Testimony of to the Ronald S. Klein to the House Labor and Commerce Committee

HB 202, "An Act relating to the sale of food products by the producer to the consumer"

March 21, 2012

Mr. Chairman, thank you for the opportunity to testify on HB 202. My name is Ron Klein and I have strong interest in growing Alaska's food industry, promoting the development of markets for local food as well as maintaining our current national and global markets for Alaska food products.

I retired in October as the State's Food Safety and Sanitation Program Manager. I am Past-President of the Association of Food and Drug Officials which represents state agricultural and health food safety directors and program managers on a national level to promote safe food systems in the United States. I am currently working as a food safety and emergency preparedness subject matter expert for institutions such as Louisiana State University, University of Tennessee Knoxville, and the International Food Protection Training Institute in Michigan. Locally, I am serving on the Governing Board of the Alaska Food Policy Council and part of the effort to help develop a market for safe, healthy, local foods.

My comments today are my own and do not represent my clients, employers or any organization I am associated with.

I do not think HB 202 is a productive way to grow Alaska's food industry. In fact it would have a deleterious effect on public health, harm to efforts to build a local food industry and could have a serious impact on Alaska's seafood industry.

HB 202 would remove all public health controls on the sale of locally produced foods to consumers. It would enable sale direct to consumers of potentially hazardous foods such as smoked or canned fish, dried meats, shellfish, poultry products, dairy products, and acidified and low acid canned foods. Consuming improperly processed adulterated foods leads to serious illness or death from ingestion of pathogens such as, e.coli, salmonella, listeria, campylobacter, Clostridium botulinum, , and toxins such as paralytic shellfish poisoning. Problems with these products may not just make the people who consume them ill. The people infected with e.coli, salmonella, listeria, campylobacter them can make other people ill through poor sanitation and the fecal oral route.

The risk of foodborne illness from potentially hazardous foods is not hypothetical. The costs of a foodborne illness are not zero. Alaska leads the nation in the number of illnesses associated with botulism. The primary food source is fish and traditional foods. Under HB202 the foods which caused these illnesses which normally limited to family members and friends, could be sold directly to consumers and kill them. There would be no public health controls. Unfortunately, unless the consumers have health insurance the costs of their illness would be borne by Alaskans.

The lack of labeling, record keeping, and regulatory oversight for potentially hazardous foods will also complicate the efforts of to investigate food borne illnesses, their causes and sources. There will be no

way for public health professionals to trace back the source of a food borne illness to the source. There will be no ability for environmental health specialists to assist processors with identifying and correcting sanitation and processing flaws.

In 2008 there was a campylobacter outbreak in Alaska associated with the consumption of shucked raw peas, which are a non-potentially hazardous food, from a farm in the Mat-Su Valley. There were 54 laboratory confirmed illnesses associated with the peas. Five patients were hospitalized and one developed Guillain-Barré syndrome. These products would be exempt under HB 202. Under HB 202 DEC environmental health specialists would not have had the ability to help the farmer/processor identify food safety measures to implement to save his business.

Illnesses associated with these foods can harm the reputation of Alaska's food industry. Under HB202 an individual can, grow, process and sell shellfish in untested waters and without testing the shellfish for PSP beforehand. Last summer there a number of individuals became ill in southeast Alaska with PSP after consuming shellfish which they personally gathered. I recall being contacted by ASMI and growers who were concerned that the market for regulated inspected shellfish products would be harmed due to the publicity of people becoming ill after eating Alaska's shellfish. If someone contracts botulism from eating Joe's smoked or canned fish , it will have an impact on the market for inspected smoked or canned fish and harm the reputation of Alaska's seafood industry and the businesses of those who are doing it right.

Just last summer there were 7 lab confirmed cases of campylobacter illnesses associated with consuming raw milk from a farm in the Mat-Su Valley.

Last year the federal Food Safety Management Act of 2011 became law. There are a number of misconceptions about whether it applies to small producers. Apparently this appears to have been a model for HB 202. The fact is FSMA does. Facilities only have a qualified exemption. Facilities that qualify would be exempt from the preventive control/HACCP provisions in S. 510, but would still have to comply with one of the following:

(1) They would have to demonstrate that they have identified potential hazards and are implementing preventive controls to address the hazards, or

(2) they would have to demonstrate to FDA that they are in compliance with state or local food safety laws

The Tester Amendment also includes a specific NON-preemption of state and local authority. Congress expected the states to continue regulating small producers as it has done so for decades. It only intended to limit federal involvement. However Congress also didn't turn its back.

According to the Tester Amendment in the event of an active investigation of a foodborne illness outbreak that is directly linked to a facility or farm exempted under this section, or if the Secretary determines that it is necessary to protect the public health and prevent or mitigate a foodborne illness

outbreak based on conduct or conditions associated with a facility or farm that are material to the safety of food, the Secretary may withdraw the exemption provided to such facility.

HB 202 will do nothing to promote the sale of farm to schools or build sustainable value added industry. I recommend that HB 202 be efforts be tabled. DEC is completing revisions to the Alaska Food Code which essentially deregulates or simplifies regulation of non-potentially hazardous food sold directly to consumers. I think when it is in effect it will go a long ways towards meeting the needs of Alaskan's who want to selling some products directly to consumers.

Other efforts such as the one envisioned by HCR 24, which has received broad House support and which asks the Governor to create a State Food Resource development Group and the activities detailed in the Alaska Food Policy Council's Strategic plan will provide for a thoughtful reasoned approach to grow Alaska's food industry and increase the opportunities for Alaskan's to have better access to safe, nutritious, high quality local foods.

