement (Figures 2 and 3). This decline in death-to-case ratio is probably the result of improvements in intensive care of acute respiratory failure and the beneficial effect of botulinum antitoxin.

The decline in the death-to-case percentage is more striking for types A and B than for type E (Table 2). During the period 1960 - 1967, type E had the highest death-to-case ratio, which was more than twice as high as for type A and four times as high as for type B.

The age-specific-case fatality ratio was significantly higher for adults than for children from 1962 through 1967, during which time data were collected on 98 cases reported by age (Figure 4). This is probably a dose-related phenomenon rather than an inherent resistance of the young, since children are often more fastidious in their eating habits than adults and are less likely to eat foods that are contaminated.

D. Geographic Distribution

There are distinctive geographic distributions. Outbreaks have been reported from 44 states (Figure 5), but five western states, California, Washington, Colorado, Oregon, and New Mexico, accounted for well over half of all reported outbreaks.

Of the 139 type A outbreaks recorded from 1899 through 1967, 128 (91 percent) were in western states (Figure 6), i.e., west of the Mississippi River. California, Washington, New Mexico, and Oregon accounted for 43, 11.5, 8, and 7.2 percent, respectively, of type A outbreaks. Twenty-six states, most of them in the East, have never reported type A outbreaks.

Type B has been reported as the cause of outbreaks in 15 states (Figure 7). Of the 34 type B outbreaks, documented from 1899 through 1967, 23 were reported from eastern states; New York reported 10 outbreaks.

Type E outbreaks have been reported from 10 states (Figure 8). A geographic predilection is apparent in Alaska and in the Great Lakes area.

California, which has reported more outbreaks of botulism than any other state, has had only one outbreak due to type E, and this involved a nonmarine product, mushrooms. New York State, which ranked first in type B outbreaks, reported only one outbreak due to type A, and this was also traced to mushrooms. Alaska has never reported types A or B but is the leading state reporting type E.

These regional distributions of outbreaks by toxin type are in keeping with a spore survey of soil samples reported by Meyer and Dubovsky (1922). These investigators found a predominance of type A in soil specimens from the West and a predominance of type B in soils of the Northeast and Central States. Types A and B were not found in soil samples from Alaska. Type E spores have been found in marine life and sediment from the Great Lakes (Bott et al., 1966) and from the Pacific Northwest (Eklund and Poysky, 1966).

E. Food Sources and Products Causing Outbreaks

Until a few years ago, outbreaks of botulism for which toxin types were determined were most frequently caused by type A or B toxins (Figure 1)and were usually associated with ingestion of home-canned vegetables, fruits, or meat products. Botulism due to type E toxin, although recognized as having occurred in the United States as early as 1932, was not recognized as a major problem until 1963, when 22 cases of this type were reported (Rogers, 1964; Rogers et al., 1964). Sixteen of the 17 outbreaks of type E botulism have been traced to fish or fish products; as noted above, one outbreak was traced to canned mushrooms (Geiger, 1941). One outbreak of type F botulism has been reported in this country; it was traced to home-prepared venison jerky (National Communicable Disease Center, Morbidity and Mortality Weekly Report, Vol. 15, No. 41, Oct. 15, 1966, and No. 42, Oct. 22, 1966).

Home canned and preserved foods have accounted for most outbreaks since 1910 (Table 3). A smaller number have been ascribed to commercially preserved foods. The sources of many outbreaks have remained unknown.

The type of toxin isolated from various food products in which the toxin type was determined is shown in Table 4. Vegetables, fruits, fish, and condiments were the most important vehicles of toxin. Beef, milk products, pork, poultry, and other vehicles caused relatively fewer outbreaks. It is a widely held view that if botulism is caused by a marine product, type E toxin is responsible, but of the 23 outbreaks caused by fish products, 16 were due to type E, 5 were due to type A, and 2 to type B. It is also noteworthy that a nonmarine product, mushrooms, caused a type E outbreak.

III. DIAGNOSIS

A. General

Botulism should be considered the diagnosis for patients who have acute cranial nerve impairment with symmetrical, descending weakness or paralysis. Diplopia, dysarthria, and dysphagia are common symptoms of botulism. There are no sensory changes, and mental processes are clear. Pupils are dilated and fixed, the pulse is normal, fever is absent, cerebrospinal fluid is normal, deep tendon reflexes are depressed but equal and symmetrical, and there are no pyramidal tract signs. Mucous membranes of the mouth, tongue, and pharynx are usually extremely dry. Gastrointestinal symptoms are variable. Major complications which may alter the clinical picture include respiratory failure and pulmonary and urinary infection.

B. Cardinal Features

1. Fever is absent early in the disease, but may develop later with pneumonia or other complications.
2. Mental processes are clear. Patients may be anxious or agitated for obvious reasons, but some are unusually drowsy; however, most patients are responsive.
3. Pulse is normal, or slow, but it may speed up after hypotension develops.
4. Although vision may be impaired and hearing may be distorted, there is no numbness or decreased perception of touch, and no paresthesia or other sensory disturbance.
5. Neurological manifestations are symmetrical.

Onset of signs and symptoms can begin as soon as a few hours or as late as 8 days after ingestion of contaminated food; the usual time lapse is 18 to 36 hours (Meyer, 1964). Generally, persons with early onset of illness (i.e., within 24 hours) will be severely affected, be more likely to die, and if they survive to have a protracted course (Koenig et al., 1964, and 1967). Severity of illness is sometimes, but not always, due to ingestion of large quantities of the contaminated food; however, fatal cases have been reported after tasting only a small piece of bean pod or asparagus. Some exposed individuals may be spared because of unequal distribution of toxin within food and perhaps varying human susceptibility to toxin.

Table 5 summarizes symptoms and signs of types A, B, and E botulism reported to the NCDC in 56 outbreaks since 1953. Gastrointestinal symptoms, dizziness, and vertigo were found in outbreaks caused by all toxins but were more common in type B and E outbreaks. Signs and symptoms were otherwise equally common in outbreaks caused by all types. Postural hypotension has been emphasized as an important sign (Rogers, 1964), but it was reported only once in these 56 outbreaks.

The first manifestations of illness in most patients with type E botulism are gastrointestinal: nausea or vomiting, substernal burning or pain, abdominal distention, decreased bowel sounds, and dilated loops of small bowel on radiologic examination (Rogers, 1964; Meyer, 1964). Some patients have initial transitory diarrhea, but later become constipated. Many, but not all, patients with type B and some with type A also have initial gastrointestinal symptoms (Koenig et al., 1967; Stricker and Geiger, 1924; Tucker and Swanson, 1939; Dolman and Murakami, 1961). These symptoms and signs may be so prominent that clinicians may be misled to diagnose the illness as appendicitis, bowel obstruction, or diaphragmatic myocardial infarction. Mucous membranes of the mouth, tongue, and pharynx may be red, dry, and painful leading to the misdiagnosis of pharyngitis (Koenig et al., 1964 and 1967).

C. Differential Diagnosis

Diseases most likely to be confused with botulism include myasthenia gravis, cerebrovascular accidents involving branches of the basilar artery in the mid-brain, Guillain-Barré syndrome, tick paralysis, chemical intoxications (e.g., carbon monoxide, barium carbonate, methyl chloride, methyl alcohol, organic phosphorus compounds, atropine), trichinosis, and diphtheria.

During the period 1964 - 1967, NCDC investigated 53 suspected outbreaks of botulism, of which 18 (34 percent) proved to be botulism (Table 6). Among the remainder, staphylococcal food poisoning accounted for 9 (17 percent); chemical food poisoning, carbon monoxide poisoning, and Guillain-Barré syndrome each accounted for 3 to 5 percent of outbreaks; and the remainder were attributed to a variety of other disorders.

Thus, many illnesses have been mistaken for botulism. In the common bacterial food poisonings (staphylococcal intoxication, C. perfringens food poisoning, and salmonella or shigella gastroenteritis), diarrhea, and the absence of cranial nerve involvement are usually sufficiently distinctive to distinguish them from botulism. Chemical food poisoning, while sometimes causing neurological manifestations, almost always has its onset within minutes or hours after consumption of contaminated food. Atropine poisoning in a recent episode was initially diagnosed as botulism; however, the very rapid onset, flushing of the face, bizarre hallucinations, and other findings pointed to atropine poisoning (Eichner et al., 1967). Shellfish poisoning and tetraodon (tropical fish) and other forms of fish poisoning have rapid onsets and often cause characteristic patterns of paresthesias, tremors, and other signs (Dack, 1957). Mushroom poisoning (Amanita phalloides) causes severe abdominal pain, violent vomiting and diarrhea, and coma (Dack, 1957).

