

Botulism in Alaska

Surveillance

Botulism is a public health emergency and health care providers should report all suspected cases to the Alaska Division of Public Health, Section of Epidemiology. In the past, the Alaska Area Native Health Service of the Indian Health Service, the Arctic Investigations Program of the U.S. Centers for Disease Control and Prevention (CDC), and the Alaska Division of Public Health conducted epidemiologic investigations of all patients with possible botulism. Although early records contain less detail, results of the investigations have been collected and analyzed. Currently all cases are investigated by the Alaska Section of Epidemiology.

Cases

Case definitions of confirmed and probable botulism are provided in Table 2. Botulism cases may go undiagnosed, and therefore unreported, if a person either does not seek medical care or the diagnosis is not considered. The botulism cases summarized in this monograph represent all **confirmed** cases in Alaska from 1947 through 2010. Forty-nine **probable** cases of botulism (from 29 outbreaks), which also occurred between 1947 and 2010, were excluded from the current analysis. Data regarding probable cases from 1947–85 have been summarized;²⁷ probable cases from 1986–2010 have not.

There were suspected but no confirmed cases during 1947–9 (Figure 1). From 1950 to 2010, 141 confirmed outbreaks of foodborne botulism involving 283 persons were reported in Alaska. Almost half (66 of 141 or 47%) of the outbreaks were associated with more than one case, and nine outbreaks were associated with five or more cases. The largest outbreak, with nine cases, occurred in 1973; the next largest outbreak, with eight cases, occurred in 2002.

All documented cases of foodborne botulism have occurred in Alaska Natives. The average annual incidence among Alaska Natives increased from 3.5 cases per 100,000 population during 1950–4 to a peak rate of 12.6 cases per 100,000 during 1985–9 (Table 3). Reasons for the increase are unclear but may relate to changes in food preparation practices or improved recognition of mild cases. Since 1989, rates have slowly declined to reach 4.4 cases per 100,000 in 2005–10. The reasons for this declining trend are also not clear.

There have been a total of 20 deaths for an overall case fatality rate of 7%. The case fatality rate declined from 31% during 1950–9 to 2% for the past 11 year period, 2000–10. A review of 14 fatal cases from 1970–2007 revealed that an initial diagnosis other than botulism (therefore resulting in no administration of antitoxin or mechanical ventilation) and botulism due to toxin type A were significant predictors of death.²⁸

Table 2. Case definitions of confirmed and probable botulism.

A **confirmed** case of botulism was any person in Alaska with a compatible illness having one or more of the symptoms listed in Table 7 (see page 12), and who met at least one of the following conditions:

1. The identification of botulinum toxin in an implicated food; or in serum, stool, gastric aspirate or vomitus collected from the person.
2. The isolation of *C. botulinum* organism from the person's stool or gastric aspirate/vomitus.
3. A history of eating the same implicated food as a person meeting one of the first two conditions.

A **probable** case of botulism was a person with a compatible illness following consumption of food frequently associated with botulism, but who did not meet any of the three above conditions.

An outbreak was the occurrence of botulism among one or more persons who had eaten a common food.

The mean age of persons with confirmed foodborne botulism has ranged from 35-50 years (Table 4). In general, more than 50% of cases were aged between 40 and 70 years, which is not surprising as previous research has shown that the proportion of persons who reported eating traditional foods increased with increasing age.²⁹

Figure 1. Botulism outbreaks, cases, and deaths, by year — Alaska, 1950 to 2010.