Attachments:

Botulism cases over the last 10 years

Three Outbreaks of Foodborne Botulism Caused by Unsafe Home Canning of Vegetables—Ohio and Washington, 2008 and 2009

Campylobacteriosis Outbreak due to Consumption of Raw Peas — Alaska, 2008

Ongoing Raw Milk Campylobacter Outbreak — Southcentral Alaska, July 2011

Year	Month	State	Genus Species	Etiological Status	Location of Consumption	Total ill	Total Hospitalizations	Total Deaths	Food Vehicle
2006	October	Alaska	Clostridium botulinum	Confirmed	Private home	5	2	0	ethnic style, unspecified
2008	August	Alaska	Clostridium botulinum	Confirmed	Private home	2	2	0	fish, white
2008	September	Ohio	Clostridium botulinum	Confirmed	Private home	4	4	0	home-canned vegetable, unspecified
2008	May	Alaska	Clostridium botulinum	Confirmed	Private home	2	1	0	Specialty/Ethnic dishes
2007	June	Multistate outbreak reported by CDC	Clostridium botulinum	Confirmed	Private home	8	8	0	chili
2007	November	Colorado	Clostridium botulinum	Suspected	Private home	4	3	1	peppers, chili
2006	January	California	Clostridium botulinum	Confirmed	Private home	2	2	0	home canned carrots
2007	March	Alaska	Clostridium botulinum	Confirmed	Private home	2	2	0	Specialty/Ethnic dishes
2006	November	California	Clostridium botulinum	Confirmed	Private home	2	2	0	TOFU, FERMENTED
2006	September	Multistate outbreak reported by CDC	Clostridium botulinum	Confirmed	Private home	4	4	1	Carrot juice, pasteurized
2007	May	Alaska	Clostridium botulinum	Confirmed	Private home	2	2	0	seafood, other
2009	April	California	Clostridium botulinum	Confirmed	Private Home	2	2	0	HOMECANNED TUNA
2008	November	Alaska	Clostridium botulinum	Confirmed	Private home	2	2	0	seafood, other
2000	January	California	Clostridium botulinum	Confirmed	Private home	2	2		squash, unspecified; tomato, unspecified
1998	December	Pennsylvania	Clostridium botulinum	Confirmed	Private home	2	2		soup unspecified
1998	October	California	Clostridium botulinum	Confirmed		3	3	0	tuna, unspecified

2000	April	Ohio	Clostridium botulinum	Confirmed	Workplace, not cafeteria	3	3	1 home canned pepper, unspecified
1999	September	Florida	Clostridium botulinum	Confirmed	Private home	3	3	0 garlic and oil
1998	June	Alaska	Clostridium botulinum	Confirmed	Private home	3	3	0 unspecified fish
2001	January	Alaska	Clostridium botulinum	Confirmed	Private home	4	4	0 beaver, fermented tail (muktuk)
2001	July	Alaska	Clostridium botulinum	Confirmed	Private home	2		fish, white
2001	August	Alaska	Clostridium botulinum	Suspected	Private home	2		fish eggs, unspecified
2001	August	Texas	Clostridium botulinum	Confirmed	Church, temple, etc; Private home	16	9	0 beef, stew
2002	January	Maine	Clostridium botulinum	Confirmed	Private home	4	2	meat sauce
2002	September	Alaska	Clostridium botulinum	Suspected	Private home	3	1	0 soup, other poultry/egg
2002	July	Alaska	Clostridium botulinum	Confirmed	Private home	2	2	0 salmon eggs
2002	July	Alaska	Clostridium botulinum	Confirmed	Private home	8	5	0 whale, unspecified
2002	May	Alaska	Clostridium botulinum	Suspected	Private home	6		0
2003	October	Utah	Clostridium botulinum	Confirmed	Private home	2	2	2
2005	November	California	Clostridium botulinum	Confirmed	Private home	2	2	1 salmon, unspecified
2004	June	California	Clostridium botulinum	Confirmed	Prison, jail	5	5	0 alcohol
2005	August	Alaska	Clostridium botulinum	Confirmed	Private home	4	2	0 salmon, unspecified
2005	August	Alaska	Clostridium botulinum	Confirmed	Private home	4	2	0 salmon heads
2004	December	Oregon	Clostridium botulinum	Confirmed	Private home	2	2	0 mushrooms, canned

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Three Outbreaks of Foodborne Botulism Caused by Unsafe Home Canning of Vegetables—Ohio and Washington, 2008 and 2009†

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ABSTRACT

Foodborne botulism is a potentially fatal paralytic illness caused by ingestion of neurotoxin produced by the spore-forming bacterium *Clostridium botulinum*. Historically, home-canned vegetables have been the most common cause of botulism outbreaks in the United States. During 2008 and 2009, the Centers for Disease Control and Prevention (CDC) and state and local health departments in Ohio and Washington State investigated three outbreaks caused by unsafe home canning of vegetables. We analyzed CDC surveillance data for background on food vehicles that caused botulism outbreaks from 1999 to 2008. For the three outbreaks described, patients and their family members were interviewed and foods were collected. Laboratory testing of clinical and food samples was done at the respective state public health laboratories. From 1999 to 2008, 116 outbreaks of foodborne botulism were reported. Of the 48 outbreaks caused by home-prepared foods from the contiguous United States, 38% (18) were from home-canned vegetables. Three outbreaks of Type A botulism occurred in Ohio and Washington in September 2008, January 2009, and June 2009. Home-canned vegetables (green beans, green bean and carrot blend, and asparagus) served at family meals were confirmed as the source of each outbreak. In each instance, home canners did not follow canning instructions, did not use pressure cookers, ignored signs of food spoilage, and were unaware of the risk of botulism from consuming improperly preserved vegetables. Home-canned vegetables remain a leading cause of foodborne botulism. These outbreaks illustrate critical areas of concern in current home canning and food preparation knowledge and practices. Similar gaps were identified in a 2005 national survey of U.S. adults. Botulism prevention efforts should include targeted educational outreach to home canners.

