Slide 1

Botulism

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Overview

- Organism
- History
- Epidemiology
- Transmission
- Disease in Humans
- Disease in Animals
- Prevention and Control

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In today's presentation we will cover information regarding the organism that causes botulism and its epidemiology. We will also talk about the history of the disease, how it is transmitted, species that it affects, including humans, and the clinical and necropsy signs observed. Finally, we will address prevention and control measures for botulism.

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Organism

- *Clostridium botulinum*
  - Gram positive
  - Obligate anaerobic bacillus
  - Spores
    - Ubiquitous
    - Resistant to heat, light, drying and radiation
    - Specific conditions for germination
      - Anaerobic conditions
      - Warmth (10-50ºC)
      - Mild alkalinity

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Botulism is caused by the bacterium *Clostridium botulinum*. It is a gram positive, spore-forming, obligate anaerobic bacillus. The clostridial spores are ubiquitous in soil and are very resistant to heat, light, drying and radiation. Spores may survive boiling for several hours at 100 ^{o}C, however exposure to moist heat at 120 ^{o}C for 30 minutes will kill the spores. Specific conditions are required for the germination of spores. These include anaerobic conditions (such as rotting carcasses or canned food), warmth, and mild alkalinity. The photo shows the bacillus shape of the bacterium *C. botulinum*. (Source: CDC Public Health Image Library: http://phil.cdc.gov/Phil/detail.asp?id=2131).

Slide 4

Neurotoxins

- Seven different types: A through G
  - Different types affect different species
  - All cause flaccid paralysis
  - Only a few nanograms can cause illness
  - Binds neuromuscular junctions
- Toxin: Destroyed by boiling
- Spores: Higher temperatures to be inactivated

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After germination, clostridial spores release neurotoxins. There are 7 antigenic types of neurotoxins, classified as A through G. Typically, different neurotoxin types affect different species. Only a few nanograms of these toxins can cause severe illness. All cause flaccid paralysis in the species affected. Toxin is produced in improperly processed, canned, low-acid or alkaline foods, and in pasteurized and lightly cured foods held without refrigeration, especially in airtight packaging. The physiologic mechanism of the neurotoxin is to irreversibly bind at neuromuscular junctions to prevent the release of acetylcholine (Ach). This causes muscular paralysis. The peripheral sensory nerves and the central nervous system are usually not affected. The toxin can be destroyed heat and cooking food at 80^{o}C for 30 minutes, however, inactivation of spores requires a much higher temperature.

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Neurotoxins

Neurotoxin	A	B	C	D	E	F	G
Human	X	X			X	X	
Horses		X	X				
Cattle		X	X	X			
Sheep			X				
Dogs			X	X			
Avian			X		X		
Mink & Ferret	X		X		X		

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This table summarizes the most common neurotoxin type affecting the various species affected by *C. botulinum*. All types of botulinum toxins produce the same clinical signs; however, the toxin type is important if antiserum is used for treatment. Type G has been isolated from soil and autopsy specimens but an etiologic role has not been established. Type E outbreaks are usually related to fish, seafood and meat from marine mammals.

Slide 6	History	
Slide 7	**History** • 1793, Justinius Kerner – "Wurstgift" • "Botulus" = Latin for sausage • 1895, Emile von Ermengem – Isolated organism during Belgium outbreak • U.S. outbreaks led to improved industry processing  Center for Food Security and Public Health Iowa State University - 2004	Botulism was first discovered by the German physician, Justinius Kerner in 1793. He called the substance "wurstgift" since he found it in spoiled sausages. During this period of time, sausage was made by filling a pig's stomach with meat and blood, boiling it in water then storing it at room temperature. These were ideal conditions for clostridial spores to survive. Botulism gets it name from "botulus" which is Latin for sausage. In 1895, Emile von Ermengem identified *Clostridium botulinum* as the actual source of a botulism outbreak in Belgium. Several outbreaks of botulism in the US have led to federal regulations for food preservation. In 1919, an outbreak from canned olives (15 deaths) led to the use of high temperatures as industry standards for preserving foods. In 1973, an outbreak from canned soup led to further regulations for the safe processing of canned foods.
Slide 8	Transmission	
Slide 9	**Transmission** • Ingestion – Organism – Spores – Neurotoxin • Wound contamination • Inhalation • Person-to-person not documented Center for Food Security and Public Health Iowa State University - 2004	Botulism transmission typically occurs through ingestion of the organism, neurotoxin or spores. If the organism is ingested, it then incubates in the stomach and produce spores which then germinate to release neurotoxin. If spores are ingested, germination follows and neurotoxin is released. Finally if spores have germinated within contaminated food, the neurotoxin itself is ingested, causing rapid progression of the disease. Other forms of transmission involve contamination of open wounds with clostridial spores. Additionally, inhalation of the neurotoxin is also possible. This is the most likely bioterrorism method that would be used for this agent. No instance of secondary person-to-person transmission has been documented.
Slide 10	Epidemiology	

