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Agent ¹	Usual Incubation Period (Range) ²	Symptom Profile ^{2,3}	Duration of Illness ²	Period of Communicability	Characteristic Foods ^{3,4}	Criteria for Confirmation in an Outbreak Setting ************************************
I. Agen	ts typified by	nausea and vo	miting, <i>witho</i>	<i>ut</i> fever, within 8 ho	urs of eating	
Bacillus cereus ("emetic variety")	2–4 hours (1–6 hours)	Vomiting, with nausea and diarrhea (abrupt onset)	24 hours	Not communicable (preformed enterotoxin). N.b., emetic toxin is heat resistant.	Cooked rice, meats, vegetables.	Isolation of 10 ⁵ organisms per gram from stool or two or more ill persons OR isolation of 10 ⁵ organisms per gram of epidemiologically implicated food. ***********************************
Staphylococcus aureus	2–4 hours (30 minutes – 8 hours)	Vomiting, with nausea, cramps, and diarrhea (abrupt onset)	24–48 hours	Not communicable (preformed enterotoxin) N.b., emetic toxin is heat resistant.	Sliced or chopped ham and meats, custards, cream fillings, mushrooms, egg salad	Isolation of organism from stool or vomitus from two or more ill persons OR detection of enterotoxin in epidemiologically implicated food OR isolation of 10 ⁵ organisms per gram of epidemiologically implicated food. ***********************************
		Agents typ	oified by diarr	hea and abdominal	cramps, without	<i>t</i> fever, within 24 hours of eating
<i>Bacillus cereus</i> ("diarrheal variety")	8–16 hours	Cramps and diarrhea	~24 hours	Not communicable (enterotoxin formed <i>in vivo</i>)	Custards, cereals, puddings, sauces, meatloaf	Isolation of 10 ⁵ organisms per gram from stool or two or more ill persons and not from stool of control patients OR isolation of 10 ⁵ organisms per gram of epidemiologically implicated food. Testing not performed at OSPHL. Contact ACDP for testing options.

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Clostridium perfringens	10–12 hours (6 – 24 hours)	Cramps and diarrhea	Up to 24 hours	Not communicable (enterotoxin formed <i>in vivo</i>)	Meat, poultry, gravy, Mexican foods	Isolation of 10 ⁵ organisms per gram from stool or two or more ill persons OR demonstration of enterotoxin in the stool of two or more ill persons OR isolation of 10 ⁵ organisms per gram of epidemiologically implicated food. Testing not performed at OSPHL. Contact ACDP for testing options. A loss of viability of <i>C. perfringens</i> will occur if foods are frozen or held under prolonged refrigeration.
	III.	Agents typ	ified by diarr	hea and abdominal	cramps, with fev	ver, within 12–48 hours of eating
Campylobacter jejuni	2–5 days (1–10 days)	Cramps and diarrhea (sometimes bloody), with vomiting and fever	48 hours – 10 days	2–7 weeks	Raw milk, poultry and poultry products, liver parfait and pâté, contaminated water, young cats, dogs and livestock	Isolation of organism from clinical specimens from two or more ill persons OR isolation of organism from epidemiologically implicated food. ***********************************
Escherichia coli Enteroinvasive (EIEC)	12–48 hours	Cramps and diarrhea, with fever, headache	5–10 days	Weeks to months	Uncooked vegetables, salads, water, cheese	Isolation of same enteroinvasive serotype from stool of two or more ill persons. Testing may be available at OSPHL upon special approval from ACDP. Contact ACDP to discuss.

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<i>Salmonella</i> (non- typhoid)	29–54 hrs (1–5 days) [According to ACDP] 12–36 hrs (6 hours – 10 days) [according to everyone else]	Cramps and diarrhea, with vomiting and fever	4–7 days	Several days to several years, depending on type. Concentrations/in fectivity typically higher when symptomatic	Poultry, eggs, meat, raw tuna, raw milk or milk products, sprouts, other produce, raw nuts and nut butters, spices (cross- contamination important)	Isolation of organism of same serotype from clinical specimens from two or more ill persons OR isolation of organism from epidemiologically implicated food. ***********************************
				abdominal cramps,		n 12–48 hours of eating (continued)
Shigella	1-3 days (12 hours – 7 days)	Cramps and diarrhea (may be bloody), with fever	2–7 days	4 weeks after illness	Eggs, salads, lettuce, infected food handlers, soiled diapers, direct oral-anal contact, foreign travel	Isolation of organism of same serotype from clinical specimens from two or more ill persons OR isolation of organism from epidemiologically implicated food. ***********************************

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Agent ¹	Usual Incubation Period (Range) ²	Symptom Profile ^{2,3}	Duration of Illness ²	Period of Communicability	Characteristic Foods ^{3,4}	Criteria for Confirmation in an Outbreak Setting
Vibrio parahaemolyticus and other noncholerae vibrios	12–24 hours (4–96 hours)	Cramps and watery diarrhea, sometimes with nausea, vomiting	1 –7 days (according to Orpheus, up to 3 weeks)	Not communicable	Seafood, especially oysters and other shellfish; occasionally salmon	Isolation of organism from stool of two or more ill persons.

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	III. Agents typified by diarrhea and abdominal cramps, with fever, within 12–48 hours of eating (continued)										
Yersinia enterocolitica	35–48 hours (24 hours – 10 days)	Cramps, diarrhea, fever, headache, vomiting, pseudo- appendicitis	1–3 weeks	2–3 weeks	Raw or undercooked pork and pork products such as chitterlings, milk, tofu, farm animals, dogs	Isolation of organism of same serotype from clinical specimens from two or more ill persons OR isolation of organism from epidemiologically implicated food. ***********************************					

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						ache with fever, within 24 hours of eating
Listeria monocytogenes	18–31 hours	Fever, with diarrhea, myalgia, and headache.	Days to weeks	Not known	Inadequately pasteurized milk, lunch meats and cold cuts, unpasteurized soft cheeses, sprouts, and pâtés	Isolation of <i>Listeria monocytogenes</i> of the same subtype from two or more ill persons exposed to the epidemiologically implicated food or to food from which the same subtype of <i>Listeria monocytogenes</i> has been isolated. ************************************

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		ents typified by	/ vomiting, di			hout fever, within 24–48 hours of eating
Norwalk virus and other caliciviruses	Typically 24–48 hours (10–72 hours)	Vomiting, with diarrhea, headache, and myalgia. Usual symptom profile: Diarrhea: 80% Vomiting: 60% Nausea: 75% Fever: 30%	24–72 hours	Throughout the period of vomiting and diarrhea and 2–3 days after symptoms end	Shellfish, water, salads, frosting, "handled" foods.	Detection of viral RNA in stool or vomitus by reverse transcriptase-polymerase chain reaction (RT-PCR) from two or more persons. ************************************
	VI.	Agents typ	ified by water	ry diarrhea and hea	dache <i>without</i> fe	ver, within 24–48 hours of eating
Cyclospora cayetanensis	7 days (1- 11 days)	Watery diarrhea, fatigue, protracted diarrhea, often relapsing	Several weeks to a month	Not communicable	Water, uncooked food, raw produce	Detection of <i>Cyclopora</i> organisms or DNA in stool, intestinal fluid/aspirate or intestinal biopsy specimens. ************************************

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	VI. Age	ents typified by	watery diarrh	ea and headache w	ithout fever, with	nin 24–48 hours of eating (continued)
Escherichia coli Enterotoxigenic (ETEC) ⁵	10–72 hours (10 hours – 7 days)	Cramps, profuse watery diarrhea, some vomiting	3–4 days; up to 3 weeks	Weeks to months	Food and water, produce, seafood, sushi	Isolation of organism of same serotype demonstrated to produce heat-stable (ST) or heat labile (LT) enterotoxin from stool of two or more ill persons. ************************************
Vibrio cholerae O1 and O139	2–3 days (several hours – 5 days)	Profuse watery diarrhea and vomiting, which can lead to severe dehydration and death within hours	72 hours – 7 days; causes life- threatening dehydratio n	Usually a few days after recovery except when in a carrier state	Shellfish, water, "street food," foods contaminated by infected food handlers	Isolation of toxigenic organism from stool or vomitus of two or more ill persons OR significant rise in vibriocidal, bacterial-agglutinating, or anti-toxin antibodies in acute and early convalescent phase sera among persons not recently immunized OR isolation of toxigenic organism from epidemiologically implicated food. ***********************************

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		VII. Ag	ents typified	by bloody diarrhea	a <i>without</i> fever, w	vithin 48 hours of eating
Escherichia coli Shiga-toxin- producing (STEC; Entero- hemorrhagic: <i>E. coli</i> O157:H7 and others)	48 hours – 8 days (24 hours – 10 days)	Bloody diarrhea with cramps, vomiting; hemolytic uremic syndrome (2%–7 % of cases)	5–10 days	1–4 weeks	Beef, venison, raw milk, sprouts, water, leafy greens, other produce, unpasteurized apple cider.	Isolation of <i>E. coli</i> O157:H7 or other shiga-toxin-producing <i>E. coli</i> from clinical specimens from two or more ill persons OR isolation of <i>E. coli</i> O157:H7 or other STEC from epidemiologically implicated food. ***********************************
						Food testing not performed at OSPHL. Contact ACDP for testing options.
						with fever, within 15–50 days of eating
Hepatitis A	28–30 days (15– 50 days)	Fever, malaise, nausea, abdominal pain, jaundice. Children <5 are typically asymptomatic	Weeks to months	2 weeks before symptom onset and 1 week after onset of jaundice	Raw or undercooked shellfish from contaminated waters, raw produce, water, foods contaminated by an infected food handler	Collect and submit 1.5 mL serum or 5mL whole blood in a red top or serum separator tube (SST). Store and transport at refrigerated temperatures for receipt at OSPHL less than 7 days after collection. Submit with OSPHL Form 42, Virology/Immunology Test Request Form. ************************************

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Clostridium botulinum	12–72 hours (2 hours – 8 days)	Double vision, eyelid drooping, and descending paralysis, sometimes accompanied or preceded by nausea, vomiting, or diarrhea	Days to months	IX. Botulisr Not communicable (preformed enterotoxin)	n (foodborne) Improperly canned or similarly preserved foods; fermented fish ("stink heads"); honey (infants)	Detection of botulinum toxin in serum, stool, gastric contents, or implicated food OR isolation of organism from stool or intestine