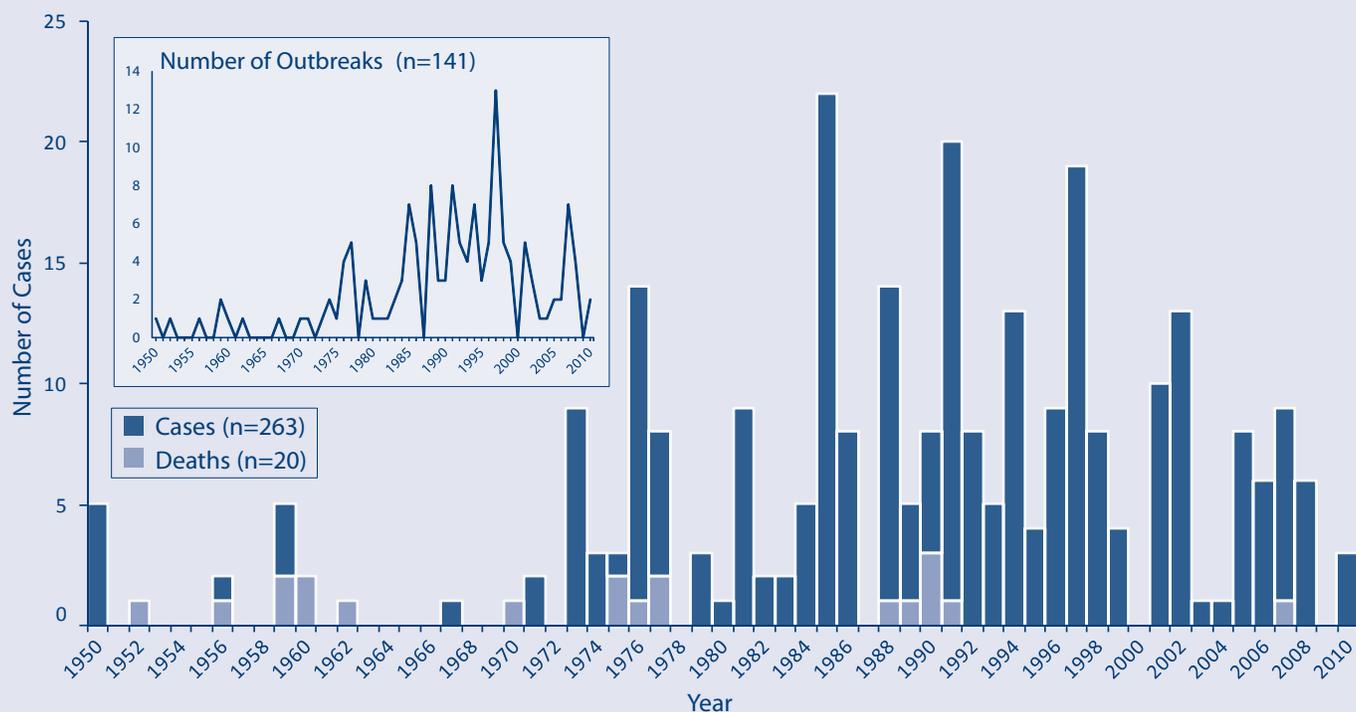


Table 3. Incidence, deaths, and case fatality rates by 5-year* intervals of confirmed botulism cases — Alaska, 1950 to 2010.

Intervals (Years)	Number	Incidence** of Cases	Number of Deaths	Case Fatality Rate
1950 – 1954	6	3.5	1	0.16
1955 – 1959	7	3.6	3	0.43
1960 – 1964	3	1.3	3	1.00
1965 – 1969	1	0.4	0	0.00
1970 – 1974	15	5.6	1	0.07
1975 – 1979	28	9.4	5	0.18
1980 – 1984	19	5.6	0	0.00
1985 – 1989	49	12.6	2	0.04
1990 – 1994	54	11.7	4	0.09
1995 – 1999	44	8.6	0	0.00
2000 – 2004	25	4.5 [†]	0	0.00
2005 – 2010*	32	4.4 [†]	1	0.03
1950 – 2010	283	5.9	20	0.07

*The final interval, 2005-2010, was 6 years.

**Annual incidence per 100,000 Alaska Natives. Mid-period population was used for each interval except 1950–2010, which was calculated by averaging the individual intervals.

[†]Beginning in 2000, persons could select multiple race designations. Rates calculated after 2000 are based on population bridged estimates.³⁰