Slide 11

Epidemiology

- In U.S., average 110 cases each year
  - Approximately 25% food-borne
  - Approximately 72% infant form
  - Remainder wound form
- Case-fatality rate
  - 5-10%
- Infective dose- few nanograms

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In the US, there are on average 110 cases of botulism per year. Typically about 25% are food-borne related illnesses. Approximately 72% are the infant botulism form and the remainder are wound related. In 1995, the reported case-fatality rate for botulism cases was 5-10%.

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Epidemiology

- 1977, Largest botulism outbreak
  - Michigan - 59 people
  - Poorly preserved jalapeno peppers
- Alaska
  - 27% of U.S. foodborne botulism cases
  - 1950-2000
    - 226 cases from 114 outbreaks

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To date, the largest botulism outbreak in the US occurred in 1977 in Michigan. Fifty-nine people were affected after eating poorly preserved jalapeno peppers. Approximately 27% of U.S. food-borne botulism cases occur in Alaska. During 1950-2000, Alaska recorded 226 cases of food-borne botulism from 114 outbreaks. All were Alaska Natives and were associated with eating fermented foods, which is a part of their culture. Due to changes in the fermentation process (use of closed storage containers), an increase in botulism rates occurred in Alaska from 1970-1989.

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Disease in Humans

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Human Disease

- Three forms
  - Foodborne
  - Wound
  - Infant
- All forms fatal and a medical emergency
- Incubation period: 12-36 hours

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Human botulism illness can occur in three forms: foodborne illness, infant botulism and wound contamination. These forms vary by how the toxin is obtained. All forms of the disease can be fatal and should be considered a medical emergency. The incubation period can range from 6 hours to 2 weeks. However, signs typically occur 12-36 hours after toxin release. Humans can be affected by types A, B, E and rarely F neurotoxins.

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Foodborne Botulism

- Preformed toxin ingested from contaminated food
- Most common from home-canned foods
  - Asparagus, green beans, beets, corn, baked potatoes, garlic, chile peppers, tomatoes; type A
  - Improperly fermented fish (Alaska); type E

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Foodborne botulism occurs when the preformed neurotoxin is ingested. The most common source of the preformed toxin is contaminated food, usually from improperly home-canned vegetables or fermented fish. Fifty percent of food-borne outbreaks in the US are caused by type A toxins. The most commonly isolated neurotoxin is type A for canned foods and type E for improperly fermented fish products.

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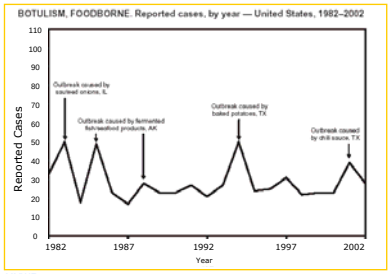

This graph depicts the trends of foodborne botulism cases in the U.S. from 1982-2002. In 1983, 28 persons in Illinois obtained food-borne botulism from a batch of sautéed onions. Twelve required ventilator support, however no deaths occurred (MMWR 1984:33(2):22-23). During 1950-2000, Alaska recorded 226 cases of food-borne botulism from 114 outbreaks. All were Alaska Natives and were associated with eating fermented foods. In 1994, an outbreak at a Greek restaurant in Texas affected 30 persons from improperly stored foil-wrapped baked potatoes. The 2001 Texas outbreak resulted in 39 cases of foodborne botulism from persons eating commercially produced chili sauce that had been improperly stored. Overall botulism is a rare disease, but it can be fatal and every case of botulism is treated as a public health emergency. Graph from the Summary of Notifiable Diseases 2002, CDC website.