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		X. Agent	s most readi	ly diagnosed from t	he history of eat	ing a particular type of food
Heavy metals (antimony, arsenic, cadmium, copper, iron, lead, mercury, tin, zinc)	5 minutes – 8 hours (usually <1 hour)	Vomiting, with nausea, cramps, and diarrhea, vision impairment, muscle weakness, other neurological symptom	Usually self-limited	Not communicable	Acidic foods and beverages prepared, stored, or cooked in containers coated, lined, or contaminated with offending metal, environmental exposure, rice or produce grown in contaminated soil.	Demonstration of high concentration of metal in epidemiologically implicated food. ***********************************
Poisonous mushrooms	<2 hours	Vomiting, diarrhea, confusion, visual disturbances, salivation, diaphoresis, hallucinations , disulfiram- like reaction	Usually self-limited	Not communicable	Wild mushrooms	Clinical syndrome among persons who have eaten mushrooms identified as toxic type OR demonstration of toxin in epidemiologically implicated mushrooms or food containing mushrooms. ************************************

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	Х.		eadily diagno	sed from the histor		ticular type of food (continued)
Shellfish poisoning (diarrheic, neurotoxic, amnesic) ⁴	20 minutes – 3 hours	Cramps, diarrhea, headaches, vomiting, amnesia, seizures, tingling or numbness of lips and throat, can cause death	Days	Not communicable	Mussels, oysters, scallops, razor clams, squid, anchovy	Detection of toxin in epidemiologically implicated food OR detection of large numbers of shellfish-poisoning- associated species of dinoflagellates in water from which epidemiologically implicated mollusks are gathered. ************************************
Ciguatera poisoning⁴	1–6 hours (usually within 6 hours)	Diarrhea, nausea, vomiting, paresthesias, sensitivity of extreme temperatures , arrhythmia	Days to weeks to months	Not communicable	Large, tropical ocean fish (grouper, amberjack, barracuda, snapper)	Demonstration of ciguatoxin in epidemiologically implicated fish OR clinical syndrome among persons who have eaten a type of fish previously associated with ciguatera fish poisoning. ************************************
Scombroid fish poisoning (histamine fish poisoning) ^{4,5}	1 minute – 3 hours (usually within 6 hours)	Facial / trunk flushing, heart palpitations, nausea, vomiting, diarrhea, food tasting "peppery" or "metallic"	6–12 hours	Not communicable	Tuna-like fish (mahi-mahi, tuna, mackerel, bluefish, salmon, bonito, skipjack)	Demonstration of histamine in epidemiologically implicated fish OR clinical syndrome among persons who have eaten a type of fish previously associated with histamine fish poisoning (especially fish of the <i>Scombridae</i> family). ************************************

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	Х.	Agents most re	eadily diagno	sed from the histor	y of eating a part	ticular type of food (continued)
Paralytic shellfish poisoning⁴	30 minutes – 3 hours	Paresthesias, feeling of floating, loss of balance, dry mouth, diplopia, dysarthria, shortness of breath, respiratory paralysis; death is possible	Days	Not communicable	Clams, mussels, cockles, oysters, scallops	Demonstration of toxin in epidemiologically implicated fish OR detection of large numbers of shellfish-poisoning- associated species of dinoflagellates in water from which epidemiologically implicated mollusks are gathered. Collect epidemiologically implicated shellfish. ************************************

Acknowledgments and references

- 1. The OHA Compendium of Acute Food-borne Diseases is based on a similar table developed by epidemiologists at the Foodborne and Diarrheal Diseases Branch, Division of Bacterial and Mycotic Diseases, National Center for Infectious Diseases, Centers for Disease Control and Prevention
- 2. Heymann DL. Control of communicable diseases manual. 20th ed. Washington, DC: American Public Health Association, 2015.
- Symptom profiles and characteristic foods are taken from Dalton CB, Mintz ED, Wells JG et al. Outbreaks of enterotoxigenic *Escherichia coli* infection in American adults: a clinical and epidemiologic profile. *Epidemiol Infect* 1999; 123:9–16. "Characteristic foods" for each FBD agent are based on epidemiological data gathered by epidemiologists in the Acute and Communicable Disease Prevention Section, Center for Public Health Practice, Public Health Division, Oregon Health Authority;
- 4. Food and Drug Administration. Bad Bug Book, Foodborne Pathogenic Microorganisms and Natural Toxins. Second Edition. Foodborne Pathogenic Microorganisms and Natural Toxins. 2012.
- 5. Feng C, Teuber S, Gershwin ME. Histamine (Scombroid) Fish Poisoning: a Comprehensive Review. Clin Rev Allergy Immunol 2016;50:64–9.

Note: Use laboratory submission instructions with caution. Current criteria are posted at www.healthoregon.or/labtests.

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Revision History

November 2018 – Updated to reflect testing no longer performed at OSPHL for the following pathogens: *Bacillus cereus*, *Staphylococcus aureus*, *Clostridium perfringens*, and *Cyclospora cayetanensis* (Poissant).

February 2018 – updated formatting, references, lab submission instructions, OSPHL address, added hepatitis A, updated disease facts (Poissant)

August 2008 - Compendium updated (Lee)

August 2001 – Compendium adapted from CDC table (Cieslak, Lee)



Recommendations and Reports

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Diagnosis and Management of Foodborne Illnesses

A Primer for Physicians and Other Health Care Professionals

INSIDE: Continuing Education Examination

DEPARTMENT OF HEALTH AND HUMAN SERVICES CENTERS FOR DISEASE CONTROL AND PREVENTION

MMWR

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Disclosure of Relationship

Elaine F. Brainerd, R.N., M.A., has indicated that she has a financial relationship with CDC because she is the Director of a Food Safe Schools project that is funded under a cooperative agreement by CDC. The remaining preparers have signed a conflict of interest disclosure form that verifies no conflict of interest.

Diagnosis and Management of Foodborne Illnesses A Primer for Physicians and Other Health Care Professionals

Produced collaboratively by the American Medical Association American Nurses Association–American Nurses Foundation Centers for Disease Control and Prevention Center for Food Safety and Applied Nutrition, Food and Drug Administration Food Safety and Inspection Service, US Department of Agriculture

Preface

Foodborne illness is a serious public health problem. CDC estimates that each year 76 million people get sick, more than 300,000 are hospitalized, and 5,000 die as a result of foodborne illnesses. Primarily the very young, the elderly, and the immunocompromised are affected. Recent changes in human demographics and food preferences, changes in food production and distribution systems, microbial adaptation, and lack of support for public health resources and infrastructure have led to the emergence of novel as well as traditional foodborne diseases. With increasing travel and trade opportunities, it is not surprising that now there is a greater risk of contracting and spreading a foodborne illness locally, regionally, and even globally.

Physicians and other health care professionals have a critical role in the prevention and control of food-related disease outbreaks. This primer is intended to provide practical and concise information on the diagnosis, treatment, and reporting of foodborne illnesses. It was developed collaboratively by the American Medical Association, the American Nurses Association-American Nurse Foundation, CDC, the Food and Drug Administration's Center for Food Safety and Nutrition, and the United States Department of Agriculture's Food Safety and Inspection Service.

Clinicians are encouraged to review the primer and participate in the attached continuing medical education (CME) program.

Background

This primer is directed to primary care and emergency physicians, who are likely to see the index case of a potential foodrelated disease outbreak. It is also a teaching tool to update physicians and other health care professionals about foodborne illness and remind them of their important role in recognizing suspicious symptoms, disease clusters, and etiologic agents, and reporting cases of foodborne illness to public health authorities.

Specifically, this guide urges physicians and other health care professionals to

- Recognize the potential for a foodborne etiology in a patient's illness;
- Realize that many but not all cases of foodborne illness have gastrointestinal tract symptoms;

An earlier edition of this Primer, covering different foodborne illnesses, was published in *MMWR* in 2001 (MMWR 2001;50[No. RR-2]) and also as a separate publication by the American Medical Association, CDC, the Food and Drug Administration, and the U.S. Department of Agriculture. This report updates and supplements the previous edition. It is being reprinted here as a courtesy to the collaborating agencies and the MMWR readers.

- Obtain stool cultures in appropriate settings, and recognize that testing for some specific pathogens, eg, *E. coli* O157:H7, *Vibrio* spp., must be requested;
- Report suspect cases to appropriate public health officials;
- Talk with patients about ways to prevent food-related diseases; and
- Appreciate that any patient with foodborne illness may represent the sentinel case of a more widespread outbreak.

Foodborne illness is considered to be any illness that is related to food ingestion; gastrointestinal tract symptoms are the most common clinical manifestations of foodborne illnesses. This document provides detailed summary tables and charts, references, and resources for health care professionals. Patient scenarios and clinical vignettes are included for selfevaluation and to reinforce information presented in this primer. Also included is a CME component.

This primer is not a clinical guideline or definitive resource for the diagnosis and treatment of foodborne illness. Safe food handling practices and technologies (eg, irradiation, food processing and storage) also are not addressed. More detailed information on these topics is available in the references and resources listed in this document, as well as from medical specialists and medical specialty societies, state and local public health authorities, and federal government agencies.

For additional copies, please contact

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Or visit the following websites:

The American Medical Association http://www.ama-assn.org/go/foodborne Centers for Disease Control and Prevention http://www.cdc.gov/foodsafety/cme.htm Center for Food Safety and Applied Nutrition, Food and Drug Administration http://www.cfsan.fda.gov Food Safety and Inspection Service, US Department of Agriculture http://www.fsis.usda.gov

Clinical Considerations

Food-related disease threats are numerous and varied, involving biological and nonbiological agents. Foodborne illnesses can be caused by microorganisms and their toxins, marine organisms and their toxins, fungi and their related toxins, and chemical contaminants. During the last 20 years, some foods that have been linked to outbreaks include milk (*Campylobacter*); shellfish (noroviruses); unpasteurized apple cider (*Escherichia coli* O157:H7), raw and undercooked eggs (*Salmonella*); fish (ciguatera poisoning); raspberries (*Cyclospora*); strawberries (hepatitis A virus); and ready-to-eat meats (*Listeria*).