Botulism is a serious neuroparalytic and sometimes fatal illness caused by potent neurotoxins produced by the gram-positive, anaerobic, spore-forming bacterium *Clostridium botulinum* and rare toxigenic strains of *Clostridium baratii* and *Clostridium butyricum*. Seven types of botulinum toxins are known (A through G), of which types A, B, E, and F cause virtually all cases of human botulism *(8)*. Botulism is characterized by rapidly progressive cranial neuropathy and symmetric descending flaccid paralysis, which may progress to respiratory arrest requiring mechanical ventilation and intensive supportive care in ~60% of patients *(24)*. Clinical recovery takes several weeks to months *(24)*. While prompt administration of the specific antitoxin can halt progression, intensive-care supportive measures remain the mainstay of treatment *(8, 24)*.

Foodborne botulism is caused by ingestion of foods contaminated with preformed botulinum toxin, and illness onset typically occurs 18 to 36 h after toxin ingestion *(24)*. *C. botulinum* spores are ubiquitous and heat resistant to processes that kill nonsporulating organisms, but spore germination and toxin elaboration require high water activity ($a_w > 0.955$) and anaerobic, low-salt, low-sugar, and low-acid conditions at nonrefrigeration temperatures *(3, 15, 16)*. In addition to being a clinical emergency, botulism is also a public health emergency because a single contaminated food can cause illness in many persons *(23)*.

From 1950 through 1996, 1,087 cases and 444 outbreaks of foodborne botulism were reported in the United States, with a mean of 23 cases and 9 outbreaks per year *(8)*. An outbreak was any occurrence of foodborne botulism associated with consumption of a specific contaminated food, whether a single case or a multicase cluster. Most U.S. outbreaks of foodborne botulism are caused by home-processed and home-canned foods *(13, 29)*, with home-canned vegetables alone accounting for 56% of outbreaks in the United States in which a specific food

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vehicle was identified *(8)*. Recent published accounts of botulism outbreaks have focused on outbreaks caused by commercial foods *(7, 17, 25),* novel or unusual food vehicles *(31),* and international outbreaks *(9–11)*. Though home-canned vegetables have long been recognized as the most common cause of U.S. botulism outbreaks, few reports of these outbreaks have been published in recent decades. We summarize foodborne botulism outbreaks in recent years and describe three recent outbreaks caused by home-canned vegetables. Our investigations show the need to improve awareness and education among home canners regarding safe processing and preparation practices to reduce the risk of foodborne botulism.

MATERIALS AND METHODS

Botulism surveillance. Immediate reporting of suspect botulism patients to state public health authorities is mandatory in all 50 states. Additional botulism consultation for clinicians and state health authorities is available on a 24-h basis through the Centers for Disease Control and Prevention (CDC). Botulism antitoxin for treatment of U.S. foodborne botulism patients is only available through CDC (and the states of California and Alaska), and confirmatory laboratory testing for botulism is available only through state health departments and CDC. The CDC National Surveillance Team compiles botulism surveillance information from state health departments and antitoxin release data into an electronic database.

Clinical and epidemiologic investigation. Botulism outbreak investigations were conducted by state health departments and CDC, and CDC collected additional demographic, clinical, and epidemiologic information from antitoxin recipients, using a standard Botulism Case Report form. CDC surveillance data were analyzed to provide background information about food vehicles that have been responsible for recent foodborne botulism outbreaks (1999 to 2008, the most recent 10 years for which data were available). For the three outbreaks presented in this report, hypothesis-generating interviews, including 5-day food histories, were administered to patients and their family members to identify suspect foods and other potentially exposed persons. Interviewers also collected detailed information about preservation (e.g., home canning), storage, and meal preparation of suspect foods. Where available, photocopies of relevant canning recipes were requested, as well as information about prior home canning experience, incentives for home canning, and knowledge regarding botulism from patients and family members. Up to three additional attempts were made to interview patients or family members who were initially unavailable for interview.

Laboratory investigation. Samples of serum, stool, and suspect foods were obtained from patients and forwarded to the respective Ohio and Washington state public health laboratories for confirmatory botulism testing. Laboratory isolation of *C. botulinum* was done by using directly inoculated agar media or enrichment cultures, and botulinum toxin was identified through diffusion-in-gel enzyme-linked immunosorbent assay and the mouse toxicity and neutralization bioassay, the laboratory methods for which are described elsewhere *(8)*. Laboratory safety precautions for work with *C. botulinum* toxin were followed in accordance to the fifth edition of *Biosafety in Microbiological and Biomedical Laboratories* (2009), published by the CDC and the National Institutes of Health. Botulinum toxin is considered a select agent, and work with this toxin is regulated under 7 CFR Part 331, 9 CFR Part 121, and 42 CFR Part 73. All materials were handled under regulations using Biosafety Level 2 and level 3 practices, containment, and facilities.

RESULTS

Types of foods implicated in U.S. foodborne botulism outbreaks, 1999 to 2008. From 1999 to 2008, 116 outbreaks (201 cases) of foodborne botulism were reported, including 82 outbreaks with information about the source and preservation methods for the implicated food. Commercial and home-prepared foods were implicated in 9% ($n = 7$) and 91% ($n = 75$) of outbreaks, respectively. Among the 75 reported outbreaks caused by home-prepared foods, home-canned foods accounted for 44% ($n = 33$), traditional Alaska Native uncooked aquatic game foods for 36% ($n = 27$), and 20% ($n = 15$) were attributed to other home-prepared foods including a variety of meats, fermented tofu, and pruno. All outbreaks from Alaska Native aquatic game foods occurred in Alaska, and no Alaska outbreaks were attributed to other types of food vehicles. Excluding Alaska, home-canned vegetables were the most common single cause of outbreaks (18 of 48) from home-prepared foods in the United States.