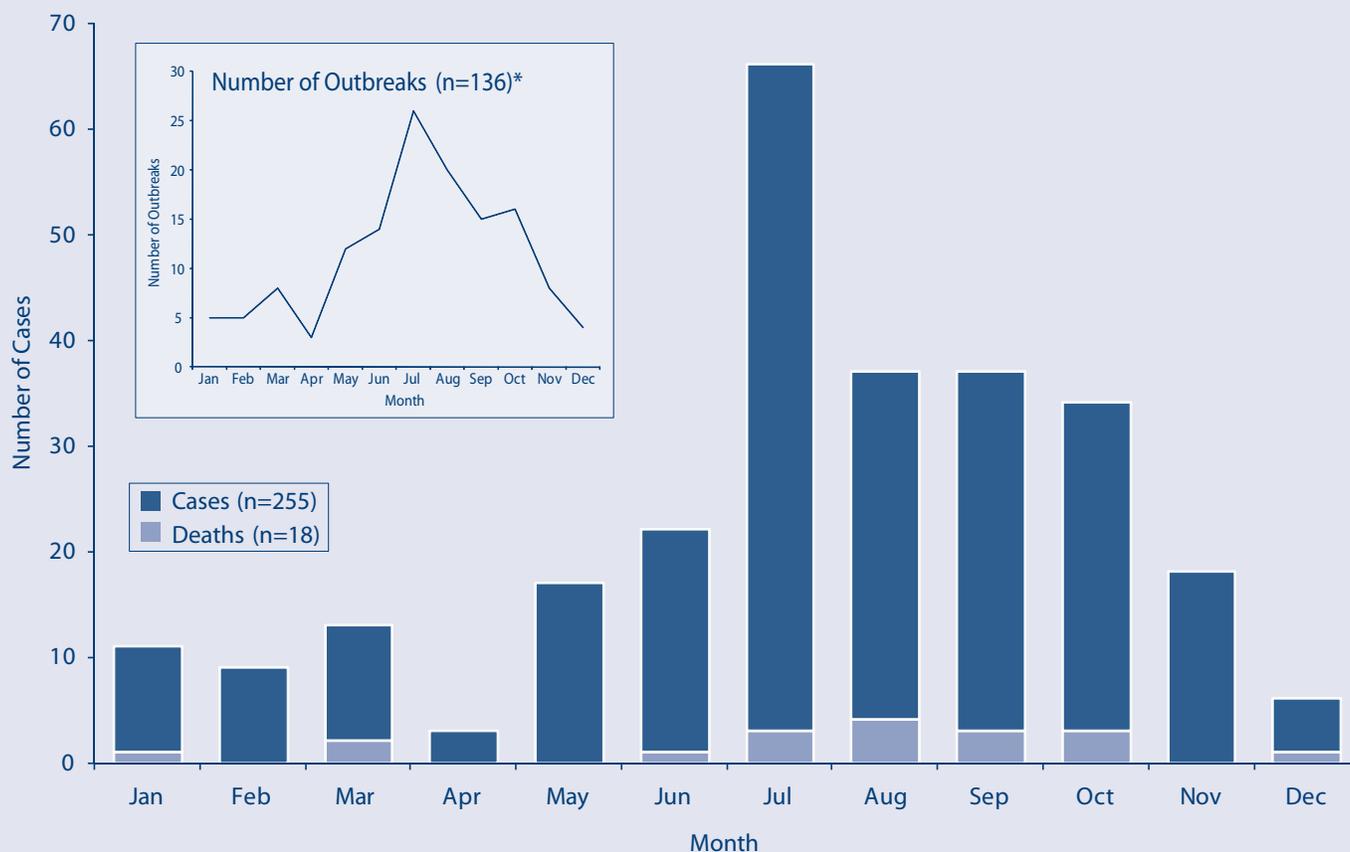
Table 4. Age of botulism cases by 10-year intervals* — Alaska, 1950 to 2010.

Intervals (Years)	Number of Cases	Median Age (Years)	Mean Age (Years)	Age Range (Years)
1950-1959	10**	30.5	35.0	8-63
1960-1969	4	45.5	50.0	39-70
1970-1979	43	34.5	36.8	6-63
1980-1989	68	44	43.7	5-77
1990-1999	98	44.5	46.7	8-93
2000-2010*	57	47	50.6	2-83

*The final interval, 2000-2010, was 11 years.

**Age not reported for three cases.

Figure 2. Botulism outbreaks, cases, and deaths, by month of onset — Alaska, 1950 to 2010.*



*Month of onset unknown for five outbreaks, corresponding to eight cases and two deaths.