Cerebrovascular accidents usually cause localized signs, such as prominent distal muscular paresis, sensory losses, and usually asymmetrical deep tendon reflex changes. The absence of fever helps exclude poliomyelitis, meningitis, and encephalitis. Myasthenia gravis can be differentiated by the presence of muscular fatigability and the response to the Tensilon test. Guillain-Barré syndrome can closely mimic botulism, but muscular cramps, paresthesias, and elevated spinal fluid protein in the absence of cells help distinguish this disease. Especially after surgery, certain antibiotic drugs, e.g., neomycin, streptomycin, kanamycin, polymyxin, bacitracin, dihydrostreptomycin, colistin, and combinations of these, may induce symmetrical flaccid paralysis (McQuillen et al., 1968).

D. Laboratory Findings*

The most effective way to confirm a diagnosis of botulism is to demonstrate toxicity of the patient's serum for mice and to prove specificity of the toxin by neutralization tests with botulinum antitoxins (Koenig et al., 1964 and 1967). The usual laboratory tests are of little value in diagnosing botulism. Blood counts, urinalyses, serum electrolytes, cerebrospinal fluids, and blood enzyme studies are normal unless there are secondary complications. An electrocardiogram is not particularly helpful, but sometimes nonspecific S-T segment changes and T wave inversion are noted (Koenig et al., 1964, and 1967).

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* Methods for laboratory diagnosis of botulism are given in Section V.

IV. THERAPY

A. Prophylaxis

Close medical supervision in hospital is indicated for all known or possibly exposed individuals. Induced vomiting, gastric lavage, and purgation are recommended to facilitate elimination of unabsorbed toxin. Because of the serious risk of anaphylaxis and serum sickness whenever horse serum is given, the decision to administer antitoxin to asymptomatic individuals should be weighed very carefully. Each situation should be considered separately.

B. Treatment of Cases

Recent studies substantiate the efficacy of antitoxin, especially type E antitoxin, if administered early in the illness (Dolman and Iida, 1963). The sooner antiserum is given the better the prognosis; however, it may also be beneficial if administered as late as several days after toxin ingestion, since circulating toxin has been detected in serum as late as 3½ weeks after consumption of contaminated food (Koenig et al., 1964 and 1967; Ager and Dolman, 1964; Dolman, 1961). Equally important is prompt symptomatic treatment. All patients must be kept under close medical supervision. Early tracheostomy should be performed in patients with respiratory impairment (Rogers, 1964; Koenig et al., 1964). The use of cathartics, high enemas, and gastric lavage are recommended to eliminate residual toxin (Koenig et al., 1964).

Although its efficacy is unproven, penicillin is recommended by some because of the theoretical possibility that toxin may be released in vivo following the germination of spores. While not universally accepted, this concept of pathogenesis, is supported by laboratory evidence (Coleman and Meyer, 1922). Antibiotic drugs should be used for the treatment of infectious complications such as respiratory and urinary tract infections.

C. Therapeutic Preparations

Because types A and B as well as E toxins can contaminate marine products and because plant products can be contaminated with type E, patients with illness diagnosed as botulism should immediately receive A, B, and E antitoxins until laboratory tests determine which toxin is responsible (see Section II, E). Monovalent E and bivalent AB antitoxins should be reserved for use after these specific toxins have been identified.

Four preparations are currently available. All are of equine origin. 1) Trivalent A, B, E (Connaught*), available from NCDC, should be given in cases of botulism when the toxin type is unknown, regardless of the vehicle. 2) Monovalent E (Connaught), distributed by NCDC, should be reserved for outbreaks known to be caused by type E toxin. 3) Bivalent AB (Lederle) is indicated when either type A or type B is incriminated; it is available commercially. 4) Polyvalent ABEF (Serum Institute of Denmark) is reserved exclusively for type F outbreaks and is available only from NCDC.

These preparations can be obtained on a 24-hour basis from NCDC (see I. Emergency Assistance). Lederle's preparation, also available on an emergency basis can be obtained directly from the manufacturer by calling any of the following numbers:

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* Names of manufacturers and trade names are provided for identification only, and inclusion does not imply endorsement by the Public Health Service or the U.S. Department of Health, Education, and Welfare.

City	Area	Phone	City	Area	Phone
Atlanta	404	457-0261	Los Angeles	213	723-6411
Boston	617	782-6000	Minneapolis	612	935-1721
Chicago	312	827-8871	New Orleans	504	831-1301
Cincinnati	513	771-5400	Pearl River, N. Y.	914	735-5000
Dallas	214	631-2130	New York City	212	562-7000
Denver	303	377-2773	Philadelphia	215	646-7000
Kansas City	816	363-3305	Portland, Oreg.	503	228-6281
			St. Louis	314	664-5306

Table 7 compares these preparations and gives recommended dosages. A pentavalent toxoid is available from NCDC for active immunization of laboratory workers who are at high risk.

All suspect cases of human botulism should be reported immediately to health authorities.

V. LABORATORY PROCEDURES

Since botulinum toxin is one of the most poisonous substances known, all materials suspected of being contaminated with toxins should be handled with maximum precaution. Liquids should never be pipetted by mouth; Pro-Pipettes or other suitable safety pipettes should be used. Laboratory workers who might routinely be exposed to toxins should be actively immunized with botulinum toxoid.

A. Collection and Shipment of Samples

Suspect foods should be refrigerated, preferably not frozen, and examined as quickly as possible after collection. Food in sealed containers should be kept sealed in the original container whenever possible. If the food must be transferred to other containers, they should be sterile.

Specimens should be placed in a leak-proof container, packed with ice in a second leak-proof, insulated shipping container and shipped by the most rapid means possible. The recipient laboratory should be notified in advance as to when and how specimens are being shipped, when they should arrive, and the waybill or shipping number.

Body fluids and tissues: Serum, gastric contents, and autopsy specimens should be packed in suitable sterile containers, rapidly frozen to inactivate enzymes, and maintained frozen until examined. All specimens should be carefully labeled to allow prompt identification in the laboratory.

B. Identification of *C. botulinum* and Its Toxins

The following procedures are suggested to detect *C. botulinum* and botulinum toxins in food:

1. Preparation of food extract
   a. Record all information sent with food sample.
   b. If canned foods are to be tested, place the can in a large plastic bag to prevent aerosolization and wipe the top of the can with a 1:1 mixture of 10 percent Roccal (Winthrop) and 70 percent isopropyl alcohol before opening. Use a separate sterile can opener for each can.
   c. Record the condition of food (gassy, dark, putrid, etc.).
   d. Grind food in the following manner:
      (1) Place food in a sterile, chilled, pre-weighed mortar. Calculate the weight of the food sample and record. Use a 50 g sample if possible.
      (2) Add 1 to 2 g of sterile sand.
      (3) Add a small amount (approximately 5 ml) of cold gelatin diluent* and grind with a sterile pestle until a homogeneous suspension is obtained. If the food is extremely dry, add more gelatin diluent.
   e. After grinding, add a volume (ml) of diluent equal to the weight (g) of the food sample, cover the suspension, and place in the refrigerator at 4°C for 12 to 18 hours.

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* 0.2 percent gelatin, 0.4 percent Na_2HPO_4 in distilled water adjusted to pH 6.2 with hydrochloric acid and sterilized by autoclaving at 120°C for 15 minutes.

2. Culture of food sample

a. Treat part of the food sample with alcohol, as follows:

(1) Using a safety Pro-Pipette, put approximately 0.5 ml of the food suspension in a 13 x 100 mm sterile screw-cap tube.

(2) Add an equal volume of 100 percent ethanol; incubate at room temperature for 1 hour, mixing every 15 minutes. The alcohol treatment kills vegetative cells, but leaves spores viable.

b. Heat 5 tubes of chopped-meat medium containing 0.3 percent glucose and 0.2 percent soluble starch in boiling water for 10 minutes. Transfer 3 tubes to a 70°C waterbath, and cool the other 2 tubes in cold water.

c. Inoculate one of the cooled tubes with the alcohol-treated food and the other with untreated food. Introduce 0.5 to 1.0 ml of inoculum near the bottom of the tubes with a capillary pipette. Try to avoid introducing air bubbles into the medium.

d. Leave the 3 tubes of medium in the 70°C waterbath for about 10 minutes, then inoculate all 3 tubes of medium with the food suspension. After 10 minutes, remove 1 tube to cold water and transfer the other 2 tubes to an 80°C waterbath. After 10 minutes at 80°C, cool 1 tube and transfer the other to a boiling waterbath for an additional 10 minutes before cooling.

e. Incubate the 5 tubes in an anaerobic jar at a temperature of 30°C. (Some types of C. botulinum produce little or no toxin at temperatures above 30°C.) Maximum toxin production usually occurs after 3 to 5 days' incubation.

f. To isolate C. botulinum in pure culture, inoculate by streaking on suitable agar media, such as blood agar or egg yolk agar. Incubate plates in an anaerobic jar at 35° to 37°C. After incubation, pick isolated colonies and inoculate tubes of chopped-meat-dextrose-starch medium; incubate at 30°C. Establish the identity of pure cultures by conventional cultural and biochemical procedures. Establish the toxin type by the mouse neutralization test described in the following section.