Outbreak 1: Ohio, September 2008. On 12 September 2008, two family members (patient no. 1, a 76-year-old male, and patient no. 2, a 15-year-old male, grandson of patient no. 1) were hospitalized with gastrointestinal symptoms accompanied by bilateral cranial nerve palsies and progressive peripheral muscle weakness (Table 1). Paralysis progressed rapidly, and both patients required mechanical ventilatory support that day. On 13 September, four additional family members were evaluated at the same hospital: patient no. 3 (80-year-old female, wife of patient no. 1), who also progressively worsened and underwent intubation; patient no. 4 (38-year-old male, son of patients no. 1 and no. 3), who had milder symptoms and did not require mechanical ventilation; and two children, patients no. 5 and no. 6 (both 10-year-old females, grandchildren of patients no. 1 and no. 3), who were admitted for observation. Patient no. 5 developed mild subjective symptoms without objective neurologic findings, and patient no. 6 remained asymptomatic.

The investigation revealed that the four botulism patients were part of a group of seven family members who had shared a meal on the evening of 10 September, which included a home-canned blend of carrots and green beans (referred to as ''blend''). The blend was ''briefly heated in a microwave'' before being served and was reported to have an unpleasant odor and taste by persons who ate it. Patient no. 1 (who reportedly ''did not like wasting food'') and patient no. 2 ate most of the blend; patient no. 3 handled the blend and had it on her plate, though she was unsure whether she had eaten any; and patient no. 4 ''tried a forkful'' of the blend but stopped because of the bad taste. Patients no. 5 and no. 6 reported having the blend on their plates, patient no. 5 may have eaten some, and patient no. 6 did not consume any. The seventh family member, who was asymptomatic, neither consumed the blend nor had it on her plate. The meal of

TABLE 1. *Clinical and microbiological features among botulism patients, Ohio and Washington, 2008 and 2009*

Outbreak		Patient characteristics					Laboratory findings (botulinum toxin detected in:)	
No.	Location	Clinical features	Hospitalization	Mechanical ventilation	Antitoxin prescribed	Implicated food	Clinical samples	Food
1	Ohio State	Patient 1: Nausea, vomiting, blurred vision, diplopia, dizziness, slurred speech, change in sound of voice, hoarseness, dry mouth, difficulty swallowing, shortness of breath, subjective weakness, bilateral neurologic signs (extraocular palsy, ptosis, facial paralysis)	Yes	Yes	Yes	Home-canned green bean and carrot blend	Serum from patients 1 and 2 and stool from patient 1	5 of 10 unopened jars of the carrot and green bean blend from same canning batch as the one from which food was consumed
		Patient 2: Nausea, vomiting, blurred vision, diplopia, dizziness, slurred speech, shortness of breath, subjective weakness, fatigue, bilateral neurologic signs (extraocular palsy, ptosis, facial paralysis)	Yes	Yes	Yes			
		Patient 3: Nausea, vomiting, blurred vision, diplopia, dizziness, slurred speech, change in sound of voice, dry mouth, difficulty swallowing, shortness of breath, subjective weakness, bilateral ptosis	Yes	Yes	Yes			
		Patient 4: Nausea, diarrhea, blurred vision, dizziness, difficulty swallowing	Yes	No	Yes			
		Patient 5: Subjective weakness	Yes (for observation)	No	No			
		Patient 6: Asymptomatic	Yes (for observation)	No	No			
2	Washington State	Patient 7: Nausea and vomiting, blurred vision, diplopia, dizziness, slurred speech, change in sound of voice, hoarseness, dry mouth, shortness of breath, subjective weakness, fatigue, bilateral neurologic signs (extraocular palsy, ptosis, facial paralysis, palatal weakness, impaired gag reflex)	Yes	Yes	Yes	Home-canned green beans	Serum and stool from patients 7 and 8	Leftover green beans
		Patient 8: Slurred speech, thick tongue, change in sound of voice, hoarseness, dysphagia, subjective weakness, bilateral palatal weakness	Yes	No	Yes			
		Patient 9: Diplopia, slurred speech, change in sound of voice, subjective weakness, bilateral neurologic signs (extraocular palsy, palatal weakness, impaired gag reflex)	Yes	No	Yes			
3	Washington State	Patient 10: Nausea, vomiting, blurred vision, diplopia, dizziness, slurred speech, thick tongue, hoarseness, dysphagia, shortness of breath, subjective weakness, fatigue, bilateral neurologic signs (extraocular palsy, ptosis, dilated pupils)	Yes	Yes	Yes	Home-canned asparagus	Serum and stool from patient 10	Residues obtained from jars containing the implicated asparagus
		Patient 11: Nausea, blurred vision, diplopia, dizziness, slurred speech, change in sound of voice, subjective weakness, fatigue, bilateral neurologic signs (extraocular palsy, ptosis, sluggish pupils)	Yes	Yes	Yes			
		Patient 12: Blurred vision, diplopia, dry mouth, fatigue	Yes	No	Yes			

10 September was the only shared meal among the ill family members, and none of the other foods during the meal were home canned.

C. botulinum toxin type A was detected in serum samples from patients no. 1 and no. 2 and in stool from patient no. 1. No leftover food remained from the jar of the blend consumed at the implicated meal, and the empty jar had been discarded. However, 10 unopened jars of the blend from the same canning batch were collected and tested. The jars were described by the laboratory as having ''effervescence like opening cans of soda'' with noticeable bubbles in several jars. Botulism toxin type A was detected in 5 of the 10 jars (Table 1).