While physicians and other health care professionals have a critical role in surveillance for and prevention of potential disease outbreaks, only a fraction of the people who experience gastrointestinal tract symptoms from foodborne illness seek medical care. In those who do seek care and submit specimens, bacteria are more likely than other pathogens to be identified as causative agents. Bacterial agents most often identified in patients with foodborne illness in the United States are *Campylobacter, Salmonella*, and *Shigella* species, with substantial variation occurring by geographic area and season. Testing for viral etiologies of diarrheal disease is rarely done in clinical practice, but viruses are considered the most common cause of foodborne illness.

This section and the accompanying Foodborne Illnesses Tables summarize diagnostic features and laboratory testing for bacterial, viral, parasitic, and noninfectious causes of foodborne illness. For more specific guidance, consult an appropriate medical specialist or medical specialty society, as well as the various resources listed in this primer. Also refer to this section and the accompanying Foodborne Illnesses Tables when working through the various Patient Scenarios and the Clinical Vignettes portion of this primer.

Recognizing Foodborne Illness

Patients with foodborne illnesses typically present with gastrointestional tract symptoms (eg, vomiting, diarrhea, abdominal pain); however, nonspecific symptoms and neurologic symptoms may also occur. Every outbreak begins with an index patient who may not be severely ill. A physician or health care professional who encounters this person may be the only one with the opportunity to make an early and expeditious diagnosis. Thus, the physician or health care professional must have a high degree of suspicion and ask appropriate questions to recognize that an illness may have a foodborne etiology.

Important clues to determining the etiology of a foodborne disease are the

- Incubation period;
- Duration of the resultant illness;
- Predominant clinical symptoms; and
- Population involved in the outbreak.

Additional clues may be derived by asking whether the patient has consumed raw or poorly cooked foods (eg, raw or undercooked eggs, meats, shellfish, fish), unpasteurized milk or juices, home-canned goods, fresh produce, or soft cheeses made from unpasteurized milk. Inquire as to whether any of the patient's family members or close friends have similar symptoms. Inquiries about living on or visiting a farm, pet contact, day care attendance, occupation, foreign travel, travel to coastal areas, camping excursions to mountains or other areas where untreated water is consumed, and attendance at group picnics or similar outings also may provide clues for determining the etiology of the illness.

If a foodborne illness is suspected, submit appropriate specimens for laboratory testing and contact the state or local health department for advice about epidemiologic investigation. For the physician or other health care professional, implication of a specific source in disease transmission is difficult from a single patient encounter. Attempts to identify the source of the outbreak are best left to public health authorities.

Because infectious diarrhea can be contagious and is easily spread, rapid and definitive identification of an etiologic agent may help control a disease outbreak. Early identification of a case of foodborne illness can prevent further exposures. An individual physician who obtains testing can contribute the clue that ultimately leads to identification of the source of an outbreak.

Finally, health care professionals should recognize that while deliberate contamination of food is a rare event, it has been documented in the past. The following events may suggest that intentional contamination has occurred: an unusual agent or pathogen in a common food, a common agent or pathogen affecting an unusually large number of people, or a common agent or pathogen that is uncommonly seen in clinical practice, as might occur with pesticide poisoning.

Diagnosing Foodborne Illnesses

Differential Diagnosis

As shown in Table 1 and the Foodborne Illnesses Tables, a variety of infectious and noninfectious agents should be considered in patients suspected of having a foodborne illness. Establishing a diagnosis can be difficult, however, particularly in patients with persistent or chronic diarrhea, those with severe abdominal pain, and when there is an underlying dis-

Clinical presentation	Potential food-related agents to consider
Gastroenteritis (vomiting as primary symptom; fever and/or diarrhea also may be present)	Viral gastroenteritis, most commonly rotavirus in an infant or norovirus and other caliciviruses in an older child or adult; or food poisoning due to preformed toxins (eg, vomitoxin, <i>Staphylococcus aureus</i> toxin, <i>Bacillus cereus</i> toxin) and heavy metals.
Noninflammatory diarrhea (acute watery diarrhea without fever/dysentery; some patients may present with fever)*	Can be caused by virtually all enteric pathogens (bacterial, viral, parasitic) but is a classic symptom of Enterotoxigenic <i>Escherichia coli</i> <i>Giardia</i> <i>Vibrio cholerae</i> Enteric viruses (astroviruses, noroviruses and other caliciviruses, enteric adenovirus, rotavirus) <i>Cryptosporidium</i> <i>Cyclospora cayetanensis</i>
Inflammatory diarrhea (invasive gastroenteritis; grossly bloody stool and fever may be present) [†]	Shigella species Campylobacter species Salmonella species Enteroinvasive <i>E. coli</i> Enterohemorrhagic <i>E. coli</i> <i>E. coli</i> O157:H7 Vibrio parahaemolyticus Yersinia enterocolitica Entamoeba histolytica
Persistent diarrhea (lasting ≥14 days)	Prolonged illness should prompt examination for parasites, particularly in travelers to mountainous or other areas where untreated water is consumed. Consider <i>Cyclospora cayetanensis, Cryptosporidium, Entamoeba histolytica</i> , and <i>Giardia lamblia</i> .
Neurologic manifestations (eg, paresthesias, respiratory depression, bronchospasm, cranial nerve palsies)	Botulism (<i>Clostridium botulinum</i> toxin) Organophosphate pesticides Thallium poisoning Scombroid fish poisoning (histamine, saurine) Ciguatera fish poisoning (ciguatoxin) Tetradon fish poisoning (tetradotoxin) Neurotoxic shellfish poisoning (brevitoxin) Paralytic shellfish poisoning (saxitoxin) Amnesic shellfish poisoning (domoic acid) Mushroom poisoning Guillain-Barré syndrome (associated with infectious diarrhea due to <i>Campylobacter jejuni</i>)
Systemic illness (eg, fever, weakness, arthritis, jaundice)	Listeria monocytogenes Brucella species Trichinella spiralis Toxoplasma gondii Vibrio vulnificus Hepatitis A and E viruses Salmonella Typhi and Salmonella Paratyphi Amebic liver abscess

TABLE 1. Etiologic agents to consider for various manifestations of foodborne illness

* Noninflammatory diarrhea is characterized by mucosal hypersecretion or decreased absorption without mucosal destruction and generally involves the small intestine. Some affected patients may be dehydrated because of severe watery diarrhea and may appear seriously ill. This is more common in the young and the elderly. Most patients experience minimal dehydration and appear mildly ill with scant physical findings. Illness typically occurs with abrupt onset and brief duration. Fever and systemic symptoms usually are absent (except for symptoms related directly to intestinal fluid loss).

[†]Inflammatory diarrhea is characterized by mucosal invasion with resulting inflammation and is caused by invasive or cytotoxigenic microbial pathogens. The diarrheal illness usually involves the large intestine and may be associated with fever, abdominal pain and tenderness, headache, nausea, vomiting, malaise, and myalgia. Stools may be bloody and may contain many fecal leukocytes.

ease process. The extent of diagnostic evaluation depends on the clinical picture, the differential diagnosis considered, and clinical judgment.

The presentation of a patient with a foodborne illness is often only slightly different from that of a patient who presents with a viral syndrome. In addition, viral syndromes are so common that it is reasonable to assume that a percentage of those diagnosed with a viral syndrome have actually contracted a foodborne illness. Therefore, the viral syndrome must be excluded in order to suspect the foodborne illness and take appropriate public health action. Fever, diarrhea, and abdominal cramps can be present or absent in both cases so they are not very helpful. The absence of myalgias or arthralgias would make a viral syndrome less likely and a foodborne illness (that does not target the neurologic system) more likely. Foodborne illnesses that do target the neurologic system tend to cause paraesthesias, weakness and paralysis that are distinguishable from myalgias or arthralgias (see below). The presence of dysentery (bloody diarrhea) is also more indicative of a foodborne illness, particularly if it is early in the course.

If any of the following signs and symptoms occur in patients, either alone or in combination, laboratory testing may provide important diagnostic clues (particular attention should be given to very young and elderly patients and to immunocompromised patients, all of whom are more vulnerable):

- Bloody diarrhea
- Weight loss
- Diarrhea leading to dehydration
- Fever
- Prolonged diarrhea (3 or more unformed stools per day, persisting several days)
- Neurologic involvement, such as paresthesias, motor weakness, cranial nerve palsies
- Sudden onset of nausea, vomiting, diarrhea
- Severe abdominal pain

In addition to foodborne causes, a differential diagnosis of gastrointestinal tract disease should include underlying medical conditions such as irritable bowel syndrome; inflammatory bowel diseases such as Crohn's disease or ulcerative colitis; malignancy; medication use (including antibiotic-related *Clostridium difficile* toxin colitis); gastrointestinal tract surgery or radiation; malabsorption syndromes; immune deficiencies; and numerous other structural, functional, and metabolic etiologies. Consideration also should be given to exogenous factors such as the association of the illness with travel, occupation, emotional stress, sexual habits, exposure to other ill persons, recent hospitalization, child care center attendance, and nursing home residence. The differential diagnosis of patients presenting with neurologic symptoms due to a foodborne illness is also complex. Possible food-related causes to consider include recent ingestion of contaminated seafood, mushroom poisoning, and chemical poisoning. Because the ingestion of certain toxins (eg, botulinum toxin, tetrodotoxin) and chemicals (eg, organophosphates) can be life-threatening, a differential diagnosis must be made quickly with concern for aggressive therapy and life support measures (eg, respiratory support, administration of antitoxin or atropine), and possible hospital admission.

Clinical Microbiology Testing

When submitting specimens for microbiologic testing, it is important to realize that clinical microbiology laboratories differ in protocols used for the detection of pathogens. To optimize recovery of an etiologic agent, physicians and other health care professionals should understand routine specimencollection and testing procedures as well as circumstances and procedures for making special test requests. Some complex tests (eg, toxin testing, serotyping, molecular techniques) may only be available from large commercial or public health laboratories. Contact your microbiology laboratory for more information.