3. Identification of toxin in food or culture by mouse neutralization test

a. Transfer either the liquid from the cultures or the food suspension described in Section 1, part e, to plastic centrifuge tubes. Centrifuge at 10,000 RPM for 10 minutes (preferably in a refrigerated centrifuge). Remove supernatant fluid for testing. A second centrifugation may be necessary for clarification.

b. Because the toxicity of the toxins of type E and of some type F and type B isolates of C. botulinum is greatly increased by the addition of trypsin, the test should be performed with trypsinized as well as untrypsinized material (Duff et al., 1956). Mix 9 parts of the food extract or culture fluid with 1 part of trypsin solution (1 gram Difco 2:250 trypsin diluted to 100 ml with distilled water): Check the pH of the mixture, adjust to pH 6.0-6.2 and incubate at 37°C for 45 minutes.

c. Reconstitute the respective C. botulinum diagnostic antitoxins as directed by the manufacturer. For the antitoxins prepared at NCDC, instructions are printed on vial labels. Dilute the antitoxin so that each 0.1 ml contains 1 international unit. In the neutralization test, each mouse will receive 1 international unit of antitoxin. One unit of antitoxin neutralizes 10,000 mouse IP LD_{50} doses of types A, B, C, D, and F, or 1,000 mouse IP LD_{50} doses of type E toxin.

d. The following procedure is designed to allow the detection of all types of botulinum toxins (A-F). If not enough material is available to carry out the complete procedure as outlined, modify it as circumstances warrant by deleting some test mixtures. If necessary, use polyvalent mixtures of antitoxin for preliminary screening, and then repeat the test with specific antitoxins, as required.

e. Because botulinum toxins are heat labile, a portion of the food extract or culture fluid should be heated at 100°C for 10 minutes to serve as a control.

f. For neutralization tests, mix 1.2 ml of the extract or trypsinized extract with 0.3 ml of the specific botulinum antitoxin or with 0.3 ml normal rabbit serum (NRS) for the controls, as outlined below.

g. Label 13 (15 x 85 mm) tubes 1-13 and prepare the various mixtures as shown in the following table:

Tube Number	Extract or Culture Fluid*	Antitoxin or Normal Rabbit Serum (NRS)	
1	1.2 ml	0.3 ml	NRS
2	1.2 ml (heated 100°C, 10 min.)	0.3 ml	NRS
3	1.2 ml	0.3 ml	Anti A
4	1.2 ml	0.3 ml	Anti B
5	1.2 ml	0.3 ml	Anti C
6	1.2 ml	0.3 ml	Anti D
7	1.2 ml	0.3 ml	Anti E
8	1.2 ml	0.3 ml	Anti F
9	1.2 ml trypsinized	0.3 ml	NRS
10	1.2 ml trypsinized (heated 100°C, 10 min.)	0.3 ml	NRS
11	1.2 ml trypsinized	0.3 ml	Anti B
12	1.2 ml trypsinized	0.3 ml	Anti E
13	1.2 ml trypsinized	0.3 ml	Anti F

* Certain cultures of C. botulinum may produce more toxin than will be neutralized by the quantity of antitoxin used in this procedure (1 unit); therefore, the tests should also be performed on diluted culture fluid, e.g., 10^{-1}, 10^{-2}, etc.

h. After preparing the test mixtures, incubate the tubes in a 37°C waterbath for 30 minutes and inject two 15 to 20 gram mice intraperitoneally with each test mixture (0.5 ml per mouse).

Although botulism intoxication usually kills mice within 6 to 24 hours, delayed deaths are occasionally observed. If toxin is present in sufficient quantities to be detected under test conditions, the unheated mixtures will kill all mice except those receiving specific antitoxin. Mice that receive heated mixtures (100°C) should survive.

NOTE: Botulism signs in mice begin with ruffling of the fur, followed by labored abdominal breathing, then weakness of the limbs, and finally, total paralysis. Death is caused by respiratory failure. The time between the first sign of distress and death varies greatly. Death, without clinical signs, is not adequate evidence that botulinum toxin was present.

4. Demonstration of toxin in blood serum

Obtain blood samples before antitoxin is given, as soon as possible after the onset of symptoms. Additional specimens are often helpful at intervals during the acute and convalescent stages of the illness. Collect enough blood to provide at least 10 ml of serum* for mouse toxicity tests. To detect toxin in serum, prepare mixtures as follows:

Tube Number	Patient's Serum	Serum or Antitoxin	Treatment
1	1.2 ml	0.3 ml NRS	
2	1.2 ml	0.3 ml Anti A	Incubate
3	1.2 ml	0.3 ml Anti B	mixtures at
4	1.2 ml	0.3 ml Anti C	37°C for
5	1.2 ml	0.3 ml Anti D	30 minutes.
6	1.2 ml	0.3 ml Anti E	
7	1.2 ml	0.3 ml Anti F	

a. Inoculate two mice intraperitoneally with each test mixture, using 0.5 ml per mouse. Do not inject more than 1.0 ml per mouse (I.P.), since excessive amounts of normal human serum can cause death (Koenig et al., 1964, and Rogers et al., 1964). Trypsinization of serum is not necessary for activation of toxin.

b. If toxin is present in sufficient quantities to be detected, all mice will die except those receiving specific antitoxin.

c. In cases where the quantity of patient's serum is limited, inoculate two mice with a mixture of patient's serum plus normal rabbit serum (prepared as shown above) and inoculate two other mice with a mixture of polyvalent antitoxin (ABEF) mixed in the same proportions with the patient's serum; repeat with type specific botulinum antitoxins if necessary.

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* Acute and convalescent serum should also be frozen and held for other studies in case the illness is not botulism.

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TABLES AND FIGURES

TABLE 1
Botulism in the United States, 1899-1967
(outbreaks/cases/deaths)

State	A	B	E	F	A&B	Subtotal toxin specified	Subtotal toxin unknown	Grand total
Alabama	1/ 3/ 2		1/ 3/ 0			2/ 6/ 2	5/ 5/ 1	7/ 11/ 3
Alaska			8/17/ 9			8/ 17/ 9	1/ 7/ 1	9/ 24/ 10
Arizona	1/ 4/ 2	1/ 5/ 5				2/ 9/ 7	2/ 2/ 0	4/ 11/ 7
Arkansas							2/ 3/ 0	2/ 3/ 0
California	60/157/ 83	5/ 15/ 8	1/ 3/ 1	1/3/0	2/6/2	69/184/ 94	149/115/177	218/499/273
Colorado	11/ 35/ 19	1/ 5/ 1				12/ 40/ 20	32/ 90/ 55	44/130/ 75
Connecticut		1/ 2/ 1				1/ 2/ 1	2/ 3/ 2	3/ 5/ 3
Delaware							0/ 0/ 0	0/ 0/ 0
Florida							2/ 8/ 7	2/ 8/ 7
Georgia							2/ 2/ 1	2/ 2/ 1
Hawaii							0/ 0/ 0	0/ 0/ 0
Idaho	3/ 6/ 6					3/ 6/ 6	7/ 20/ 9	10/ 26/ 15
Illinois		1/ 2/ 1	1/ 3/ 1			2/ 5/ 2	10/ 14/ 6	12/ 19/ 8
Indiana	2/ 4/ 2	1/ 7/ 4				3/ 11/ 6	2/ 11/ 4	5/ 22/ 10
Iowa							1/ 5/ 3	1/ 5/ 3
Kansas	1/ 7/ 1					1/ 7/ 1	0/ 0/ 0	1/ 7/ 1
Kentucky		3/ 11/ 1	1/ 2/ 0*			4/ 13/ 1	16/ 43/ 13	20/ 56/ 14
Louisiana							4/ 4/ 0	4/ 4/ 0
Maine							1/ 4/ 4	1/ 4/ 4
Maryland		2/ 7/ 1				2/ 7/ 1	5/ 7/ 3	7/ 14/ 4
Massachusetts							5/ 9/ 7	5/ 9/ 7
Michigan	3/ 41/ 10		2/ 5/ 4			5/ 46/ 14	5/ 18/ 8	10/ 64/ 22
Minnesota			1/ 2/ 2			1/ 2/ 2	4/ 12/ 4	5/ 13/ 6
Mississippi							3/ 21/ 4	3/ 21/ 4
Missouri	1/ 1/ 0					1/ 1/ 0	1/ 1/ 1	2/ 2/ 1
Montana	4/ 15/ 13					4/ 15/ 13	10/ 20/ 11	14/ 35/ 24
Nebraska	2/ 5/ 5					2/ 5/ 5	7/ 22/ 16	9/ 27/ 21
Nevada							3/ 6/ 5	3/ 6/ 5
New Hampshire							0/ 0/ 0	0/ 0/ 0
New Jersey							9/ 30/ 12	9/ 30/ 12
New Mexico	11/ 54/ 38	1/ 4/ 3				12/ 58/ 41	16/ 23/ 13	28/ 81/ 54
New York	1/ 2/ 0	10/ 27/ 14	2/ 6/ 2			13/ 35/ 16	17/ 42/ 34	30/ 77/ 40
North Carolina							3/ 3/ 0	3/ 3/ 0
North Dakota	1/ 13/ 13					1/ 13/ 13	5/ 18/ 11	6/ 31/ 24
Ohio	2/ 16/ 9					2/ 16/ 9	7/ 17/ 10	9/ 33/ 19
Oklahoma							5/ 6/ 2	5/ 6/ 2
Oregon	10/ 27/ 22					10/ 27/ 22	18/ 29/ 19	28/ 56/ 41
Pennsylvania	1/ 5/ 3	1/ 3/ 0				2/ 8/ 3	5/ 10/ 5	7/ 18/ 8