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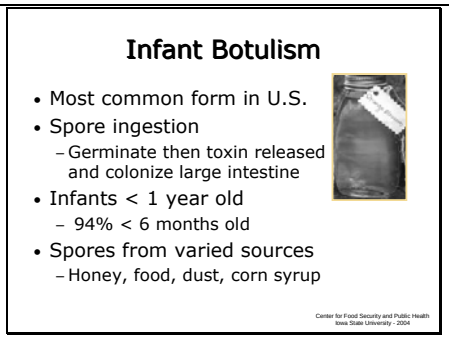

The most common form of human botulism occurs in infants. Annual incidence in the US is two cases per 100,000 live births. Spores are ingested, germinate, then release their toxin and colonize the large intestine. It occurs predominantly in infants less than 1 year old (94% are less than 6 months old). The spores are obtained from various sources such as honey, food, dust, and corn syrup.

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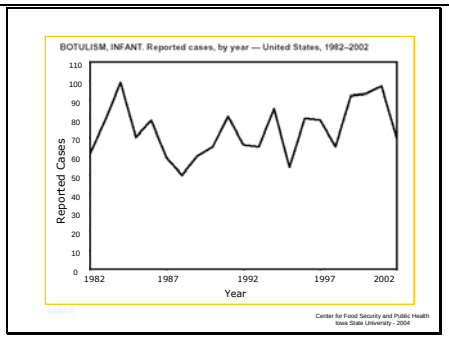

This line graph shows the reported number of cases of infant botulism from 1982 to 2002. Infant botulism is the most common type of botulism in the United States; there were a total of 69 cases reported to the CDC in 2002. Cases are sporadic and risk factors remain largely unknown. Graph is from the Summary of Notifiable Diseases 2002, CDC website.

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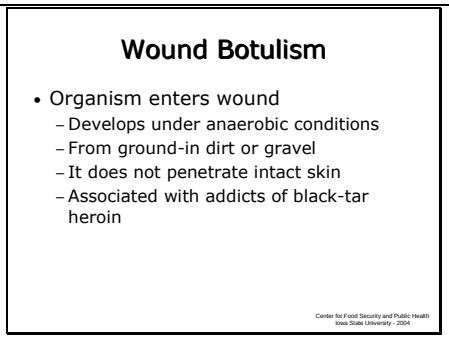

Wound botulism is rare and occurs when the organism gets into an open wound and develops under anaerobic conditions. The organism typically comes from ground-in dirt or gravel. *C. botulinum*, its spores or neurotoxin cannot penetrate intact skin. This form has also been associated with addicts of black-tar heroin. It is thought to be contaminated with dirt or boot polish during its preparation process. There have been clusters of cases each year in these drug users, some resulting in fatalities.

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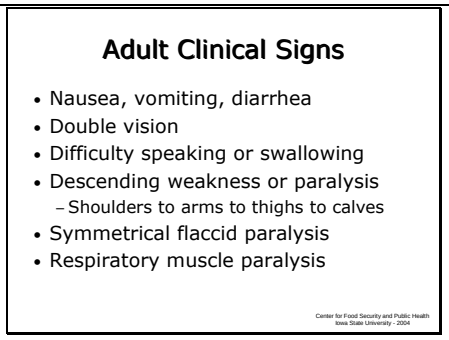

In humans, the clinical signs of botulism are similar for all forms of the disease. Gastrointestinal signs (i.e., nausea, vomiting, diarrhea) are usually the first signs to appear. They are followed acutely by neurological signs, such as bilateral cranial nerve deficits. The victim will have double vision, and difficulty seeing, speaking and swallowing. This soon develops into a descending weakness to symmetrical flaccid paralysis. This paralysis can affect the respiratory muscles and lead to death.