Stool cultures are indicated if the patient is immunocompromised, febrile, has bloody diarrhea, has severe abdominal pain, or if the illness is clinically severe or persistent. Stool cultures are also recommended if many fecal leukocytes are present. This indicates diffuse colonic inflammation and is suggestive of invasive bacterial pathogens such as *Shigella*, *Salmonella*, and *Campylobacter* species and invasive *E. coli*. In most laboratories, routine stool cultures are limited to screening for *Salmonella* and *Shigella* species and *Campylobacter jejuni/coli*. Cultures for *Vibrio* and *Yersinia* species, *E. coli* O157:H7, and *Campylobacter* species other than *jejuni/coli* require additional media or incubation conditions and therefore require advance notification or communication with laboratory and infectious disease personnel.

Stool examination for parasites generally is indicated for patients with suggestive travel histories, who are immunocompromised, who suffer chronic or persistent diarrhea, or when the diarrheal illness is unresponsive to appropriate antimicrobial therapy. Stool examination for parasites is also indicated for gastrointestinal tract illnesses that appear to have a long incubation period. Requests for ova and parasite examination of a stool specimen will often enable identification of *Giardia lamblia* and *Entamoeba histolytica*, but a special request may be needed for detection of *Cryptosporidium* and *Cyclospora cayetanensis*. Each laboratory may vary in its routine procedures for detecting parasites, so it is important to contact your laboratory.

Blood cultures should be obtained when bacteremia or systemic infection is suspected.

Direct antigen detection tests and molecular biology techniques are available for rapid identification of certain bacterial, viral, and parasitic agents in clinical specimens. In some circumstances, microbiologic and chemical laboratory testing of vomitus or implicated food items also is warranted. For more information on laboratory procedures for the detection of foodborne pathogens, consult an appropriate medical specialist, clinical microbiologist, or state public health laboratory.

Treating Foodborne Illness

Selection of appropriate treatment depends on identification of the responsible pathogen (if possible) and determining if specific therapy is available. Many episodes of acute gastroenteritis are self-limiting and require fluid replacement and supportive care. Oral rehydration is indicated for patients who are mildly to moderately dehydrated; intravenous therapy may be required for more severe dehydration. Routine use of antidiarrheal agents is not recommended because many of these agents have potentially serious adverse effects in infants and young children.

- Choice of antimicrobial therapy should be based on
- Clinical signs and symptoms;
- Organism detected in clinical specimens;
- Antimicrobial susceptibility tests; and
- Appropriateness of treating with an antibiotic (some enteric bacterial infections are best not treated).

Knowledge of the infectious agent and its antimicrobial susceptibility pattern allows the physician to initiate, change, or discontinue antimicrobial therapy. Such information also can support public health surveillance of infectious disease and antimicrobial resistance trends in the community. Antimicrobial resistance has increased for some enteric pathogens, which dictates judicious use of this therapy.

Suspected cases of botulism are treated with botulinum antitoxin. Equine botulinum antitoxin for types A, B, and E can prevent the progression of neurologic dysfunction if administered early in the course of illness. Physicians and other health care professionals should notify their local and state health departments regarding suspected cases of botulism. CDC maintains a 24-hour consultation service to assist health care professionals with the diagnosis and management of this rare disease.

Surveillance and Reporting of Foodborne Illness

Reporting of foodborne illnesses in the United States began more than 50 years ago when state health officers, concerned about the high morbidity and mortality caused by typhoid fever and infantile diarrhea, recommended that cases of "enteric fever" be investigated and reported. The intent of investigating and reporting these cases was to obtain information about the role of food, milk, and water in outbreaks of gastrointestinal tract illness as the basis for public health actions. These early reporting efforts led to the enactment of important public health measures (eg, the Pasteurized Milk Ordinance) that profoundly decreased the incidence of foodborne illnesses.

Often health care professionals may suspect foodborne illness either because of the organism involved or because of other available information, such as several ill patients who have eaten the same food. Health care professionals can serve as the eyes and ears for the health department by providing such information to local or state public health authorities. Foodborne disease reporting is not only important for disease prevention and control, but more accurate assessments of the burden of foodborne illness in the community occur when physicians and other health care professionals report foodborne illnesses to the local and state health department. In addition, reporting of cases of foodborne illness by practicing physicians to the local health department may help the health officer identify a foodborne disease outbreak in the community. This may lead to early identification and removal of contaminated products from the commercial market. If a restaurant or other food service establishment is identified as the source of the outbreak, health officers will work to correct inadequate food preparation practices, if necessary. If the home is the likely source of the contamination, health officers can institute public education about proper food handling practices. Occasionally, reporting may lead to the identification of a previously unrecognized agent of foodborne illness. Reporting also may lead to identification and appropriate management of human carriers of known foodborne pathogens, especially those with high-risk occupations for disease transmission such as foodworkers.

Table 2 lists current reporting requirements for foodborne diseases and conditions in the United States. National reporting requirements are determined collaboratively by the Council of State and Territorial Epidemiologists and CDC. Additional reporting requirements may also be mandated by state and territorial laws and regulations. Details on specific state reporting requirements are available from state health depart-

TABLE 2. Foodborne diseases and conditions designated as notifiable at the national level* — United States 2003

Notifiable BACTERIAL foodborne diseases and conditions Anthrax Botulism Brucellosis Cholera Enterohemorrhagic <i>Escherichia coli</i> Hemolytic uremic syndrome, post-diarrheal Listeriosis Salmonellosis (other than <i>S.</i> Typhi) Shigellosis Typhoid fever (<i>S.</i> Typhi and <i>S.</i> Paratyphi infections)
Notifiable VIRAL foodborne diseases and conditions Hepatitis A
Notifiable PARASITIC foodborne diseases and conditions Cryptosporidiosis Cyclosporiasis Giardiasis Trichinellosis
In the United States, additional reporting requirements may be mandated by state and territorial laws and regulations. Details on specific state reporting requirements are available from state health departments and from the
Council of State and Territorial Epidemiologists (phone number: 770-458-3811). Information available electronically at: www.cste.org/nndss/reportingrequirements.htm.
Centers for Disease Control and Prevention. Information available electronically at www.cdc.gov/epo/dphsi/phs/ infdis2003.htm.

ments and from the Council of State and Territorial Epidemiologists and CDC.

Typically, the appropriate procedure for health care professionals to follow in reporting foodborne illnesses is to contact the local or state health department whenever they identify a specific notifiable foodborne disease. However, it is often unclear if a patient has a foodborne illness prior to diagnostic tests, so health care professionals should also report potential foodborne illnesses, such as when 2 or more patients present with a similar illness that may have resulted from the ingestion of a common food. Local health departments then report the illnesses to the state health departments and determine if further investigation is warranted.

Each state health department reports foodborne illnesses to CDC. CDC compiles these data nationally and disseminates information via the weekly *Morbidity and Mortality Weekly Report* and annual summary reports. CDC assists state and local public health authorities with epidemiologic investigations and the design of interventions to prevent and control foodrelated outbreaks. CDC also coordinates a national network of public health laboratories, called PulseNet, which performs "molecular fingerprinting" of bacteria (by pulsed-field gel electrophoresis) to support epidemiologic investigations.

Thus, in addition to reporting cases of potential foodborne illnesses, it is important for physicians to report noticeable increases in unusual illnesses, symptom complexes, or disease patterns (even without definitive diagnosis) to public health authorities. Prompt reporting of unusual patterns of diarrheal/ gastrointestinal tract illness, for example, can allow public health officials to initiate an epidemiologic investigation earlier than would be possible if the report awaited definitive etiologic diagnosis.

Finally, new information on food safety is constantly emerging. Recommendations and precautions for people at high risk are updated whenever new data about preventing foodborne illness become available. Physicians and other health care professionals need to be aware of and follow the most current information on food safety.

Foodborne Illnesses (Bacterial)

Etiology	Incubation Period	Signs and Symptoms	Duration of Illness	Associated Foods	Laboratory Testing	Treatment
Bacillus anthracis	2 days to weeks	Nausea, vomiting, malaise, bloody diarrhea, acute abdominal pain.	Weeks	Insufficiently cooked contaminated meat.	Blood.	Penicillin is first choice for naturally acquired gastrointes- tinal anthrax. Ciprofloxacin is second option.
Bacillus cereus (preformed enterotoxin)	1–6 hrs	Sudden onset of severe nausea and vomiting. Diarrhea may be present.	24 hrs	Improperly refrigerated cooked or fried rice, meats.	Normally a clinical diagnosis. Clinical laboratories do not routinely identify this organism. If indicated, send stool and food specimens to reference laboratory for culture and toxin identification.	Supportive care.
<i>Bacillus cereus</i> (diarrheal toxin)	10–16 hours	Abdominal cramps, watery diarrhea, nausea.	24–48 hours	Meats, stews, gravies, vanilla sauce.	Testing not necessary, self- limiting (consider testing food and stool for toxin in outbreaks).	Supportive care.
Brucella abortus, B. melitensis, and B. suis	7–21 days	Fever, chills, sweating, weakness, headache, muscle and joint pain, diarrhea, bloody stools during acute phase.	Weeks	Raw milk, goat cheese made from unpasteur- ized milk, contaminated meats.	Blood culture and positive serology.	Acute: Rifampin and doxycycline daily for ≥6 weeks. Infections with complications require combination therapy with rifampin, tetracycline, and an aminoglycoside.
Campylobacter jejuni	2–5 days	Diarrhea, cramps, fever, and vomiting; diarrhea may be bloody.	2–10 days	Raw and undercooked poultry, unpasturized milk, contaminated water.	Routine stool culture; <i>Campylobacter</i> requires special media and incubation at 42°C to grow.	Supportive care. For severe cases, antibiotics such as erythromycin and quinolones may be indicated early in the diarrheal disease. Guillain-Barré syndrome can be a sequela.
Clostridium botulinum— children and adults (preformed toxin)	12–72 hrs	Vomiting, diarrhea, blurred vision, diplopia, dysphagia, and descending muscle weakness.	Variable (from days to months). Can be compli- cated by respiratory failure and death.	Home-canned foods with a low acid content, improperly canned commercial foods, home-canned or fermented fish, herb- infused oils, baked potatoes in aluminium foil, cheese sauce, bottled garlic, foods held warm for extended periods of time (eg, in a warm oven).	Stool, serum, and food can be tested for toxin. Stool and food can also be cultured for the organism. These tests can be performed at some state health department laboratories and CDC.	Supportive care. Botulinum antitoxin is helpful if given early in the course of the illness. Contact the state health department. The 24- hour number for state health departments to call is (770) 488-7100.
<i>Clostridium</i> <i>botulinum</i> —infants	3–30 days	In infants <12 months, lethargy, weakness, poor feeding, constipation, hypotonia, poor head control, poor gag and sucking reflex.	Variable	Honey, home-canned vegetables and fruits, corn syrup.	Stool, serum, and food can be tested for toxin. Stool and food can also be cultured for the organism. These tests can be performed at some state health department laboratories and CDC.	Supportive care. Botulism immune globulin can be obtained from the Infant Botulism Prevention Program, Health and Human Services, California (510-540-2646). Botulinum antitoxin is generally not recommended for infants.
Clostridium perfringens toxin	8–16 hrs	Watery diarrhea, nausea, abdominal cramps; fever is rare.	24–48 hrs	Meats, poultry, gravy, dried or precooked foods, time- and/or temperature-abused food.	Stools can be tested for enterotoxin and cultured for organism. Because <i>Clostridium perfringens</i> can normally be found in stool, quantitative cultures must be done.	Supportive care. Antibiotics not indicated.
Enterohemorrhagic E. coli (EHEC) including E. coli O157:H7 and other Shiga toxin-producing E. coli (STEC)	1–8 days	Severe diarrhea that is often bloody, abdominal pain and vomiting. Usually, little or no fever is present. More common in children <4 years.	5–10 days	Undercooked beef especially hamburger, unpasteurized milk and juice, raw fruits and vegetables (eg. sprouts), salami (rarely), and contaminated water.	Stool culture; <i>E. coli</i> O157:H7 requires special media to grow. If <i>E. coli</i> O157:H7 is suspected, specific testing must be requested. Shiga toxin testing may be done using commercial kits; positive isolates should be forwarded to public health laboratories for confirmation and serotyping.	Supportive care, monitor renal function, hemoglobin, and platelets closely. <i>E. coli</i> O157:H7 infection is also associated with hemolytic uremic syndrome (HUS), which can cause lifelong complications. Studies indicate that antibiotics may promote the development of HUS.