The carrots and green beans for the blend were grown in the family's home garden and were canned by patient no. 1 approximately 3 weeks prior to consumption. The canning recipe was reportedly obtained from an ''at least 50-year-old edition'' of the *Better Homes and Garden Cookbook*. The canning recipe for both vegetables suggested use of ''pressure cooker'' with processing times of 20 and 25 min for pint- and quart-sized jars, respectively, at a pressure of 10 lb in^{-2} (68 kPa). However, patient no. 1 did not possess a pressure cooker and ''was told by an old farmer's wife that it was not needed.'' Instead, he used a hot water bath for 1 h. Canning supplies included glass canning jars, of unknown brand, which were secured from a family home in a nearby town. Several jars were reported to have seal failure with lid centers ''popped up'' after canning had occurred. Patient no. 1 had home-canned pickled beets on a few previous occasions and no other foods.

Outbreak 2: Washington State, January 2009. On 21 January 2009, a 37-year-old female (patient no. 7) reported to a hospital emergency room with nausea and vomiting. She was evaluated and discharged home, but she returned to the hospital on 22 January with additional paralytic signs and symptoms consistent with botulism (Table 1), and she required intubation later that day. On 23 January, patient no. 8 (9-year-old female, daughter of patient no. 7) and patient no. 9 (7-year-old female, daughter of patient no. 7) were also evaluated for paralytic signs and symptoms consistent with botulism (Table 1).

Three botulism patients from a family shared a meal with one other family member on the evening of 20 January, which included three types of home-canned products: green beans, tomatoes, and pears. The fourth member did not develop symptoms. Patients no. 8 and no. 9 commented that the green beans ''smelled like cat litter.'' Patient no. 7 did not note an unusual taste or smell and ate more green beans than the others, including most of her daughters' portions. The fourth member did not eat any green beans due to the ''suspicious smell'' but ate the other home-canned foods served during the meal. None of the other foods served were reported to have a suspicious appearance, odor, or taste.

Laboratory testing identified *C. botulinum* toxin Type A in serum and stool samples from patient no. 7 and patient no. 8 and from samples of the leftover green beans.

All home-canned foods consumed during the meal, including green beans, were grown in the family's home garden. About 20 jars of green beans were canned shortly after harvesting by patient no. 7 in her home, approximately 5 months before the implicated meal. Recipe and process details used for canning green beans, including whether or not a pressure cooker was used, were not available; however, patient no. 7 reported having shortened the recommended canning time due to lack of air-conditioning in the house. The jars of beans were stored at room temperature in the pantry, and the family had previously consumed other jars of green beans from the same canning batch. Limited cooperation by patients and family members precluded collection of additional details.

Outbreak 3: Washington State, June 2009. On 27 June 2009, two men (patient no. 10, a 54-year-old male, and patient no. 11, a 50-year-old male, friend of patient no. 10) sought care at a hospital in Tonasket, WA, with symptoms of nausea and progressively worsening blurred vision, double vision, and difficulty speaking (Table 1). After hospital admission, both patients developed respiratory distress and were intubated. On 28 June a third patient (patient no. 12, a 56-year-old male, brother of patient no. 10), also was evaluated at the hospital with blurred vision, mild dry mouth, and loss of appetite. His symptoms remained mild, and he did not need mechanical ventilation.

The three botulism patients identified in this outbreak had shared a meal on 26 June, which included home-canned asparagus. This was the only meal the three shared, and no others participated. The asparagus did not have an unusual odor, appearance, or taste when it was removed from the can, but several jar lids from the same canning batch were reported to have ''popped up.'' The asparagus was heated on a stove top (for unknown duration) before being served and consumed.

C. botulinum toxin type A was detected in serum and stool samples from patient no. 10 and from residues obtained from the jars containing the implicated asparagus.

The asparagus was a gift from a friend's home garden, had been home canned by patient no. 10's wife ~1 month before being eaten, and was stored at room temperature. Patient no. 10's wife reported that she was a novice home canner and had only one prior canning experience with pickled asparagus and salsa. Instructions for canning were obtained from *The American Woman's Cookbook*; the date of publication and edition could not be obtained. The canning recipe instructed cooking the asparagus in boiling water for 3 to 4 min, followed by processing immediately in a pressure cooker. However, patient no. 10's wife did not use a pressure cooker but instead covered and processed the jars in a hot water bath for 15 min (details about the hot water bath were not available). The canner was also not aware of the risk of botulism with improper home canning before this illness episode.

Antitoxin administration. A presumptive clinical diagnosis of botulism was made in each of these outbreaks, and the CDC released botulism antitoxin immediately. Botulism antitoxin Bivalent (Equine) Types A and B were administered to 10 patients after sensitivity testing (Ta-

ble 1). Two patients experienced adverse reactions: patient no. 12 developed hypotension, which required supportive measures for anaphylaxis and discontinuation of antitoxin administration, and patient no. 7 developed facial edema after antitoxin administration, which resolved with corticosteroids.

DISCUSSION

Home-canned vegetables remain the most important single cause of foodborne botulism outbreaks in the contiguous United States. In these three outbreaks of foodborne botulism, epidemiologic and laboratory investigations confirmed home-grown, improperly home-canned vegetables as the source of each outbreak. The home canners did not follow recipe instructions to use pressure cookers, which is necessary for safe home canning of low-acid vegetables such as green beans and asparagus *(30)*. Additionally, warning signs of food spoilage, including unpleasant odor or taste and/or popped lids, were unheeded in all three outbreaks. These findings indicate that awareness of the risk of botulism or other serious illness from improperly preserved vegetables was low among home canners and family and friends who consumed the implicated foods.