TABLE 1 (continued)
Botulism in the United States, 1899-1967
(outbreaks/cases/deaths)

State	A	B	E	F	A&B	Subtotal toxin specified	Subtotal toxin unknown	Grand total
Rhode Island							0/ 0/ 0	0/ 0/ 0
South Carolina							0/ 0/ 0	0/ 0/ 0
South Dakota	1/ 1/ 1					1/ 1/ 1	2/ 6/ 6	3/ 7/ 7
Tennessee	1/ 7/ 7	2/ 4/ 3	1/12/ 5*			4/ 23/ 15	9/ 25/ 16	13/ 48/ 31
Texas							4/ 11/ 4	4/ 11/ 4
Utah	3/ 10/ 4					3/ 10/ 4	2/ 4/ 2	5/ 14/ 6
Vermont							0/ 0/ 0	0/ 0/ 0
Virginia		1/ 5/ 4				1/ 5/ 4	2/ 4/ 2	3/ 9/ 6
Washington	16/ 34/ 28	2/ 19/ 8	1/ 4/1			19/ 57/ 37	53/ 119/ 76	72/ 176/113
West Virginia		1/ 1/ 1				1/ 1/ 1	1/ 3/ 2	2/ 4/ 3
Wisconsin							3/ 6/ 3	3/ 6/ 3
Wyoming	3/ 16/ 10	1/ 4/ 4				4/ 20/ 14	4/ 9/ 4	8/ 29/ 18
District of Columbia							1/ 3/ 1	1/ 3/ 1
Other and unspecified							0/ 0/ 28	0/ 0/ 28
Adjustment			-2*			-2*		-2*
Totals specified	139/463/278	34/121/59	17/57/25	1/ 3/ 0	2/ 6/ 2	193/650/364	447/1019/584	640/1669/948

*One outbreak occurred in three states and was counted three times.

TABLE 2

Botulism Cases and Deaths, by Toxin Types, 1899-1967.

	YEARS								
	1899	1900-1909	1910-1919	1920-1929	1930-1939	1940-1949	1950-1959	1960-1967	Total
Toxin type A									
cases	0	0	44	156	94	110	39	20	463
deaths	0	0	31	94	69	62	18	4	278
death/case percent	-	-	70.5	60.0	73.4	56.4	46.2	20.0	60.0
Toxin type B									
cases	0	0	10	33	33	22	4	19	121
deaths	0	0	7	20	16	12	2	2	59
death/case percent	-	-	70.0	60.7	48.5	54.6	50.0	10.5	48.7
Toxin type E									
cases	0	0	0	0	6	3	14	34	57
deaths	0	0	0	0	2	1	7	15	25
death/case percent	-	-	-	-	33.3	33.3	50.0	44.1	43.9
Toxin type F									
cases	0	0	0	0	0	0	0	3	3
deaths	0	0	0	0	0	0	0	0	0
death/case percent	-	-	-	-	-	-	-	0	0
Mixed toxins A&B									
cases	0	0	0	1	5	0	0	0	6
deaths	0	0	0	1	1	0	0	0	2
death/case percent	-	-	-	100.0	20.0	-	-	-	33.3
Subtotal, toxin type known									
cases	0	0	54	190	138	135	57	76	650
deaths	0	0	38	115	88	75	27	21	364
death/case percent	-	-	70.4	60.5	63.8	55.6	47.4	27.6	56.0
Subtotal, toxin type unknown									
All other cases	1	10	189	138	245	181	176	79	1019
All other deaths	0	6	135	92	162	119	58	12	584
death/case percent	0	60.0	71.4	66.7	66.1	65.7	32.9	15.2	57.4
Total									
cases	1	10	243	328	383	316	233	155	1669
death	0	6	173	207	250	194	85	33	948
death/case percent	0	60.0	71.2	63.1	65.3	61.4	36.5	21.2	56.8
Percent unknown toxin type		100.0	77.8	42.4	63.8	57.6	75.6	51.0	

TABLE 3

Outbreaks of Botulism Attributed to Commercially Processed or Home Processed Foods, 1899-1967

Source of food	1899	1900-1909	1910-1919	1920-1929	1930-1939	1940-1949	1950-1959	1960-1967	Total
Home processed	1	1	48	77	135	120	50	31	463
Commercially processed	0	1	14	26	6	1	3	9	60
Unknown	0	0	8	13	13	13	50	20	117
Total	1	2	70	116	154	134	103	60	640

TABLE 4

Food Products Causing Botulism Outbreaks[1] 1899-1967

Botulinum toxin type	Vegetables	Fruits	Beef(2)	Pork	Poultry	Fish and fish products	Milk and milk products	Condiments(3)	Other(4)	Total
A	90	22	3	2		5	2	12	3	139
B	21	4	1	1	1	2	2	2		34
E	1					16				17
F			1							1
A&B	2									2
Total	114	26	5	3	1	23	4	14	3	193

1. Includes only outbreaks in which the toxin type was determined.
2. Includes one outbreak of type F in venison, and one outbreak of type A in mutton.
3. Includes outbreaks traced to tomato relish, chili peppers, and salad dressing.
4. Includes outbreaks traced to relish and to corn and chicken mash.

TABLE 5

Outbreaks of Botulism in Which One or More Persons was Affected by a Given Symptom or Sign in 56 Outbreaks Reported to NCDC, 1953 - 1967

	Type A	Type B	Type E	Type F	Und.*	Total
Outbreaks	15	6	7	1	27	56
Cases	42	17	31	3	52	145
Symptoms						
1. Blurred vision, diplopia, photophobia	13	4	6	1	26	50
2. Dysphagia	8	5	2		19	34
3. Dysphonia	7	4	3		12	26
4. Generalized weakness	6	3	3		11	23
5. Nausea and/or vomiting	6	6	7	1	10	30
6. Dizziness or vertigo	1	2	4		6	13
7. Abdominal pain, cramps, fullness	1	2	2		4	9
8. Diarrhea	1	2			2	5
9. Constipation				1	1	2
10. Difficulty with urination			1		1	2
11. Paresthesias	1					1
Signs						
1. Respiratory impairment	8	3	5		13	29
2. Eye muscle involvement, including ptosis	1	2	1	1	5	10
3. Dilated, fixed pupils	2	1	1		3	7
4. Specific muscle weakness or paralysis	3	1	1		3	8
5. Dry throat, mouth, or tongue	2	2	1		2	7
6. Ataxia	3			1	3	7
7. Somnolence			1			1
8. Nystagmus	1					1
9. Postural Hypotension					1	1

*Toxin type undetermined or unspecified

TABLE 6

NCDC Experience in Investigation of Suspect Botulism Outbreaks

1964-1967; Final Diagnosis after Investigation

Botulism	18
Staphylococcal food poisoning	9
Chemical food poisoning	3
Carbon monoxide poisoning	3
Guillain-Barre Syndrome	3
Cerebral vascular accident	2
Shigella or salmonella gastroenteritis	1
Hyperventilation syndrome	1
Neuropsychiatric disorder	1
C. perfringens gangrene confused with botulism	1
No illness but concerned about possibility of botulism	4
Not botulism, but no final diagnosis made	7
Total Investigations	53

TABLE 7

Types of Antitoxins Available

Type	Manufacturer	Remarks	ml/vial	Potency International units/vial	Manufacturer's recommended dosage
ABE	Connaught	Distributed by NCDC	8 ml	A 7,500 B 5,500 E 8,500	Contents of one vial i.v. + one vial i.m. repeat in 2-4 hours if symptoms persist
AB	Lederle	Distributed by Lederle. Small quantities stored by NCDC	30 ml	A 10,000 B 10,000	Contents of one vial i.v. and "repeated at 4-hour intervals until toxic condition alleviated"
E	Connaught	Distributed by NCDC	1 ml	E 5,000	Contents of one vial given i.v. and one vial i.m. repeat in 2-4 hours if symptoms persist
ABEF	State Serum Institute, Denmark	Unlicensed. Stored by NCDC and reserved for use in type F outbreaks	20 ml	Variable	40 ml (2 vials)