Etiology	Incubation Period	Signs and Symptoms	Duration of	Associated Foods	Laboratory Testing	Trastmont
Etiology Enterotoxigenic	Period 1–3 days	Signs and Symptoms Watery diarrhea,	3 to >7	Associated Foods Water or food	Laboratory Testing Stool culture. ETEC	Treatment Supportive care. Antibiotics
E. coli (ETEC)	i o days	abdominal cramps, some vomiting.	days	contaminated with human feces.	requires special laboratory techniques for identifica- tion. If suspected, must request specific testing.	are rarely needed except in severe cases. Recommended antibiotics include TMP-SMX and quinolones.
Listeria monocytogenes	9–48 hrs for gastrointestinal symptoms, 2–6 weeks for invasive disease	Fever, muscle aches, and nausea or diarrhea. Pregnant women may have mild flu-like illness, and infection can lead to premature delivery or stillbirth. Elderly or immunocompromised patients may have bacteremia or meningitis.	Variable	Fresh soft cheeses, unpasteurized milk, inadequately pasteur- ized milk, ready-to-eat deli meats, hot dogs.	Blood or cerebrospinal fluid cultures. Asymptomatic fecal carriage occurs; therefore, stool culture usually not helpful. Antibody to listerolysin O may be helpful to identify outbreak retrospectively.	Supportive care and antibiotics; Intravenous ampicillin, penicillin, or TMP- SMX are recommended for invasive disease.
	At birth and infancy	Infants infected from mother at risk for sepsis or meningitis.				
Salmonella spp.	1–3 days	Diarrhea, fever, abdominal cramps, vomiting. S. Typhi and S. Paratyphi produce typhoid with insidious onset characterized by fever, headache, constipation, malaise, chills, and myalgia; diarrhea is uncommon, and vomiting is not usually severe.	4–7 days	Contaminated eggs, poultry, unpasteurized milk or juice, cheese, contaminated raw fruits and vegetables (alfalfa sprouts, melons). <i>S</i> . Typhi epidemics are often related to fecal contamination of water supplies or street- vended foods.	Routine stool cultures.	Supportive care. Other than for S. Typhi and S. Paratyphi, antibiotics are not indicated unless there is extra-intestinal spread, or the risk of extra- intestinal spread, of the infection. Consider ampicillin, gentamicin, TMP-SMX, or quinolones if indicated. A vaccine exists for S. Typhi.
<i>Shigella</i> spp.	24–48 hrs	Abdominal cramps, fever, and diarrhea. Stools may contain blood and mucus.	4–7 days	Food or water contaminated with human fecal material. Usually person-to- person spread, fecal- oral transmission. Ready-to-eat foods touched by infected food workers, eg, raw vegetables, salads, sandwiches.	Routine stool cultures.	Supportive care. TMP-SMX recommended in the US if organism is susceptible; nalidixic acid or other quinolones may be indicated i organism is resistant, especially in developing countries.
Staphylococcus aureus (preformed enterotoxin)	1–6 hrs	Sudden onset of severe nausea and vomiting. Abdominal cramps. Diarrhea and fever may be present.	24–48 hrs	Unrefrigerated or improperly refrigerated meats, potato and egg salads, cream pastries.	Normally a clinical diagnosis. Stool, vomitus, and food can be tested for toxin and cultured if indicated.	Supportive care.
<i>Vibrio cholerae</i> (toxin)	24–72 hrs	Profuse watery diarrhea and vomiting, which can lead to severe dehydration and death within hours.	3–7 days. Causes life- threatening dehydra- tion.	Contaminated water, fish, shellfish, street- vended food typically from Latin America or Asia.	Stool culture; <i>Vibrio</i> <i>cholerae</i> requires special media to grow. If <i>V.</i> <i>cholerae</i> is suspected, must request specific testing.	Supportive care with aggressive oral and intra- venous rehydration. In cases of confirmed cholera, tetracycline or doxycycline is recommende for adults, and TMP-SMX for children (<8 years).
Vibrio para- haemolyticus	2–48 hrs	Watery diarrhea, abdominal cramps, nausea, vomiting.	2–5 days	Undercooked or raw seafood, such as fish, shellfish.	Stool cultures. <i>Vibrio</i> <i>parahaemolyticus</i> requires special media to grow. If <i>V.</i> <i>parahaemolyticus</i> is suspected, must request specific testing.	Supportive care. Antibiotics are recommended in severe cases: tetracycline, doxycy- cline, gentamicin, and cefotaxime.
Vibrio vulnificus	1–7 days	Vomiting, diarrhea, abdominal pain, bacteremia, and wound infections. More common in the immunocompro- mised, or in patients with chronic liver disease (presenting with bullous skin lesions). Can be fatal in patients with liver disease and the immunocompromised.	2–8 days	Undercooked or raw shellfish, especially oysters, other contaminated seafood, and open wounds exposed to sea water.	Stool, wound, or blood cultures. <i>Vibrio vulnificus</i> requires special media to grow. If <i>V. vulnificus</i> is suspected, must request specific testing.	Supportive care and antibiotics; tetracycline, doxycycline, and ceftazidime are recommended.

Foodborne Illnesses (Bacterial) (Continued)

Etiology	Incubation Period	Signs and Symptoms	Duration of Illness	Associated Foods	Laboratory Testing	Treatment
Yersinia enterocolytica and Y. pseudotuber- culosis	24–48 hrs	Appendicitis-like symptoms (diarrhea and vomiting, fever, and abdominal pain) occur primarily in older children and young adults. May have a scarlitiniform rash with <i>Y. pseudotuber-</i> <i>culosis</i> .	1–3 weeks, usually self- limiting	Undercooked pork,	Stool, vomitus, or blood culture. <i>Yersinia</i> requires special media to grow. If	Supportive care. If septicemia or other invasive disease occurs, antibiotic therapy with gentamicin or cefotaxime (doxycycline and ciprofloxacin also effective).

Foodborne Illnesses (Viral)

Etiology	Incubation Period	Signs and Symptoms	Duration of Illness	Associated Foods	Laboratory Testing	Treatment
Hepatitis A	28 days average (15–50 days)	Diarrhea, dark urine, jaundice, and flu-like symptoms, i.e., fever, headache, nausea, and abdominal pain.	Variable, 2 weeks – 3 months	Shellfish harvested from contaminated waters, raw produce, contami- nated drinking water, uncooked foods and cooked foods that are not reheated after contact with infected food handler.	Increase in ALT, bilirubin. Positive IgM and anti- hepatitis A antibodies.	Supportive care. Prevention with immunization.
Noroviruses (and other caliciviruses)	12–48 hrs	Nausea, vomiting, abdominal cramping, diarrhea, fever, myalgia, and some headache. Diarrhea is more prevalent in adults and vomiting is more prevalent in children.	12–60 hrs	Shellfish, fecally contaminated foods, ready-to-eat foods touched by infected food workers (salads, sandwiches, ice, cookies, fruit).	Routine RT-PCR and EM on fresh unpreserved stool samples. Clinical diagnosis, negative bacterial cultures. Stool is negative for WBCs.	Supportive care such as rehydration. Good hygiene.
Rotavirus	1–3 days	Vomiting, watery diarrhea, low-grade fever. Temporary lactose intolerance may occur. Infants and children, elderly, and immunocompromised are especially vulnerable.	4–8 days	Fecally contaminated foods. Ready-to-eat foods touched by infected food workers (salads, fruits).	Identification of virus in stool via immunoassay.	Supportive care. Severe diarrhea may require fluid and electrolyte replacement.
Other viral agents (astroviruses, adenoviruses, parvoviruses)	10–70 hrs	Nausea, vomiting, diarrhea, malaise, abdominal pain, headache, fever.	2–9 days	Fecally contaminated foods. Ready-to-eat foods touched by infected food workers. Some shellfish.	Identification of the virus in early acute stool samples. Serology. Commercial ELISA kits are now available for adenoviruses and astroviruses.	Supportive care, usually mild, self-limiting. Good hygiene.