The recipes the three home canners used were obtained from cookbooks but then were adapted based on advice from family or friends and for personal convenience. Adherence to the available recipes would have been protective. Canners should refer to the most updated information regarding guidelines and recommendations for safe home canning *(30)*. In these outbreaks, the food was not cooked again after the jars were opened. While heating suspect food is not recommended to salvage it *(30)*, botulinum toxin is inactivated by heating to 85°C for at least 5 min *(8, 28)*. Microwave warming in the absence of stirring is often nonuniform, leaving cold spots where toxin would not be inactivated *(26)*.

The popularity of food gardening and home canning is increasing in the United States, a trend that is partly explained by economic forces as well as perceived health benefits of consuming home-grown foods *(2, 4, 5, 18, 20–22)*. In a national survey conducted in 2004, about one in five U.S. households canned food, and 65% of those canned vegetables *(2)*. Careful attention to safe canning practices is critical for all foods, but especially for pH-neutral vegetables like green beans, carrots, and asparagus that will permit toxin formation if they are contaminated with *C. botulinum* spores *(30)*. This includes using a pressure canner and adhering to the recommended processing times. Boiling and cooking home-canned foods (≥10 min) before eating them provides an additional margin of safety. Alarmingly, errors similar to those that led to the three botulism outbreaks reported here were commonly reported in a national survey of home canners in 2005 *(1, 2)*. Up to 57.5% of home canners used unsafe methods like oven canning, open-kettle canning, and boiling-water canning for preserving low-acid vegetables, rather than using a pressure canner or cooker; only 12% of respondents had the dial gauge on their pressure canner tested for accuracy; 32% reported having jars that did not seal properly after canning; and 12% served home-canned foods without reheating *(2)*. Family or friends were the source of canning information for 51% of respondents. Home-canned foods are commonly distributed to friends *(22)*, underscoring the risk of potentially widespread botulism outbreaks caused by unsafe home canning and food preparation practices.

Our investigations had several limitations. During the epidemiologic investigations some home canners were intubated, so some details about the food preparation process were obtained from family members, who had limited information on the specific home canning practices used; subsequent interviews were met with limited cooperation and yielded some inconsistencies. Detailed information about certain practices could not be obtained; for example, the exact microwave heating times and power settings were not available. Furthermore, the clinical data were limited to what is routinely collected during a public health investigation; thus, we did not track longer-term clinical outcomes such as total duration of ventilation, hospitalization, or symptoms.

These outbreaks highlight the importance of proper canning and food preparation practices and the severity of foodborne botulism for patients. Although we could not determine the exact duration of illness among patients associated with these outbreaks, reviews of the clinical severity of botulism are available elsewhere *(6, 24, 27)*. Patients have required ventilatory support for up to 7 months before the return of normal function *(8)*, and prolonged recovery and long-term outcomes have been documented *(14, 19)*. Physicians should consider the diagnosis of botulism in patients who present with rapid-onset cranial neuropathy and descending flaccid paralysis, especially with a history of consumption of home-canned foods. The occurrence of acute paralysis among two or more persons who shared a common meal strongly suggests botulism. All suspected cases of botulism should be immediately reported to the local and state health departments and to the CDC. The state health departments and CDC maintain a 24-h emergency consultation service for clinical diagnostic, epidemiologic, and diagnostic laboratory services. Emergency contact information for state and local health departments is available with the CDC Emergency Operations Center, which can be reached 24 h a day, 7 days a week, every day of the year at 770-488-7100. Botulinum antitoxin is available through the States of California and Alaska and the CDC.

Although relatively rare, hypersensitivity reactions can occur with the equine antitoxin, as was seen with two patients during our investigation. Since 13 March 2010 an investigational heptavalent antitoxin (HBAT, Cangene Corporation) has replaced all licensed bivalent botulinum antitoxin AB and an investigational monovalent botulinum antitoxin E (BAT-AB and BAT-E, Sanofi Pasteur) through a CDC-sponsored U.S. Food and Drug Administration Investigational New Drug protocol *(12)*. Investigational HBAT is composed of Fab and $F(ab')_2$ immunoglobulin fragments, contains <2% of intact immunoglobulin com-

ponents, and may cause fewer allergic reactions. However, preliminary data about the frequency of adverse events among investigational HBAT recipients are still being collected.

These outbreaks and national surveys of home canners identify critical areas of concern in current home canning and food preparation practices. Public health authorities should be alert for possible botulism cases and swiftly investigate them. Information with links to approved canning practices and warnings about the risk of botulism can be targeted directly to home canners, for example, thorough improved labeling of home canning supplies and the addition of information about botulism to cookbooks and Web sites about home canning. The most updated 2009 U.S. Department of Agriculture guidelines for safe home canning can be found at http://www.uga.edu/nchfp/publications/publications_usda.html. Foods with evidence of spoilage should be discarded. Additionally, all low-acid and tomato foods should be boiled in a saucepan for 10 min or longer based on altitudes, unless the preparer is sure that up-to-date, U.S. Department of Agriculture–recommended canning procedures were followed and there is no evidence of spoilage *(30)*. More assessment of the prevalence of home canning practices in the United States and of the information sources most frequently used by home canners would be useful to further shape public health interventions.