Figure 1

CASES OF BOTULISM, BY TYPE IN 10-YEAR PERIODS
1899-1967

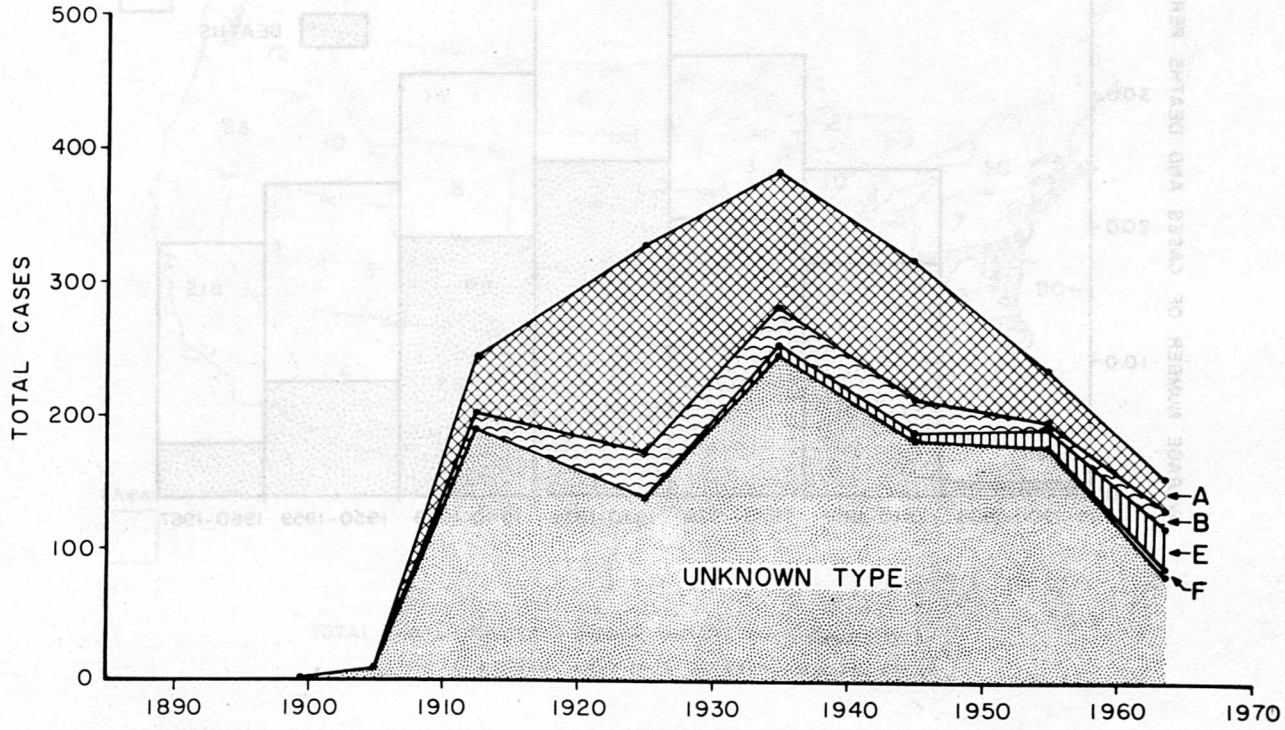


Figure 2

BOTULISM DEATH-TO-CASE RATIOS, BY 10-YEAR PERIODS
1899-1967

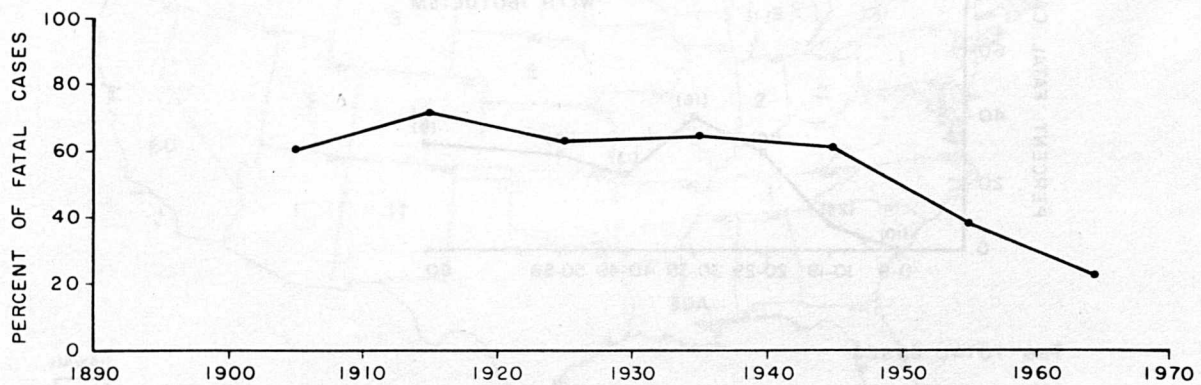


Figure 3

CASES AND DEATHS DUE TO BOTULISM, BY 10-YEAR PERIODS
1899-1967

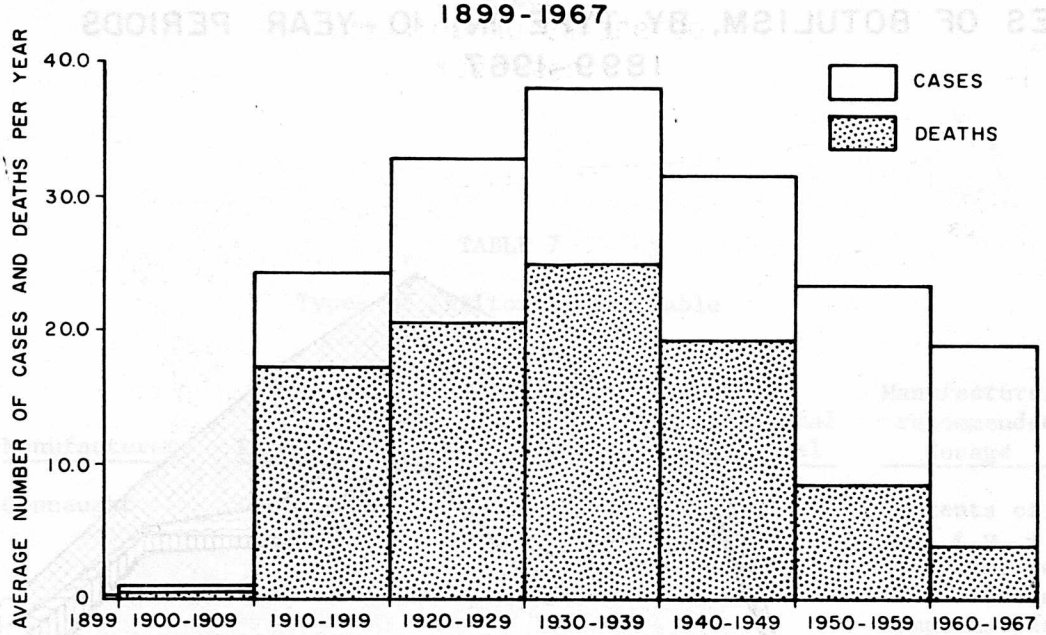
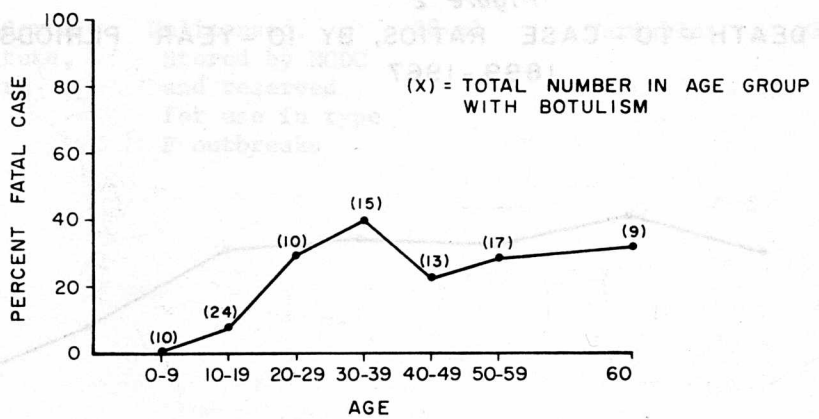