Foodborne Illnesses (Parasitic)

Etiology	Incubation Period	Signs and Symptoms	Duration of Illness	Associated Foods	Laboratory Testing	Treatment
Angiostrongylus cantonensis	1 week to ≥1 month	Severe headaches, nausea, vomiting, neck stiffness, paresthesias, hyperesthesias, seizures, and other neurologic abnormalities.	Several weeks to several months	Raw or undercooked intermediate hosts (eg, snails or slugs), infected paratenic (transport) hosts (eg, crabs, fresh water shrimp), fresh produce contaminated with intermediate or transport hosts.	Examination of CSF for elevated pressure, protein, leukocytes, and eosino- phils; serologic testing using ELISA to detect antibodies to <i>Angiostrongylus</i> cantonensis.	Supportive care. Repeat lumbar punctures and use of corticosteroid therapy may be used for more severely ill patients.
Cryptosporidium	2–10 days	Diarrhea (usually watery), stomach cramps, upset stomach, slight fever.	May be remitting and relapsing over weeks to months	Any uncooked food or food contaminated by an ill food handler after cooking, drinking water.	Request specific examination of the stool for <i>Cryptosporidium</i> . May need to examine water or food.	Supportive care, self-limited. If severe consider paromomycin for 7 days. For children aged 1–11 years, consider nitazoxanide for 3 days.
Cyclospora cayetanensis	1–14 days, usually at least 1 week	Diarrhea (usually watery), loss of appetite, substantial loss of weight, stomach cramps, nausea, vomiting, fatigue.	May be remitting and relapsing over weeks to months	Various types of fresh produce (imported berries, lettuce).	Request specific examination of the stool for <i>Cyclospora</i> . May need to examine water or food.	TMP-SMX for 7 days.

Etiology	Incubation Period	Signs and Symptoms	Duration of Illness	Associated Foods	Laboratory Testing	Treatment
Entamoeba histolytica	2–3 days to 1–4 weeks	Diarrhea (often bloody), frequent bowel move- ments, lower abdominal pain.	May be protracted (several weeks to several months)	Any uncooked food or food contaminated by an ill food handler after cooking, drinking water.	Examination of stool for cysts and parasites—may need at least 3 samples. Serology for long-term infections.	Metronidazole and a luminal agent (iodoquinol or paromomycin).
Giardia lamblia	1–2 weeks	Diarrhea, stomach cramps, gas.	Days to weeks	Any uncooked food or food contaminated by an ill food handler after cooking, drinking water.	Examination of stool for ova and parasites — may need at least 3 samples.	Metronidazole.
Toxoplasma gondii	5–23 days	Generally asymptomatic, 20% may develop cervical lymphadenopathy and/or a flu-like illness. In immunocompromised <u>patients:</u> central nervous system (CNS) disease, myocarditis, or pneumoni- tis is often seen.	Months	Accidental ingestion of contaminated substances (eg, soil contaminated with cat feces on fruits and vegetables), raw or partly cooked meat (especially pork, lamb, or venison).	Isolation of parasites from blood or other body fluids; observation of parasites in patient specimens via microscopy or histology. Detection of organisms is rare; serology (reference laboratory needed) can be a useful adjunct in diagnosing toxoplasmosis. However, IgM antibodies may persist for 6–18 months and thus may not necessarily indicate recent infection. PCR of bodily fluids. <u>For congenital infection</u> ; isolation of <i>T. gondii</i> from placenta, umbilical cord, or infant blood. PCR of white blood cells, CSF, or amniotic fluid, or IgM and IgA serology, performed by a reference laboratory.	Asymptomatic healthy, but infected, persons do not require treatment. Spiramycin or pyrimethamine plus sulfadiazine may be used for pregnant women. Pyrimethamine plus sulfadiazine may be used for immunocompromised persons, in specific cases. Pyrimethamine plus sulfadiazine (with or without steroids) may be given for ocular disease when indicated Folinic acid is given with pyrimethamine plus sulfadiaz- ine to counteract bone marrow suppression.
<i>Toxoplasma gondii</i> (congenital infection)	In infants at birth	Treatment of the mother may reduce severity and/ or incidence of congenital infection. Most infected infants have few symptoms at birth. Later, they will generally develop signs of congenital toxoplasmosis (mental retardation, severely impaired eyesight, cerebral palsy, seizures), unless the infection is treated.	Months	Passed from mother (who acquired acute infection during pregnancy) to child.	,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,	
Trichinella spiralis	1–2 days for initial symptoms; others begin 2–8 weeks after infection	Acute: nausea, diarrhea, vomiting, fatigue, fever, abdominal discomfort followed by muscle soreness, weakness, and occasional cardiac and neurologic complications.	Months	Raw or undercooked contaminated meat, usually pork or wild game meat (eg, bear or moose).	Positive serology or demonstration of larvae via muscle biopsy. Increase in eosinophils.	Supportive care plus mebendazole or albendazole.

Foodborne Illnesses (Noninfectious)

Etiology	Incubation Period	Signs and Symptoms	Duration of Illness	Associated Foods	Laboratory Testing	Treatment
Antimony	5 min – 8 hrs. usually <1 hr	Vomiting, metallic taste.	Usually self-limited	Metallic container.	Identification of metal in beverage or food.	Supportive care.
Arsenic	Few hrs	Vomiting, colic, diarrhea.	Several days	Contaminated food.	Urine. May cause eosinophilia.	Gastric lavage, BAL (dimercaprol).
Cadmium	5 min – 8 hrs. usually <1 hr	Nausea, vomiting, myalgia, increase in salivation, stomach pain.	Usually self-limited	Seafood, oysters, clams, lobster, grains, peanuts.	Identification of metal in food.	Supportive care.
Ciguatera fish poisoning (ciguatera toxin)	2–6 hrs	<u>GI:</u> abdominal pain, nausea, vomiting, diarrhea.	weeks to fish. Grouper, red	snapper, amberjack,	Radioassay for toxin in fish or a consistent history.	Supportive care, IV mannitol. Children more vulnerable.
	3 hrs	<u>Neurologic:</u> paresthesias, reversal of hot or cold, pain, weakness.				
	2–5 days	<u>Cardiovascular:</u> bradycardia, hypotension, increase in T wave abnormalities.				
Copper	5 min – 8 hrs. usually <1 hr	Nausea, vomiting, blue or green vomitus.	Usually self-limited	Metallic container.	Identification of metal in beverage or food.	Supportive care.
Mercury	1 week or longer	Numbness, weakness of legs, spastic paralysis, impaired vision, blindness, coma. Pregnant women and the developing fetus are especially vulnerable.	May be protracted	Fish exposed to organic mercury, grains treated with mercury fungicides.	Analysis of blood, hair.	Supportive care.
Mushroom toxins, short-acting (museinol, muscarine, psilocybin, coprius artemetaris, ibotenic acid)	<2 hrs	Vomiting, diarrhea, confusion, visual disturbance, salivation, diaphoresis, hallucinations, disulfiram-like reaction, confusion, visual disturbance.	Self-limited	Wild mushrooms (cooking may not destroy these toxins).	Typical syndrome and mushroom identified or demonstration of the toxin.	Supportive care.
Mushroom toxin, long-acting (amanitin)	4–8 hrs diarrhea; 24–48 hrs liver failure	Diarrhea, abdominal cramps, leading to hepatic and renal failure.	Often fatal	Mushrooms.	Typical syndrome and mushroom identified and/or demonstration of the toxin.	Supportive care, life- threatening, may need life support.
Nitrite poisoning	1–2 hrs	Nausea, vomiting, cyanosis, headache, dizziness, weakness, loss of consciousness, chocolate-brown colored blood.	Usually self-limited	Cured meats, any contaminated foods, spinach exposed to excessive nitrification.	Analysis of the food, blood.	Supportive care, methylene blue.
Pesticides (organophosphates or carbamates)	Few min to few hrs	Nausea, vomiting, abdominal cramps, diarrhea, headache, nervousness, blurred vision, twitching, convulsions, salivation and meiosis.	Usually self-limited	Any contaminated food.	Analysis of the food, blood.	Atropine; 2-PAM (Pralidoxime) is used when atropine is not able to control symptoms and is rarely necessary in carbamate poisoning.
Puffer fish (tetrodotoxin)	<30 min	Parasthesias, vomiting, diarrhea, abdominal pain, ascending paralysis, respiratory failure.	Death usually in 4–6 hours	Puffer fish.	Detection of tetrodotoxin in fish.	Life-threatening, may need respiratory support.
Scombroid (histamine)	1 min – 3 hrs	Flushing, rash, burning sensation of skin, mouth and throat, dizziness, uriticaria, parasthesias.	3–6 hrs	Fish: bluefin, tuna, skipjack, mackerel, marlin, escolar, and mahi mahi.	Demonstration of histamine in food or clinical diagnosis.	Supportive care, antihista- mines.

Incubation Duration of Etiology Period Signs and Symptoms Illness Associated Foods Laboratory Testing Treatment Shellfish toxins Diarrheic Nausea, vomiting, Hrs to 2-3 A variety of shellfish, Detection of the toxin in Supportive care, generally self-(diarrheic, shellfish diarrhea, and abdominal days primarily mussels, shellfish; high-pressure limiting. Elderly are especially neurotoxic, poisoning pain accompanied by oysters, scallops, and liquid chromatography. sensitive to ASP. (DSP) - 30 chills, headache, and shellfish from the Florida amnesic) min to 2 hrs fever. coast and the Gulf of Mexico. Neurotoxic Tingling and numbness of lips, tongue, and throat, shellfish muscular aches, dizziness, poisoning (NSP) - few reversal of the sensations min to hours of hot and cold, diarrhea, and vomiting Amnesic Vomiting, diarrhea, shellfish abdominal pain and poisoning neurologic problems such (ASP) as confusion, memory 24-48 hrs loss, disorientation, seizure, coma. Shellfish toxins 30 min – 3 hrs Diarrhea, nausea, vomiting Days Scallops, mussels, Detection of toxin in food or Life-threatening, may need (paralytic shellfish leading to parasthesias of clams, cockles. water where fish are respiratory support. poisoning) mouth, lips, weakness, located; high-pressure dysphasia, dysphonia, liquid chromatography. respiratory paralysis. Sodium fluoride Few min to Salty or soapy taste, Usually Dry foods (eg, dry milk, Testing of vomitus or Supportive care. numbness of mouth, self-limited flour, baking powder, gastric washings. Analysis 2 hrs vomiting, diarrhea, dilated cake mixes) contamiof the food. pupils, spasms, pallor, nated with sodium shock, collapse. fluoride-containing insecticides and rodenticides. Thallium Nausea, vomiting, Several Contaminated food. Supportive care. Few hrs Urine, hair. diarrhea, painful days parathesias, motor polyneuropathy, hair loss. Tin 5 min – 8 hrs. Nausea, vomiting, Usually Metallic container. Analysis of the food. Supportive care. diarrhea. self-limited usually <1 hr Few min to Nausea, headache, Usually Grains such as wheat, Vomitoxin Analysis of the food. Supportive care. self-limited abdominal pain, vomiting. 3 hrs corn. barley. Usuallv Analysis of the food, blood Zinc Few hrs Stomach cramps, nausea. Metallic container. Supportive care. self-limited and feces, saliva or urine. vomiting, diarrhea, myalgias.