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Infant Clinical Signs

- Constipation
- Lethargy
- Poor feeding
- Weak cry
- Bulbar palsies
- Failure to thrive

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Children less than 1 year of age with the following clinical signs should be suspected of infant botulism. Constipation, lethargy, poor feeding, weak cry, bulbar palsies, failure to thrive, and progressive weakness. This can lead to impaired respiration and sometimes death if not treated promptly. The child in this picture is too weak to hold up its head as noted by the limp appearance of the neck and arms. It was an infant case of botulism. 72% of natural botulism cases occur in children under 1 year of age. California Department of Health Services http://www.dhs.cahwnet.gov/dcdc/InfantBot/toxfig2.htm

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Diagnosis

- Clinical signs
- Toxin in serum, stool, gastric aspirate, suspected food
- Culture of stool or gastric aspirate
  - Takes 5-7 days
- Electromyography also diagnostic
- Mouse neutralization test
  - Results in 48 hours

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Clinical signs can provide a tentative diagnosis for botulism intoxication. The definitive diagnosis in humans involves identifying the toxin in serum, stool, gastric aspirate, or if available, the suspected food. Feces are usually the most reliable clinical sample in foodborne or infant botulism. Additionally, cultures of stool or gastric aspirate samples may produce the organism, but can take 5-7 days. Electromyography (EMG) can also be diagnostic. The most widely used and sensitive test for detecting botulism toxin is the mouse neutralization test. Serum or stool with the suspected botulism organism is injected into a mouse and observed for clinical signs of the disease. Results are available in 48 hours.

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Treatment

- Intensive care immediately
  - Ventilator for respiratory failure
- Botulinum antitoxin
  - Derived from equine source
  - CDC distributes
  - Used on a case-by-case basis
- Botulism immune globulin
  - Infant cases of types A and G

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Most cases of botulism require immediate intensive care treatment. Due to respiratory paralysis, a mechanical ventilator will be needed if respiratory failure occurs. An intravenous equine-derived botulinum antitoxin is available on a case-by-case basis from the CDC through state and local health departments. Botulism immune globulin was approved for use on October 23, 2003 for the treatment of infant botulism caused by types A and G.

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Animals and Botulism

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Animals

- Cattle and sheep
- Horses
- Birds and poultry
- Mink and ferrets
- Uncommon in dogs and pigs
  - Fairly resistant
- No natural cases documented in cats

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Botulism affects animal species such as cattle, sheep, horses, wild birds and poultry, and mink and ferrets. Dogs and pigs can also be affected, however they seem to be more resistant to the disease. Therefore, cases are much more uncommon compared to the previously mentioned species. To date no natural cases have been documented in cats.

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Cattle and Sheep

- Ingestion of toxin
- Incubation
  - 24 hours to 7 days
- Sources
  - Spoiled stored silage or grain
  - Silage using poultry litter or products
  - Phosphorus deficiency in cattle
  - Carcasses: Baled or chopped into hay

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In cattle and sheep, disease is usually caused by ingestion of neurotoxin in contaminated feed stuffs. Most cattle cases involve type B, C, and D toxin while most sheep cases involve type C toxin. The incubation time is 24 hours to 7 days. Common sources of the toxin include improperly stored silage or spoiled brewer's grains. Silage incorporating poultry litter or poultry products can also be a source of botulism toxin. Cattle with phosphorus deficiency can obtain the toxin via ingestion of soil while enacting pica. Finally, carcasses unintentionally baled into hay or chopped into hay cubes or pellets may potentially contribute to botulism in ruminants. This later source was responsible for the deaths of 400 dairy cattle in a California herd in 1998. Following the outbreak, it was discovered that the unintentional contamination occurred from the carcass of a dead cat in the feed. Photo from Israel Veterinary Medical Association http://www.isrvma.org/article/56_3_4.htm.