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State of Alaska Epidemiology **Bulletin**

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Bulletin No. 20 October 8, 2008

Campylobacteriosis Outbreak due to Consumption of Raw Peas — Alaska, 2008

Introduction

Campylobacter jejuni, a spiral-shaped bacteria, is one of the most common causes of diarrheal illness in the United States. Campylobacteriosis is characterized by diarrhea (frequently bloody), abdominal pain, malaise, fever, nausea and/or vomiting. Symptoms occur 2–10 days after infection and can persist for weeks. The vast majority of cases occur as isolated, sporadic events, rather than part of recognized outbreaks. On August 21, the Section of Epidemiology (SOE) was notified of a possible outbreak of campylobacteriosis in Southcentral Alaska. In collaboration with the Anchorage Department of Health and Human Services and the Alaska Department of Environmental Conservation (DEC), we began an immediate investigation.

Methods

Epidemiologic Investigation

A case-control study was performed to determine risk factors for illness. A case was defined as laboratory-confirmed *C. jejuni* infection diagnosed from August 1 thru September 26, 2008 in a person living in Southcentral Alaska. Two asymptomatic controls were selected for each case, matched on age-group (0–1, 2–17, 18–64, and ≥65 years) and location by progressive and sequential random-digit dialing anchored on the case-patient's telephone number. A questionnaire was administered by telephone or in person to case-patients and controls, and demographic information, illness characteristics, food and environmental exposures were recorded. Odds ratios were calculated for risk factors. Active surveillance was initiated to identify additional clinical cases, defined as a person with new onset of ≥3 episodes of watery diarrhea in a 24-hour period within 10 days of consuming raw peas grown in Alaska.

Environmental Investigation

Food suspected to have contributed to the outbreak was traced back to the primary producer. Disinfection and food processing practices were reviewed, and food, processing surfaces, and environmental samples were obtained.

Laboratory Investigation

Stool samples were collected from case-patients and forwarded to the Alaska State Public Health Laboratory (ASPHL); environmental samples were sent to Silliker Labs in Ohio and to the U.S. Centers for Disease Control and Prevention Campylobacter Reference Laboratory for analysis. Pulsed-field gel electrophoresis (PFGE) by SmaI and KpnI enzymes was performed on all available *C. jejuni* isolates.

Results

Epidemiologic Investigation

Sixty-three case-patients and 126 controls were enrolled in the case-control study. Seventy-six percent of case-patients (n=48) reported eating fresh peas versus 31% of controls (n=39). In multivariate analysis, only eating raw, shelled peas remained associated with illness (odds ratio: 24.8; 95% confidence interval: 10.9–56.4). To date, 54 persons with laboratory-confirmed and 45 with clinical campylobacteriosis reported eating peas within 10 days of illness onset (Figure). Ages ranged from 1–79 years (median: 47), and 76% were Anchorage or Eagle River residents. Five were hospitalized and one developed Guillain-Barré syndrome 9 days after symptom onset; none died.

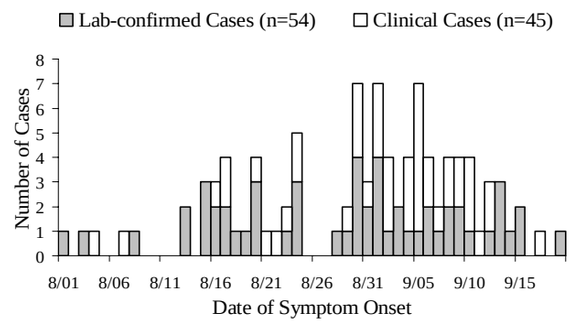
Environmental Investigation

Sanitarians traced peas consumed by several case-patients back to Farm A, the only pea farm and processor in Alaska. Several deficiencies were noted in the processing of peas at Farm A, including a lack of chlorine residual in pea-processing water. Sandhill Cranes were seen grazing in the pea fields in high numbers. From September 6–15, 42 environmental samples were collected from Farm A harvesting and processing equipment, water, produce, fertilizer, clothing, animal feces and product.

Laboratory Investigation

Campylobacter jejuni isolates from 42 ill persons were characterized by PFGE; 28 unique clinical PFGE patterns were identified. Sixteen environmental samples were positive for *C. jejuni*; 14 were from Sandhill Crane stool, one was from a mound of peas located near the pea processing building, and one was from freshly picked field peas. Fifteen unique environmental PFGE patterns were identified. Four of the clinical PFGE patterns (representing 15 ill persons) were indistinguishable from four of the environmental PFGE patterns (obtained from two of the Sandhill Crane stool samples and the two pea samples).

Figure. Campylobacter Cases Linked to Consumption of Peas — Alaska, 2008 (N=99)



Discussion

This investigation has established a firm linkage between *C. jejuni* infection and consumption of Farm A peas. Based on the molecular analysis of *C. jejuni* isolates from clinical and environmental samples, the source of contamination of the peas appears to be Sandhill Crane feces. Sanitarians identified a lack of chlorine residual in pea-processing water, suggesting that *C. jejuni* from crane feces picked up during harvesting likely contaminated shelled peas before packaging. Farm A voluntarily shut down operations on September 12 and local retailers removed remaining locally grown peas from their shelves. State officials are working with the farmer to implement future control measures.

Guillain-Barré syndrome is often preceded by *C. jejuni* infection and is associated with slow recovery and severe residual disability.[3]

Recommendations

1. Raw vegetables should be cooked or carefully washed prior to consumption.
2. Health care providers should consider testing for *Campylobacter* in any patient that presents with clinically compatible symptoms, and report cases to the Section of Epidemiology (call 907-269-8000).
3. Health care providers should advise patients with campylobacteriosis to drink extra fluids as long as diarrhea lasts. In more severe cases, providers should consider administering antibiotics such as erythromycin or a fluoroquinolone.