Seasonality, types, and location

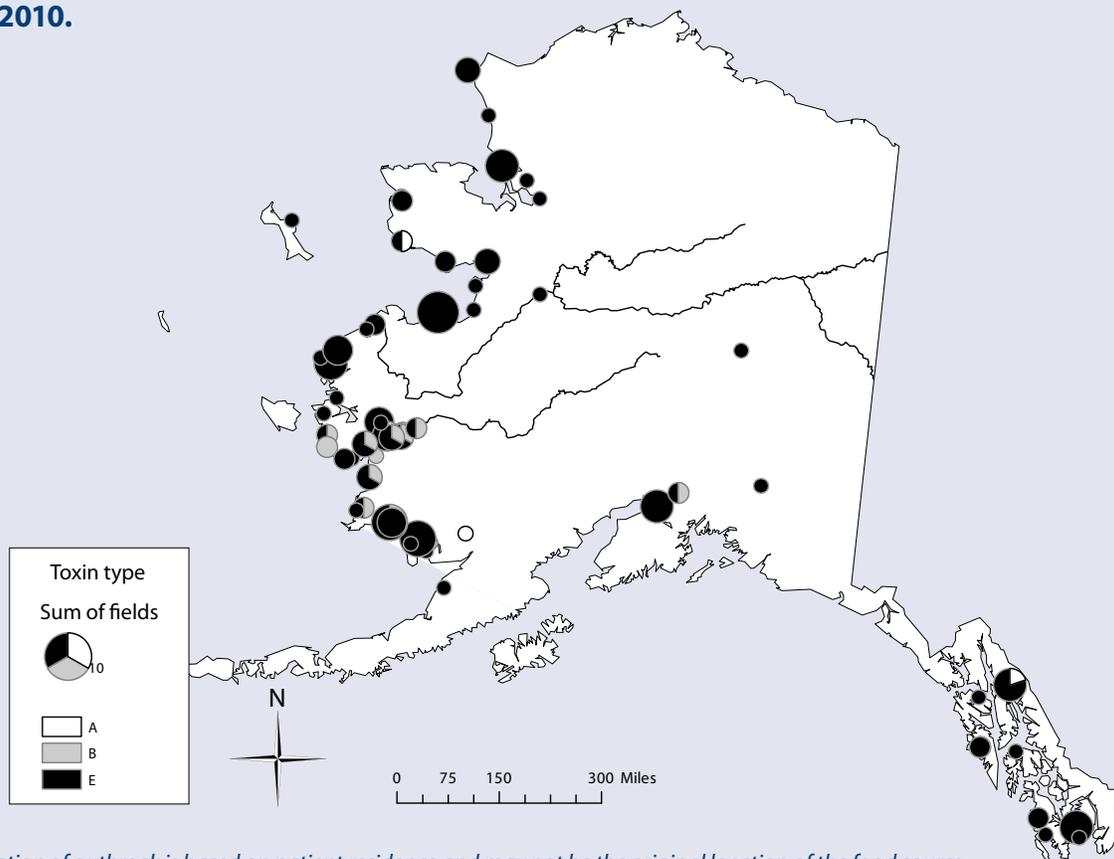
Although outbreaks have been reported in every month of the year, over half with a known date (76 of 136 or 56%) occurred between June and September (Figure 2). Dates recorded represent onset of illness and therefore do not necessarily provide information about the time of year that the food was obtained, the duration of aging, or the duration of storage.

Type E botulism was by far the most frequent toxin type documented, associated with 83% (117 of 141) of the outbreaks, 80% (226 of 283) of the cases, and 85% (17 of 20) of the deaths. Types A and B were associated with nine and 38 cases, respectively. In 1998, both types B and E toxin were isolated from the emesis of one patient who had consumed stinkheads with a large group of persons. Three other

individuals became ill with vomiting and other symptoms, including diplopia and dry mouth; one of them had stool positive for type E toxin. In 2006, there was an outbreak of five cases; in 2007, there was an outbreak of one case. In both outbreaks, nonspecific toxicity was demonstrated in clinical samples and there was insufficient quantity of samples to conduct toxin typing. For the 2006 outbreak, nonspecific toxicity was also present in the food (salmon eggs); for 2007, food (beluga whale) testing was negative.

Botulism cases were reported predominately among residents of coastal villages in the western and southeastern parts of the state (Figure 3).

Figure 3. Mapped locations of foodborne botulism outbreaks (n=141) by toxin type — Alaska, 1950 to 2010.



Notes: Location of outbreak is based on patient residence and may not be the original location of the food source.

Circles are scaled to the number of outbreaks per location, and shading reflects the proportion of outbreaks attributed to the corresponding toxin type.