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Ruminants: Clinical Signs

- Progressive ascending ataxia
- Recumbent
- Head turned into flanks
- Cranial nerve dysfunction
- Rumen stasis; bloat
- Atonic bladder - loss of urination

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Clinical signs in ruminants include progressive ascending ataxia from the hindlimbs to the forelimbs. Animals are usually recumbent and cattle will turn their heads into their flanks. Signs of cranial nerve dysfunction are present, such as dysphagia, drooling, tongue paresis, and facial muscle paresis. Eye effects include decreased pupillary light reflex, ptosis and mydriasis. Additionally, rumen stasis and bloat can occur, as well as an atonic bladder with loss of urination. Photo from Israel Veterinary Medical Association http://www.isrvma.org/article/56_3_4.htm.

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Cattle and Sheep: Diagnosis

- History
- Bloodwork and CSF tap: Normal
- ELISA test available for type C & D
- Definitive diagnosis
  - Demonstration of toxin in serum, gut contents or organs
- Electromyography (EMG)

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Diagnosis of botulism in ruminants can be determined by obtaining a good history. Bloodwork and CSF taps are usually normal. An ELISA test is available for types C and D toxin. The definitive diagnosis comes from demonstration of the toxin in serum, gut contents or organs. Additionally, electromyography (EMG) results may be diagnostic.

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Cattle and Sheep: Treatment

- Symptomatic and supportive
- Nutritional
- Ventilatory support, if needed
- Metronidazole
- Antitoxin, in early stages
  - Ineffective by the time clinical signs are present
  - Can block further uptake of toxin

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Treatment for ruminants includes symptomatic and supportive treatment. This includes general nursing care, fluids and nutrition. Ventilator support may be needed. Metronidazole may be useful, however **avoid aminoglycosides, tetracyclines and procaine penicillin as they have been associated with neuromuscular weakness**. Antitoxin may be given when diagnosed in an early stage. It is usually ineffective by the time clinical signs are present, but it can block further uptake of the toxin.

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Horses

- Horses, especially foals, are highly sensitive to botulism toxin
- Type B & C toxins
- Incubation period
  - 24 hours to 7 days
- Sources
  - Contaminated feed
  - Wound infections

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Horses, especially foals, are highly sensitive to botulism toxin. The most common toxin in equines are types B and C. The incubation period in equines is 24 hours to 7 days. In horses, the most common sources of botulism occur from contaminated feed or infection by the organism in open wounds. Photo of a foal provided by Danelle Bickett-Weddle, Iowa State University.

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Adult Horses

- "Forage poisoning"
  - Ingest preformed toxin
- Clinical Signs
  - Dyspnea
  - Flaccid tail
  - Muscle tremors
  - Severe paresis to rapid recumbency
  - Unable to retract tongue, drooling

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In adult horses, botulism is also called "forage poisoning". Horses ingest the preformed toxin from contaminated feed. Clinical signs include dyspnea, a flaccid tail and muscle tremors. They will have severe paresis which progresses to rapid recumbency. Adult horses will be unable to retract their tongue and will therefore, drool. Photo from www.aht.org.uk/fsheets/fshets10.html.

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Foals

- "Shaker Foal" syndrome
  - Most 2 weeks to 8 months old
  - On a high nutrition plane
- Spores in contaminated feed
- Usually type B
  - Most common in KY and eastern seaboard

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In foals, botulism causes "Shaker Foal Syndrome" and typically affects them from 2 weeks to 8 months old. Foals that are on a high plane of nutrition are more susceptible. Foals typically consume spores in contaminated feed which germinate and release the neurotoxin within the gastrointestinal tract. Type B toxin is the most common form found in foals. Shaker foal syndrome is most common in Kentucky and the eastern seaboard.

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Foals: Clinical Signs

- Clinical signs
  - Paresis, recumbent
  - Muscle tremors
  - Dysphagia
  - Ptosis, mydriasis, decreased PLR
  - Ileus, constipation, urine retention
  - Death due to respiratory paralysis
- Mortality greater than 90%

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Clinical signs in foals include paresis, recumbency, and muscle tremors. Cranial nerve signs also exist, such as dysphagia, ptosis, mydriasis, and decreased pupillary light reflex. Additional signs include ileus, constipation and urine retention. Death is usually due to respiratory paralysis. Mortality can be greater than 90%.