Foodborne Illnesses (Noninfectious) (Continued)

Patient Scenarios

The learning scenarios in this section can be used to reinforce medical management information pertaining to foodborne illnesses, such as that provided from the previous sections of this primer. The case studies provide questions that need to be considered when dealing with a potential case of foodborne illness. Answers are provided immediately following the questions to enhance the learning process.

Similar learning scenarios are also available for other foodborne pathogens.

Congenital Toxoplasmosis, A Patient Scenario

Susan, a 6-month-old infant, is brought to your office for evaluation of apparent blindness. Her mother reports that she had been well during the pregnancy and the delivery was uncomplicated. The baby appeared healthy until age 4 months, when the parents became concerned about her vision.

Physical examination was normal except for bilateral macular scars, microphthalmos, and unresponsiveness to visual stimuli. There were no other neurologic abnormalities, and her growth and development were appropriate for her age. A computed tomography (CT) scan of the head was obtained.

Congenital infection with which of the following should be included in the differential diagnosis?

- Viruses:
 - Cytomegalovirus
 - Rubella
 - Herpes simplex
 - -Human immunodeficiency virus
- Bacteria:
 - Treponema pallidum
 - Listeria monocytogenes
- Parasites:
 - Toxoplasma gondii

What additional information would assist with the diagnosis?

- More history from the mother, including travel to foreign country
- Vaccination record, including during pregnancy
- · History of exposure to cats and raw meat
- History of multiple sex partners and sexually transmitted disease (STD)
- History of herpes
- Evaluation of CT scan

The CT scan of the child's head showed periventricular calcifications and asymmetric dilation of the lateral ventricles. The mother is 35 years old and reiterated that she does not recall being ill during the pregnancy; however, she also indicated that she would not necessarily remember every little symptom. She also denied having a history of STDs. She had received the mumps-measles-rubella (MMR) vaccine as a child but no vaccines during pregnancy. The mother recalled eating insufficiently cooked meat while traveling in France during the first trimester of pregnancy. The family does not own a cat, and she does not recall having been exposed to cats during her pregnancy.

What diagnostic tests are needed?

Serologic evaluation of both mother and child focusing on potential congenital infection (ie, a ToRCH profile) based on the history of the mother ingesting raw meat while traveling in a foreign country during first trimester of pregnancy and the clinical findings (blindness, cerebral calcifications, and hydrocephalus).

Results of serologic testing detected both IgG and IgM antibodies to *Toxoplasma gondii* in both the baby's and mother's serum. The mother's IgM titer was 1:6400 and IgG titer was 1:6400, while those of the baby were IgM titer of 1:160 and IgG titer of 1:6400.

How does this information assist with the diagnosis?

Diagnosis of toxoplasmosis is usually confirmed by serologic tests. Occasionally, organisms are identified in tissue or body fluids or isolated by culture or animal inoculation. Polymerase chain reaction (PCR)-based assays are available from some laboratories for diagnosis of fetal infection and infection in compromised hosts. For immunocompetent persons, seroconversion or a 4-fold rise of specific IgG antibodies or demonstration of specific IgM antibodies indicate recent infection. High titers of IgG antibodies in the absence of IgM antibodies are consistent with chronic latent infection acquired in the past. The IgM-capture enzyme-linked immunosorbent assay (ELISA) is more sensitive than the IgM-indirect fluorescent-antibody assay (IFA) test. However, IgM tests may be false-positive, and true-positive IgM tests may persist for a year or more. Therefore, to determine if infection occurred during pregnancy, additional tests, such as an anti-Toxoplasma avidity test, may be required at a reference laboratory.

Immunodeficient persons usually do not have measurable IgM antibodies, even in the presence of active disease. The diagnosis of central nervous system (CNS) toxoplasmosis in such persons is therefore based on clinical picture, typical CT scan or magnetic resonance imaging (MRI) showing multiple ring-enhancing hypodense nodules, and a positive IgG test. Brain biopsy is reserved for cases that fail to respond to an empiric trial of anti-*Toxoplasma* drugs.

The baby was diagnosed with congenital toxoplasmosis.

How is toxoplasmosis best treated?

Toxoplasmosis in immunocompetent persons rarely requires treatment, whereas infection in immunodeficient persons or in infants with congenital infections usually requires treatment. The combination of pyrimethamine and sulfadiazine is the treatment of choice. Folinic acid (leucovorin) is given to prevent bone marrow suppression. Treatment must be continued for the duration of immunosuppression and for life in AIDS patients whose immunity is not reconstituted by highly aggressive antiretroviral therapy (HAART).

For persons unable to tolerate the pyrimethamine and sulfadiazine combination, high doses of pyrimethamine (and leucovorin) and clindamycin are effective.

The management of toxoplasmosis acquired during pregnancy is controversial. Testing of newly pregnant women for *T. gondii* infection is not routinely done, and routine testing is not recommended by CDC or by the American College of Obstetricians and Gynecologists. To prevent fetal infection, one approach is to administer spiramycin (a macrolide antibiotic, which is concentrated in the placenta and is not harmful to the fetus). At the same time, amniotic fluid is submitted for PCR-based testing to determine whether fetal infection has occurred. If so, options may include pyrimethamine and sulfadiazine given after the 16th week of pregnancy (since pyrimethamine is potentially teratogenic) or consideration of terminating the pregnancy. If the fetus is shown to be uninfected, spiramycin is continued throughout pregnancy.

Different protocols exist for treatment of infants born with congenital infection. The most commonly recommended treatment is pyrimethamine and sulfadiazine plus leucovorin during the first year of life. In the present case, the child was treated for 6 months with pyrimethamine and sulfadiazine plus leucovorin. Human infection with the intracellular protozoan parasite *Toxoplasma gondii* occurs globally. Infection is usually subclinical or produces a mild illness, except in immunodeficient persons and fetuses infected in utero. Most infants with congenital toxoplasmosis appear healthy at birth but have a high incidence of developing serious ophthalmologic and neurologic sequelae during the next 20 years of life. Severe congenital toxoplasmosis may be apparent at birth or become apparent during the first 6 months of life. Chorioretinitis, intracerebral calcifications, and hydrocephalus, as in the present case, are typical features

The child was treated with pyrimethamine, sulfadiazine, and folinic acid for 6 months. She remains blind, and has developed moderate psychomotor retardation.

How could *Toxoplasma* infection have been prevented in this child?

Toxoplasma gondii may be transmitted transplacentally to the fetus if the mother acquired toxoplasmosis during pregnancy. There is almost no risk of transplacental transmission if the mother was infected prior to conception; accordingly, women with positive IgG antibody tests for toxoplasmosis at the onset of pregnancy are not at risk for developing acute toxoplasmosis. Women with negative IgG antibody tests during pregnancy should avoid eating insufficiently cooked or uncooked meat and should avoid ingestion of soil and water or food that may be contaminated with cat feces.

Transmission occurs by a) ingestion of tissue cysts in raw or insufficiently cooked meat, especially lamb, pork, and wild game; b) accidental ingestion of food, water, or soil contaminated with cat feces that contain infective oocysts; c) transplacental passage of infective tachyzoites; d) transfusion of infected white blood cells or transplantation of an infected organ; and e) laboratory accidents.

Prevention of toxoplasmosis is particularly important for uninfected (ie, seronegative) pregnant mothers, HIVinfected persons, and other immunocompromised patients:

- Avoid ingestion of raw or insufficiently cooked meat and poultry; cook meat to 160°F (71°C) or freeze to -4°F (-20°C). For more details on preventing toxoplasmosis, please see the Suggested Resources and Suggested Reading List.
- Avoid ingestion of environmental oocysts by avoiding contact with cat litter, soil, water, and vegetables potentially contaminated with cat feces.

Infection acquired by healthy persons is usually asymptomatic or may lead to painless lymphadenopathy or a mononucleosis syndrome. Maternal infection is usually unrecognized.

Disease in persons with depressed cellular immunity (eg, persons with AIDS, transplant recipients, persons receiving immunosuppressants) usually is due to reactivation of latent infection but can result from acute infection. Toxoplasmosis in these persons leads to lethal meningoencephalitis, focal lesions of the CNS, and less commonly, myocarditis or pneumonitis. The clinical picture may include headache, seizures, mental status changes, focal neurologic signs, and aseptic meningitis. Thirty to forty percent of AIDS patients with IgG antibodies to *T. gondii* (indicating chronic latent infection) develop active toxoplasmosis unless they take preventive medication.

Congenital infection occurs when a previously uninfected mother develops infection during pregnancy. Infection prior to conception, demonstrated by specific IgG antibodies, in nearly all cases guarantees against infection of the fetus. However, transplacental transmission occurs from mothers whose prior infections reactivate when they receive immunosuppressant medications or develop AIDS. Congenital toxoplasmosis may result in abortion, stillbirth, mental retardation, and retinal damage. Recurrent toxoplasmic chorioretinitis in children and young adults is frequently the result of congenital infection that was asymptomatic at birth.