Figure 4

AGE SPECIFIC BOTULISM CASE* FATALITY RATES
1962-1967

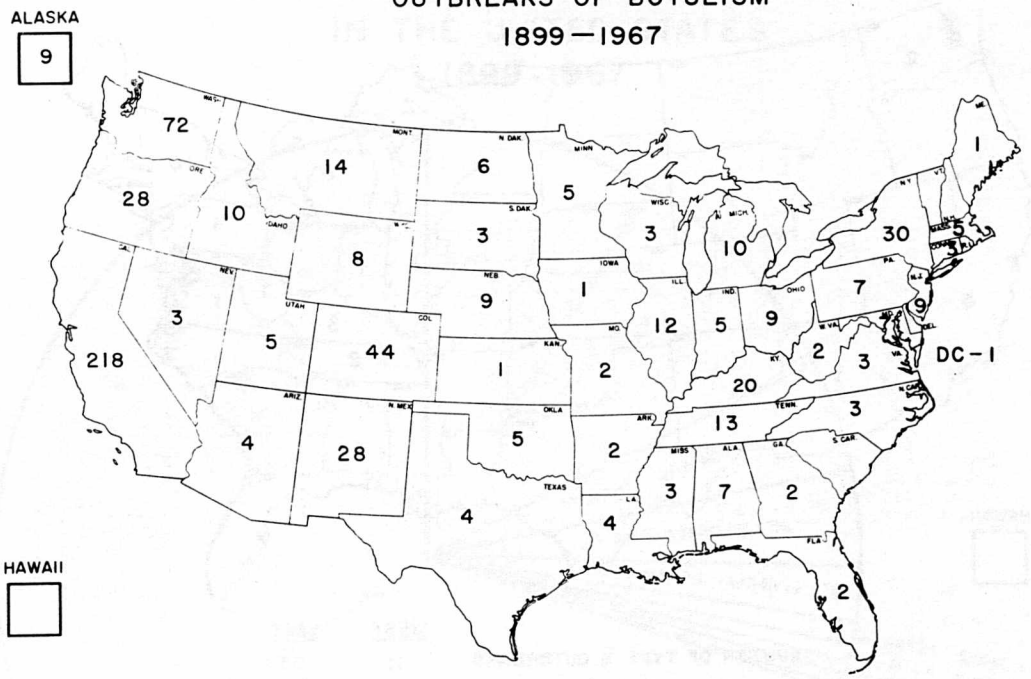


*98 TOTAL CASES

Figure 5

OUTBREAKS* OF BOTULISM

1899—1967

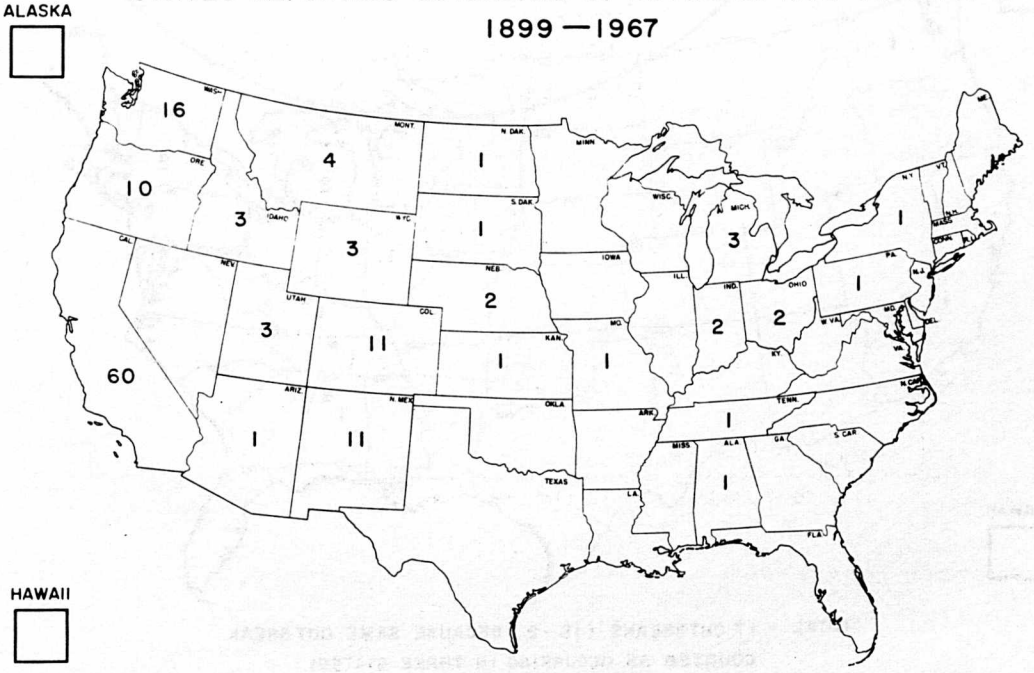


* TOTAL = 640 OUTBREAKS (ONE OUTBREAK IS COUNTED AS OCCURRING IN THREE STATES; SEE FIGURE 8)