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Birds and Poultry

- "Limber neck"
- Types C and E
- Good sentinel species
- Sources:
  - Decomposed vegetation or invertebrates
  - Ingest toxin or invertebrates with toxin
  - Contaminated feed or water of chickens

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Botulism infection in avian species is commonly referred to as "limber neck" disease. The most common neurotoxin type found in birds is type C for waterfowl and shorebirds (especially ducks in the western U.S.) and poultry. Type E neurotoxins affect gulls and loons. Wild birds can be a good sentinel species. The most common source for wild birds is decomposing (anaerobic conditions) vegetation and invertebrates. The birds ingest the toxin or invertebrates with the toxin. Outbreaks occur from coast to coast in the United States and Canada, generally from July through September. Thousands of birds may die during a single outbreak. Botulism toxin can be common in the gut of poultry and wild birds as well as the litter, feed and water of chickens. Photo from Canadian Cooperative Wildlife Health Centre at http://wildlife.usask.ca/bookhtml/botulism/botulismc.htm

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Birds and Poultry: Clinical Signs

- Occurs 12-48 hours after ingestion
- Droopy head
- Drowsy
- Wing and leg paralysis
  - Unable to hold their head up
  - Unable to use their wings or legs
- Eyelid paralysis

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Avian species typically show clinical signs of botulism 12-48 hours after ingestion of the toxin. They will have a 'limber neck', with a droopy head and appear drowsy. Infection makes these birds unable to use their wings or legs or to hold their heads up, so they drown. Death can also result from water deprivation, electrolyte imbalance, respiratory failure and predation.

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Mink and Ferrets

- Type C
  - Occasionally A and E
- Sources
  - Chopped raw meat or fish
  - Improper storage of meat by-products
- Vaccine available for type C

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Mink and ferrets are extremely susceptible to botulism. They are usually affected by type C toxin, occasionally types A and E can be isolated. The most common sources are from chopped raw meat or fish or can come from improper storage of meat by-products. A vaccine is available for these animals. Annual vaccination of kits and breeding animals with botulism (type C) toxoid is recommended to prevent outbreaks. Kits should be vaccinated after 6 weeks of age.

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Dogs

- Rare
- Type C; few cases type D
- Source
  - Ingestion of carrion
  - Wetland areas with avian botulism epizootics
- Incubation period
  - Few hours to 6 days

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Botulism in dogs is rare however, the majority of cases of canine botulism are caused by neurotoxin type C; a few are caused by type D. Cases are typically caused by ingestion of the toxin. This may come from the ingestion of contaminated carrion, or in hunting breeds exposed to wetland areas with avian botulism epizootics. The incubation period in dogs ranges from a few hours to 6 days. Duration of illness is from 14-24 days.

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Dogs

- Progressive symmetric ascending weakness
  - Rear limbs to forelimbs
- Cranial nerve deficits
- Respiratory paralysis
- Lose ability to urinate and defecate

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Clinical signs involve progressive symmetric ascending weakness from rear to forelimbs that can result in quadriplegia. Cranial nerves are also affected causing decreased pupillary light reflexes, jaw tone, and gag reflexes. Pain perception is still maintained and the dog is alert. Death from respiratory paralysis can occur. Dogs can also lose their ability to urinate and defecate.

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Dogs

- Diagnosis
  - Bloodwork and CSF: Normal
  - Electromyography (EMG)
  - Toxin in serum, vomitus, feces, or suspect food/carrion
  - Mouse neutralization test preferred
- Treatment
  - Supportive
  - Antitoxin

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The diagnosis of botulism in dogs can be difficult. History of carrion ingestion and physical exam can be helpful. Bloodwork and CSF taps are usually within normal limits. A electromyography (EMG) can be diagnostic. Additionally, demonstration of the toxin in serum, vomitus, feces or the suspected food/carrion can also be diagnostic. The preferred method is the mouse neutralization test. Treatment involves supportive and nursing care because dogs will not be able to swallow, eat or drink well. Additionally, loss of urination and defecation ability may require assistive measures. Antibiotics are usually not indicated, since a toxin is the cause of the clinical signs. Antitoxin can be administered, but it is usually not effective once the toxin has bound to neuromuscular junctions. It can however prevent further binding of any toxin remaining in the system. There is a potential risk of anaphylactic shock with the antitoxin.