The Aleutian Islands west of Unimak Island are not shown because of space considerations and because no botulism outbreaks have been reported from those areas.

Foods

Implicated food samples were laboratory-confirmed in 56% (79 of 141) of the outbreaks corresponding to 56% (159 of 283) of the cases (Table 5). The remaining outbreaks were confirmed based on results from direct toxin testing of a patient’s serum, stool or gastric contents, or from a culture of a patient’s stool specimen. When food samples did not contain botulinum toxin, the results of the epidemiologic investigation were used to identify the food most likely responsible for the outbreak.

Table 5. Foods implicated in confirmed botulism outbreaks — Alaska, 1950 to 2010.

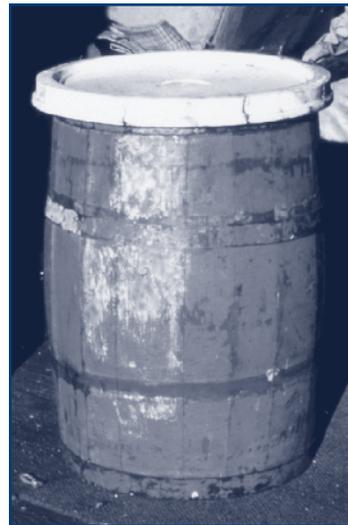
Type of Food Implicated	Number of Outbreaks	Number of Outbreaks with Positive Food*
Sea Mammal		
Seal (including seal oil)	54	36
Whale	15	8
Fish		
Salmon eggs	27	16
Salmon heads	14	6
Salmon, other	3	1
Whitefish	9	5
Herring	1	0
Semi-aquatic Mammal		
Beaver tail or paw	6	5
Other	5	2
Unknown	7	0
Total	141	79

*Note: For some outbreaks, foods were not available for testing.

Regardless of the animal source implicated in botulism outbreaks, the most common method of preparation for any food stuff was aging — where fresh food was allowed to cure for 1 to 2 weeks either in a pit in the ground or a closed or air-tight container (Table 6). No cases of foodborne botulism in Alaska have been associated with home-canned food.

Table 6. Method of preservation for implicated foods in confirmed botulism outbreaks — Alaska, 1950 to 2010.

Method of Preservation	Number Implicated
Aging (“fermentation”)	85
Drying	7
Rendering (oil)	30
Salting	1
Other / Unknown	18
Total	141



Distribution of botulism spores in the environment

In the 1970s, several studies documented the presence of *C. botulinum* in the environment. Miller et al. demonstrated type E botulism toxicity in enrichment cultures in 17 of 23 beach soil samples collected in the Kotzebue region.³¹ Other investigators detected low-level intrinsic contamination of Alaska salmon with type E spores.³² Among 589 pink, sockeye, chinook, and chum salmon collected from Bristol Bay, Southeastern Alaska, Kodiak, and the Yukon River, six (1%) had gill specimens yielding positive cultures for type E toxin. None of the 494 viscera specimens were positive.

The most extensive published environmental survey for *C. botulinum* in Alaska was conducted by Miller.³³ Samples of beach soil, ocean water and sediments, salmon, and marine mammals were collected from 23 sites in both interior and coastal areas; type E spores were widely distributed. No other type of *C. botulinum* was identified and, with one exception, no specimens from north of Point Hope were positive. Although no more recent surveys have been conducted, or surveys of any non-coastal areas, it is assumed that *C. botulinum* spores are widely distributed in the Alaska environment.

Die-offs from botulism among bird populations in the U.S. and Canada have occurred sporadically during the summer months for many years. Birds ingest botulinum toxin present in decomposing vegetation or invertebrates that have already accumulated toxin. *C. botulinum* can be found in many different natural environments. However, the majority of birds affected by botulism are waterbirds or waterfowl. Avian botulism is usually associated with toxin type C and sometimes type E. Humans appear to be relatively resistant to type C.³⁴ Since 1980, only one outbreak in 1999 has been recorded among Alaska birds (personal communication 2004, Dr. Kimberlee Beckmen, Alaska Department of Fish and Game). The die-off occurred in Haines and involved more than a dozen birds of several different species, including five trumpeter swans and two golden eyes. Blood samples from two of the swans and a goldeneye tested positive for botulinum toxin type E. No human illness was associated with this incident.