Figure 6

STATES REPORTING OUTBREAKS OF BOTULISM DUE TO TYPE A

1899—1967

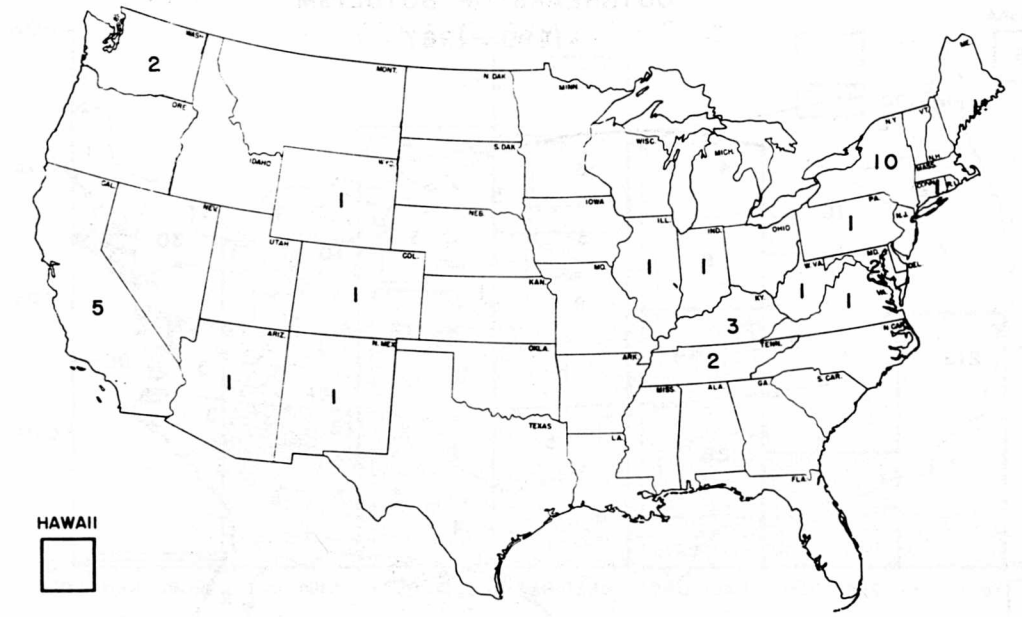


	WEST	EAST
NUMBER OF TYPE A OUTBREAKS	128	11

TOTAL = 139

Figure 7

STATES REPORTING OUTBREAKS OF BOTULISM DUE TO TYPE B
1899—1967

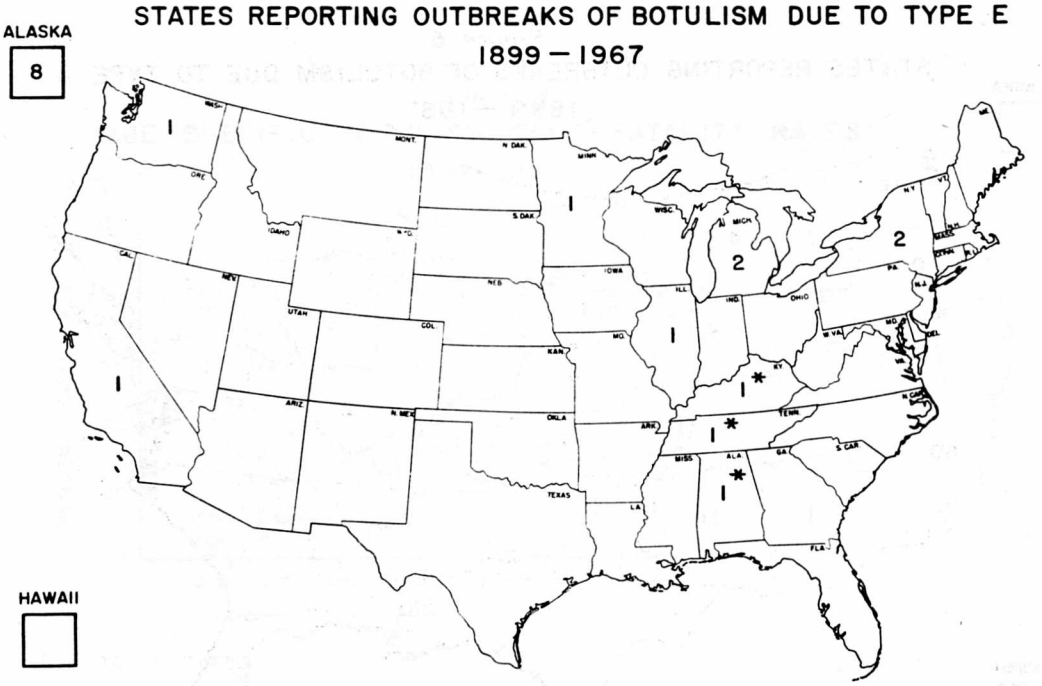


	WEST	EAST
NUMBER OF TYPE B OUTBREAKS	11	23

TOTAL = 34

Figure 8

STATES REPORTING OUTBREAKS OF BOTULISM DUE TO TYPE E
1899—1967

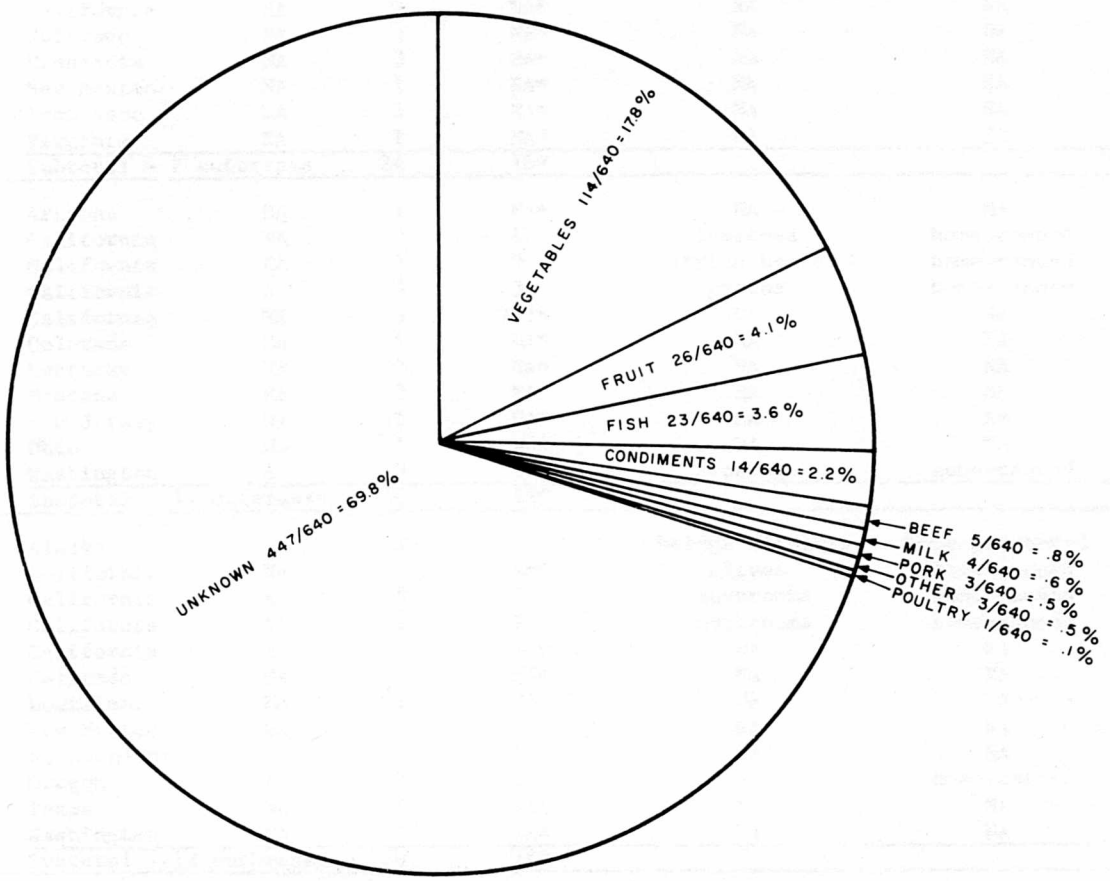


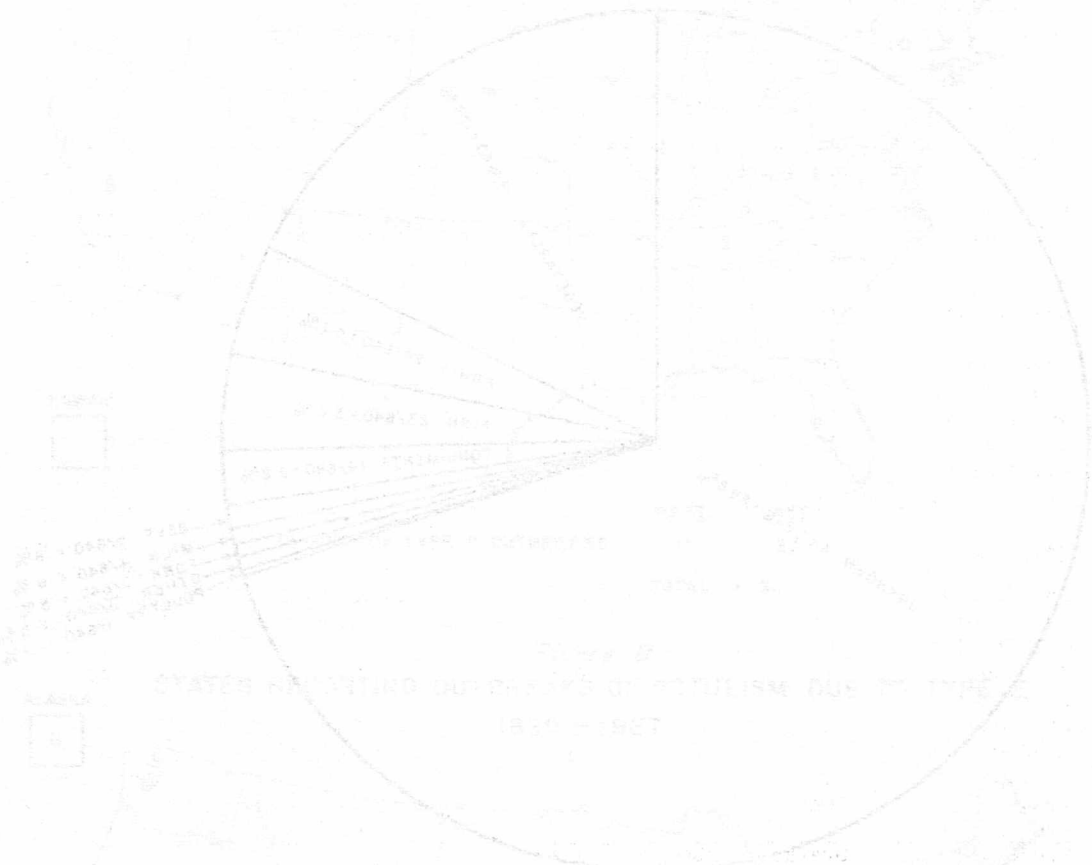
TOTAL = 17 OUTBREAKS (19−2, BECAUSE SAME OUTBREAK COUNTED AS OCCURRING IN THREE STATES)

*SAME OUTBREAK

Figure 9

FOODS INVOLVED IN BOTULISM OUTBREAKS
IN THE UNITED STATES
1899-1967





APPENDIX

BOTULISM OUTBREAKS 1950 - 1967*

Year	State	Toxin type	Cases	Deaths	Vehicle	Source
1950	Alaska	E	5	NA*	beluga	home-preserved
	California	NA	7	NA*	NA	NA
	Colorado	NA	3	NA*	NA	NA
	Minnesota	NA	3	NA*	NA	NA
	New Mexico	NA	1	NA*	NA	NA
	Tennessee	NA	4	NA*	NA	NA
	Virginia	NA	1	NA*	NA	NA
	Subtotal - 7 outbreaks		24	16*		
1951	Arizona	NA	1	NA*	NA	NA
	California	NA	2	1	tomatoes	home-canned
	California	NA	1	0	string beans	home-canned
	California	A	3	3	greens	home-canned
	California	NA	6	2+?*	NA	NA
	Colorado	NA	1	NA*	NA	NA
	Kentucky	NA	3	NA*	NA	NA
	Montana	NA	3	NA	NA	NA
	New Jersey	NA	12	NA*	NA	NA
	Ohio	NA	1	NA*	NA	NA
	Washington	A	3	2	asparagus	home-canned
	Subtotal - 11 outbreaks		36	12*		
1952	Alaska	E	1	1	beluga flippers	home-preserved
	California	NA	1	NA*	olives	home-canned
	California	A	3	2	mushrooms	home-canned
	California	NA	1	1	mushrooms	home-canned
	California	NA	5	NA*	NA	NA
	Colorado	NA	1	NA*	NA	NA
	Louisiana	NA	1	NA*	NA	NA
	New Mexico	NA	1	0	NA	NA
	N. Carolina	NA	1	NA*	NA	NA
	Oregon	A	2	2	beets	home-canned
	Texas	NA	1	NA*	NA	NA
	Washington	NA	2	NA*	NA	NA
	Subtotal - 12 outbreaks		20	14*		
1953	California	A	1	0	huckleberry juice	home-canned
	California	B	1	0	string beans	home-canned
	Colorado	NA	3	3	beets	home-canned
	Colorado	NA	3	0	NA	NA
	Illinois	NA	2	0	cheese	home-made
	Illinois	NA	1	1	corn	home-canned
	Illinois	NA	1	0	NA	NA
	Kentucky	NA	1	0	NA	NA
	Louisiana	NA	1	0	NA	NA
	N. Carolina	NA	1	0	frozen lobster tail	commercially processed
	Ohio	NA	1	0	NA	NA
	Oklahoma	NA	1	0	NA	NA
	Washington	NA	1	0	NA	NA
	Subtotal - 13 outbreaks		18	4		

* Data for 1950, 1951, and 1952 are incomplete. Reports of cases are derived from reports to NCDC and reports in Public Health Reports. The outcome of cases was not usually reported in these reports, so Vital Statistics Reports were used to estimate deaths. Data after 1952 are based entirely on reports to NCDC. Numbers of cases and deaths often exceed previously published data because additional reports have come to our attention.