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Prevention and Control

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Human: Prevention

- Do not feed honey to children <1 yr of age
- Proper food preservation methods
  - Proper time, temperature and pressure
    - 80ºC for 30 min or 100ºC for 10 min
- Prompt refrigeration of foods
- Boil foods for > 10 minutes
- Decontamination
  - Boil suspected food before discarding
  - Boil or chlorine disinfect utensils used

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Prevention of botulism in humans includes educating yourself and clients about this disease. Because honey can contain botulinum spores and is not chemically treated/boiled/pasteurized before consuming, it is recommended that children under one year of age should not eat honey. Other recommendations include proper home canning and food preservation methods which will destroy the spores. Prompt refrigeration of foods will also help. Boiling foods, especially those that are home canned, for over 10 minutes to destroy the toxin. Additionally, avoid feeding honey to infants. Before discarding any suspected food be sure to boil it for the appropriate time to detoxify it. Boil or chlorine disinfect any utensils that were in contact with the suspected food. Finally, report any suspect cases to the state or local health authorities.

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Ruminants: Prevention

- Good husbandry practices
- Rodent and vermin control
- Prompt disposal of carcasses
- Avoid spoiled feedstuff or poor quality silage
- Vaccination in endemic areas

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The best prevention against botulism in ruminants includes good husbandry practices. Rodent and vermin control will minimize potential carcass sources. Prompt disposal of carcasses will be helpful. Avoid spoiled feedstuffs or poor quality silage. Vaccines can be used in cattle, sheep and goats in endemic areas.

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Equine: Prevention

- Good husbandry
- Rodent and vermin control
- Avoid spoiled feed
- Prophylactic vaccine for pregnant mares
  - Currently only type B botulinum toxoid available for horses

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Prevention of botulism in horses includes good husbandry practices. Rodent and vermin control will help to reduce carcass contamination of feed. Avoid feeding spoiled feed stuffs. A prophylactic vaccine is available for pregnant mares. However, only type B botulinum toxoid is available for horses. Initially, mares should be vaccinated during gestation with a series of three doses administered 1 month apart, with the last dose 2-4 weeks before foaling to ensure optimal protection of the foal via colostrum. Mares should be booster vaccinated with a single dose 1 month before foaling. Vaccination of horses with type B toxoid will not induce protection against other neurotoxin types, since there is no cross-protection between them. Currently there is no approved equine vaccine for protection against type C botulism.

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Potential Bioterrorism Threat

- Aum Shinriky cult
- Extremely potent and lethal
- Easily produced and transported
- Signs of deliberate aerosol or foodborne release of toxin
  - No common source
  - Large number of acute cases clustered
  - Uncommon toxin type (C, D, F, G)

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Botulinum toxin has been used as an attempted bioweapon. Between 1990 and 1995, the Japanese cult Aum Shinriky used botulinum toxin aerosols at multiple sites in Tokyo, Japan. Fortunately, these attempts failed. As a potential bioterrorism agent, botulism toxin is extremely potent and lethal. It is easily produced and transported. Signs of a deliberate release of the toxin, either via aerosol or food, would be a large number of acute cases from no common source and occurring as a cluster. Additionally, uncommon toxin types, such as C, D, F, or G, may raise suspicion.

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Potential Bioterrorism Threat

- Point source aerosol release
  - Incapacitate or kill 10% of persons within 0.5 km downwind
- CDC surveillance system
  - Prompt detection of botulism related events

It is estimated that a point source aerosol release of botulinum toxin could incapacitate or kill 10% of persons within 0.5 km downwind (JAVMA 2001;285:1059-1070). However, the CDC maintains a well-established surveillance system for reporting human botulism cases that would promptly detect such an event.

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Additional Resources

- CDC – Division of Bacterial and Mycotic Diseases
  - http://www.cdc.gov/ncidod/dbmd/diseaseinfo/botulism_g.htm
- Center for Civilian Biodefense Strategies
  - http://www.hopkins-biodefense.org/pages/agents/ agentbotox.html

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