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State of Alaska Epidemiology Bulletin

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Ongoing Raw Milk *Campylobacter* Outbreak — Southcentral Alaska, July 2011

Background

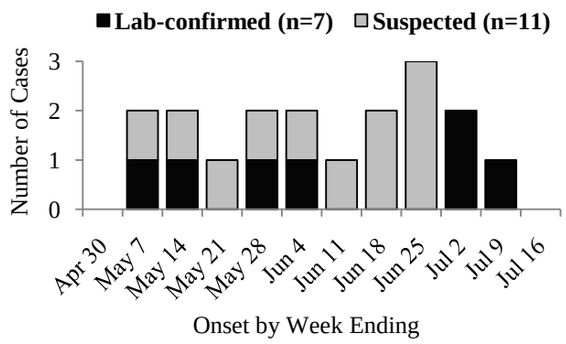
On June 27, 2011, the Alaska Section of Epidemiology (SOE) released an Epidemiology *Bulletin* detailing an outbreak of campylobacteriosis associated with the consumption of raw milk from Farm A in the Mat-Su Valley.[1] This follow-up *Bulletin* provides updated information about the investigation.

Active Case finding

On June 24, SOE distributed an Advisory through the Public Health Alert Network (PHAN) to alert health care providers of the outbreak and to recommend testing for *Campylobacter* in patients who present with acute gastrointestinal (GI) illness and a history of raw milk consumption. Through the PHAN, the June 27 *Bulletin*, and associated press releases, members of the public were also asked to contact SOE and report acute GI illness following consumption of raw milk. SOE received calls from five community members reporting current and previous GI illness among persons in their households with a preceding history of consuming Farm A raw milk or cream. Public health nurses facilitated collection of stool specimens from recently ill persons for enteric bacterial pathogen testing at the Alaska State Public Health Laboratory (ASPHL).

Stool specimens were collected from six persons with recent GI illness and consumption of Farm A raw dairy products. Three of the six samples tested positive for *Campylobacter jejuni*; all isolates were the same rare strain of *C. jejuni* found in the four other laboratory-confirmed cases in this outbreak (pulsed-field gel electrophoresis [PFGE] pattern AKDBRS16.0166/AKDBRK02.0093). These three persons shared raw dairy products obtained from Farm A during the first week of July. A total of 11 persons who reported acute GI illness with routine consumption of Farm A dairy products but were not-laboratory confirmed were considered to have suspected campylobacteriosis (Figure).

Figure. Cases of *Campylobacter jejuni* Infection associated with Consumption of Farm A Raw Milk, by Onset Date — Southcentral AK, May–July 2011



Environmental and Milk Testing Results

Eleven composite cow manure samples, one composite chicken manure sample, and one swab from the milking parlor drain were collected on June 22. ASPHL cultured the samples with guidance from the Centers for Disease Control and Prevention (CDC) *Campylobacter* Laboratory. Any culture suspicious for *Campylobacter* growth had up to six colonies chosen and plated for identification. From the 13 samples collected at the farm and submitted for culture, 18 separate colonies were identified as *C. jejuni* and underwent PFGE analysis; seven different PFGE patterns were identified. PFGE pattern AKDBRS16.0166/AKDBRK02.0093 was isolated in manure samples from the grazing field and the calf barn; this pattern was identical to the pattern of the *C. jejuni* isolated from the seven laboratory-confirmed patients.

Farm A bulk tank milk samples collected on June 22 and 27 tested negative for *C. jejuni*, but positive for *Listeria monocytogenes*. None of the Farm A raw milk that was actually consumed by ill persons was available for testing.

Discussion

The identical rare strain of *C. jejuni* has been identified in all laboratory-confirmed patients associated with this outbreak and Farm A cow manure specimens. These laboratory findings combined with the epidemiologic finding that Farm A raw dairy product consumption is the only exposure common to all seven laboratory-confirmed cases (and the 11 suspect cases) affirms the conclusion that this outbreak is due to consumption of Farm A raw dairy products. Contamination might have resulted from introduction of manure into the milk or cream at some point in time from milking to filling the containers, or a cow (or cows) with an infected udder may be intermittently shedding *Campylobacter* directly into the milk. Regardless of the exact mechanism of contamination, with confirmed cases reporting consumption of dairy products over an 8-week period from May to July, this outbreak poses an *ongoing threat to Farm A raw dairy product consumers.*

It is not surprising that *C. jejuni* was not detected in Farm A bulk tank samples because *C. jejuni* is notoriously difficult to culture from environmental specimens other than raw stool,[2] and few campylobacteriosis outbreak investigations yield laboratory confirmation of an implicated food source such as raw milk or produce.[3] Furthermore, none of the raw milk that was actually consumed by ill persons prior to their illness onset was available for testing. Numerous *C. jejuni* strains were detected on Farm A, which was anticipated given that many farm animals are known reservoirs for the bacteria. Finding only a single or predominant strain shared by the human cases is not unusual, and might relate to factors associated with seasonality or adaptation of the strain to humans.[4] Finally, as was the case in May, the Farm A raw milk samples collected in June tested positive for *L. monocytogenes*, which can cause life-threatening meningitis in children and persons with compromised immune systems.

Recommendations

1. Health care providers should be aware that this *C. jejuni* outbreak is ongoing and should collect stool specimens for enteric bacterial pathogen testing on all persons with acute GI illness and a recent history of raw dairy product consumption. ASPHL offers free testing; collection guidelines are available at: http://www.hss.state.ak.us/dph/labs/publications/image/Lab_Svcs_Manual.pdf
2. Health care providers are required to report all clinical and laboratory-confirmed cases of *Campylobacter* infection (7 AAC 27.005). Please call 907-269-8000 Mon–Fri 8AM to 5PM, or 907-561-1324 or 800-478-1700 if calling after hours or from outside of Anchorage.
3. Providers should educate their patients about the potential serious risks of raw dairy product consumption. Educational materials are available on-line.[5]

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