Abbreviations: NA = Information not available.

Year	State	Toxin type	Cases	Deaths	Vehicle	Source
1954	California	A	4	2	peaches	home-canned
	California	A	2	0	okra	home-canned
	Colorado	NA	2	2	asparagus	home-canned
	Indiana	NA	2	0	NA	commercial
	Kentucky	NA	1	0	NA	NA
	Maryland	NA	1	0	NA	NA
	Nevada	NA	3	3	beets	home-canned
	Ohio	NA	1	0	NA	NA
	Oregon	A	1	0	beets	home-canned
	Washington	NA	2	1	NA	NA
	Subtotal - 10 outbreaks		19	8		
1955	Arizona	NA	1	0	NA	NA
	California	B	2	1	green olives	home-canned
	California	NA	1	0	NA	NA
	Colorado	A	5	0	chili peppers	home-canned
	Louisiana	NA	1	0	NA	NA
	New Mexico	A	4	4	spinach	home-canned
	New Mexico	NA	1	0	NA	NA
	Pennsylvania	NA	2	1	mushrooms	home-canned
	Subtotal - 8 outbreaks		17	6		
1956	Alaska	E	3	2	beluga	home-preserved
	Alaska	E	2	1	ougruk	home-preserved
	California	A	4	2	olives	home-canned
	California	NA	1	1	potatoes	home-canned
	California	NA	1	1	pickled pigs feet	home-canned
	California	NA	1	0	NA	home-canned
	Colorado	NA	6	1	beet greens	home-canned
	Illinois	NA	1	0	NA	NA
	Kentucky	NA	4	0	NA	NA
	Maryland	NA	2	2	NA	NA
	New York	NA	1	1	NA	NA
	New York	B	1	1	swiss chard	home-preserved
	Oklahoma	NA	1	0	NA	NA
	Subtotal - 13 outbreaks		28	12		
1957	California	A	5	0	tuna fish	home-canned
	Colorado	NA	1	0	NA	NA
	Kentucky	NA	1	0	green beans	home-canned
	Maryland	NA	2	1	string beans	home-canned
	Mississippi	NA	16	0	NA	NA
	New Jersey	NA	1	1	mushrooms	home-canned
	New Mexico	NA	1	0	sausage	home-made
	Ohio	NA	1	0	NA	NA
	Tennessee	NA	1	1	NA	NA
	Washington	NA	5	1	gluten	home-canned
	Subtotal - 10 outbreaks		34	4		
1958	California	NA	1	0	mushrooms	home-canned
	Kentucky	NA	2	0	beans	home-canned
	Kentucky	NA	1	0	NA	NA
	N. Carolina	NA	1	0	NA	NA
	Oklahoma	NA	1	0	NA	NA
	Washington	NA	1	0	NA	NA
	Subtotal - 6 outbreaks		7	0		

Year	State	Toxin type	Cases	Deaths	Vehicle	Source
1959	Alaska	E	2	3	fish eggs	home-preserved
	Alaska	E	7	1	seal or whale flippers	home-preserved
	Alaska	E	1	1	fish eggs	home-preserved
	California	NA	1	1	string beans	home-canned
	California	NA	1	0	mushrooms	home-canned
	California	A	1	0	corn and chicken mash	home-canned
	Colorado	NA	1	0	beans	home-canned
	Colorado	A	1	1	green beans	home-canned
	Idaho	NA	6	3	beets	home-canned
	Illinois	NA	1	0	NA	NA
	Michigan	NA	4	0	beets	home-canned
	Mississippi	NA	1	0	NA	NA
	Washington	NA	2	0	NA	NA
	Subtotal - 13 outbreaks		29	9		
1960	Alaska	E	2	2	salmon eggs	home-preserved
	Kentucky	NA	2	0	beets	home-canned
	Michigan	NA	1	1	green beans	home-canned
	Michigan	NA	3	0	beets	home-canned
	Minnesota	E	2	2	smoked fish	commercially processed
	Minnesota	NA	2	0	frozen chicken pie	commercially processed
	Subtotal - 6 outbreaks		12	5		
1961	Arkansas	NA	1	0	NA	NA
	Florida	NA	1	0	NA	NA
	Idaho	NA	3	0	NA	NA
	Louisiana	NA	1	0	NA	NA
	Washington	E	4	1	salmon eggs	home-processed
	Washington	NA	4	1	chili	home-processed
	Subtotal - 6 outbreaks		14	2		
1962	Alabama	NA	1	0	NA	NA
	California	NA	2	0	NA	NA
	Colorado	NA	3	0	NA	NA
	Kentucky	NA	1	0	green beans	home-canned
	Massachusetts	NA	1	1	mushrooms	home-canned
	New Jersey	NA	2	1	red peppers	home-canned
	New Mexico	A	3	2	chili	home-canned
	Tennessee	NA	3	2	corn	home-canned
	Subtotal - 8 outbreaks		16	6		
1963	Alabama	E	3	0	smoked whitefish chubs	commercially packed
	Kentucky		2	0		
	Tennessee		12	5		
	California	NA	6	1	mushrooms	home-canned
	California	NA	2	1	figs	home-canned
	California	A	2	0	chili peppers	home-canned
	Colorado	A	2	1	green beans	home-canned
	Kentucky	B	5	1	corn	home-canned
	Michigan	E	2	2	whitefish	commercially processed
	Michigan	E	3	2	tuna fish	commercially-canned
	Minnesota	E	1	0	smoked whitefish	commercially processed
	New York	A	2	0	liver paste	commercially-canned
	Pennsylvania	B	3	0	string beans	home-canned
	W. Virginia	B	1	1	green beans	home-canned
	Subtotal - 12 outbreaks		46	14		

Year	State	Toxin type	Cases	Deaths	Vehicle	Source
1964	Alabama	NA	1	0	NA	NA
	California	NA	3	2	chili beans or green peppers	home-canned
	California	NA	4	0	peppers	home-canned
	Georgia	NA	1	0	NA	NA
	Kansas	A	7	1	pickles	home-canned
	Kentucky	NA	2	0	NA	home-processed
	Kentucky	B	4	0	green beans	home-canned
	Maryland	NA	1	0	NA	NA
	North Dakota	NA	1	0	NA	NA
	Washington	NA	4	1	beans	home-canned
	Subtotal - 10 outbreaks		28	4		
1965	Alabama	NA	1	0	tomato juice	home-canned
	Alabama	NA	1	0	NA	NA
	California	A	2	0	tuna	home-processed
	Idaho	NA	3	0	?luncheon meat	commercially processed
	Kentucky	NA	5	0	NA	NA
	Maryland	NA	1	0	NA	NA
	New Jersey	NA	1	0	NA	NA
	Washington	NA	6	0	NA	NA
	Subtotal - 8 outbreaks		20	0		
1966	California	F	3	0	venison jerky	home-made
	California	NA	1	0	NA	NA
	California	NA	1	0	NA	NA
	Indiana	A	2	0	beets	home-canned
	Maryland	B	3	0	?ham from Germany	commercially packed
	New York	B	1	0	mushrooms	home-canned
	Subtotal - 6 outbreaks		11	0		
1967	Alaska	E	1	0	seal flipper	home-prepared
	Colorado	NA	1	1	?green beans	home-canned
	Illinois	E	3	1	whitefish	commercial source but home-canned
	New York	B	2	0	peppers	home-canned
	Subtotal - 4 outbreaks		7	2		

1950-1967 Totals

Toxin type	Outbreaks	Cases	Deaths
A	21	59	21
B	10	23	4
E	15	56	24
F	1	3	0
NA	116	245	69
Total	163	386	118

DHEW,PHS,HSMHA,NCDC