Acute Hepatitis A: A Patient Scenario

While working in an emergency room, you are asked to see a 31-year-old Asian-American woman who has had fever, nausea, and fatigue for the past 24 hours. She also reports dark urine and has had 3 light colored stools since yesterday. She has previously been healthy and has no previous history of jaundice. Her physical examination shows a low-grade fever of 100.6°F/38.1°C, faint scleral icterus, and hepatomegaly. Her blood pressure and neurologic exam are normal and there is no rash. Initial laboratory studies show an alanine aminotransferase (ALT) result of 877 IU/L, aspartate amino transferase (AST) enzyme levels of 650 IU/L, an alkaline phosphatase of 58 IU/L and a total bilirubin of 3.4 mg/dL. White blood cell count is 4.6, with a normal differential; electrolytes are normal; the blood urea nitrogen level is 18 mg/dL; and serum creatinine level is 0.6 mg/dL. Pregnancy test is negative.

What should be included in the differential diagnosis of acute hepatitis?

- Viral infections:
 - hepatitis A, B, C, D, and E
 - varicella
 - cytomegalovirus
 - herpes virus
 - Epstein-Barr virus
- Bacterial infections:
 - typhoid fever
 - -Q fever
 - Rocky Mountain spotted fever
 - leptospirosis
 - secondary syphilis
 - sepsis
- Parasitic infections:
 - toxocariasis
 - liver flukes
- Drugs:
 - acetaminophen
 - isoniazid
 - rifampin
 - oral contraceptives
 - anti-seizure medications
 - sulfonamides
- Toxins:
- alcohol, carbon tetrachloride
- Autoimmune disease:
 - autoimmune hepatitis
 - systemic lupus erythematosus

What additional information would assist with the diagnosis?

- Has she traveled outside the United States recently?
- Does she use illicit drugs?
- Is anyone else in the household ill?
- How many sex partners has she had in the past 6 months?
- Does she have regular contact with animals?
- What medications is she taking?
- Has she ever had a transfusion?
- Does she drink alcohol?
- Does she take care of children?
- Has she ever received hepatitis B vaccination?
- Has she ever received hepatitis A vaccination?
- Did she receive immune globulin within the past 3 months?
- What is her occupation?

She has no children, and her boyfriend is not ill. She has been in a monogamous relationship with her boyfriend for 2 years. She was born in the United States; her parents immigrated to the United States from Taiwan in the 1950s. She works as a food preparer for a catering business. She returned 4 weeks ago from a 1-week vacation in Mexico (Mexico City and nearby areas), where she stayed with her boyfriend in several hotels. She drank only bottled water but ate both cooked and uncooked food at numerous restaurants while in Mexico, and she visited a family friend and her 3 young children in a Mexico City suburb.

She did not receive hepatitis A vaccine or immune globulin before going on vacation. She is not sure if she has received hepatitis B vaccine. She has not gone camping or hiking and had no recent tick exposures. She has never used illicit drugs, drinks alcohol rarely, and has never received a transfusion. She is taking oral contraceptives but no other prescription medication, and took 500 milligrams of Tylenol[®] once after onset of her current symptoms. She has a pet cat but no other animal exposures. She had chickenpox and mononucleosis during childhood.

How does this information assist with the diagnosis?

Lack of animal or tick exposures makes leptospirosis and Rocky Mountain spotted fever unlikely, and Q fever less likely. Yellow fever and typhoid fever are very unlikely with no history of travel to rural endemic areas, and assuming exposure occurred in Mexico, inconsistent with the long incubation period. Hepatitis A virus (HAV), hepatitis B virus (HBV), hepatitis C virus (HCV), and hepatitis E virus (HEV) infection are all possible diagnoses. A drug reaction to the oral contraceptive is a possible cause of hepatitis. The history of travel to an endemic area makes hepatitis A the most likely diagnosis.

What diagnostic tests are needed?

Specific diagnostic serologic studies are necessary to distinguish one form of viral hepatitis from another. Testing for total (IgG+ IgM) anti-HAV does not distinguish between a past history of hepatitis A virus infection and current infection and is not useful in diagnosing acute hepatitis A. Hepatitis A can be easily confirmed with an anti-IgM anti-HAV test. This test is widely available and results are usually available within 24 hours. A hepatitis panel is ordered, and results from such a panel are shown here. You obtain the following results from the serologic testing:

- Total anti-HAV: positive
- IgM anti-HAV: positive
- Total anti-HBc: positive
- IgM anti-hepatitis B core antigen: negative
- HBsAg: negative
- anti-HBs: positive
- anti-HCV: negative

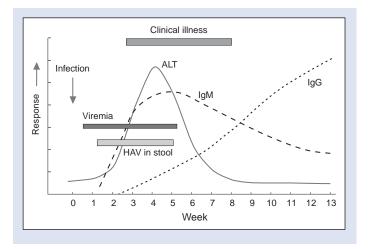
What is the diagnosis?

The diagnosis is hepatitis A. The hepatitis B serologic tests indicate past, resolved infection with no chronic infection. Acute hepatitis C is also possible; the appearance of anti-HCV may be delayed for as long as 9 months after exposure. However, with a confirmed diagnosis of hepatitis A, further testing for HCV RNA is not indicated at this point. Finally, note that hepatitis E is rarely reported in travelers, and results of serologic tests for hepatitis E virus (HEV) are difficult to interpret. Tests for HEV should only be performed if other more common causes of hepatitis have been excluded.

The incubation period for hepatitis A is 15–50 days, with an average of 28 days. The most common signs and symptoms associated with acute hepatitis A include jaundice, fever, malaise, anorexia, and abdominal discomfort. The illness can be severe and approximately 10% to 20% of reported cases require hospitalization. The likelihood of having symptoms with HAV infection is related to the person's age. In children <6 years of age, most (70%) infection is asymptomatic; if illness does occur it is not usually accompanied by jaundice. Older children and adults are more likely to have symptomatic disease, although jaundice may be absent in as many as one third of adults with HAV infection. In many developing countries in Asia, Africa, and Central and South America, infection is nearly universal during early childhood and is often asymptomatic.

What treatment is indicated?

There is no specific treatment for hepatitis A. Bed rest does not hasten recovery. Hepatitis A is never a chronic infection, although 10% to 15% of symptomatic persons have prolonged or relapsing disease lasting up to 6 months. While rarely fatal in younger persons, the case-fatality rate is nearly 2% among reported patients who are more than 50 years old. Following is a depiction of a typical course, including times of peak fecal excretion of HAV, liver function test abnormalities, and clinical symptoms.



How is hepatitis A virus transmitted, and who is at risk for this disease?

HAV is an RNA virus that only infects primates. HAV has a fecal-oral route of transmission and is easily transmitted person to person. HAV is also transmitted through contaminated food or water. Because HAV is present in the blood during acute infection, bloodborne transmission is also possible, but rare. The highest levels of HAV are found in the stool, and peak levels occur in the 2 weeks before onset of illness.

Groups at increased risk for hepatitis A include travelers to developing countries, men who have sex with men, and injecting and noninjecting drug users. In the United States, 4% to 6% of reported cases occur among international travelers, many of whom presumably acquired HAV infection from contaminated food or water. Approximately 50% of persons with hepatitis A do not report any known risk factors, and some of these infections may be from unrecognized transmission via HAV-contaminated food.

How might this illness have been prevented?

Persons planning to travel to an endemic region should receive hepatitis A vaccine or immune globulin before departure. Hepatitis A vaccination can be given to anyone 2 years of age and older, and has the advantage of providing long-term protection (at least 20 years). Hepatitis A vaccine is an inactivated HAV preparation; the first dose of vaccine provides protective anti-HAV levels within 30 days for >90% of vaccine recipients. Licensed hepatitis A vaccines available in the United States are considered to be equivalent in effectiveness, and include Havrix[®] (manufactured by Glaxo SmithKline), VAQTA[®] (Merck & Co.), and Twinrix[®] (combined hepatitis A and hepatitis B vaccine, Glaxo SmithKline). Vaccination is administered in a 2-dose schedule (0, 6 months) for Havrix[®] and VAQTA[®], and a 3-dose schedule (0, 1, 6 months) for Twinrix[®]. The second (or third) dose is provided to ensure protection in those who did not respond to the first dose of vaccine. Ninety-nine percent of vaccinees will be protected after 2 doses of vaccine.

For persons who present for hepatitis A immunoprophylaxis <30 days before departure to an endemic region and for children <2 years old, immune globulin (IG) is an effective means of preventing hepatitis A. IG is the appropriate immunoprophylaxis for children <2 years old. IG is a sterile preparation of concentrated antibodies (immunoglobulins) made from pooled human plasma. IG provides protection against hepatitis A for 3–5 months, depending on dosage, through passive transfer of antibody. Vaccine and IG may be given simultaneously.

Hepatitis A is the most common vaccine-preventable disease among travelers. The risk varies according to region visited and the length of stay, and is increased even among travelers who report observing measures to protect themselves against enteric infection or stay only in urban areas. In the United States, children account for approximately one third of reported travel-related cases.

What else needs to be done?

Cases of hepatitis A should be reported to the local health department immediately. The patient's boyfriend and any other household or sexual contacts whose last exposure to the patient was <14 days ago should be given IG. Screening for immunity before administering IG is not recommended in this situation because it is more costly than IG and would delay its administration. IG is not indicated for family members or friends not living in the household.

Prompt reporting of hepatitis A cases allows time to decide on a course of action and provide timely immunoprophylaxis when appropriate. Because this patient works as a food preparer, the health department will need to visit the establishment to assess the likelihood that her duties and hygiene practices pose a significant risk of food contamination. IG is often recommended for co-workers of commercial food handlers with hepatitis A. In addition, if she worked at any time during the 2 weeks before onset of jaundice to 1 week after onset, persons who ate food prepared or handled by this patient may be candidates for IG prophylaxis. Determinations of the need for IG prophylaxis are made on a case-bycase basis by experienced health department personnel. Again, immediate reporting of hepatitis A cases allows time to decide on a course of action and provide timely treatment and intervention when appropriate.

Norovirus Infection: A Patient Scenario

Nancy is a 25-year-old previously well graduate student who presents to the emergency department with a 12-hour history of nausea, diarrhea, abdominal cramping, and vomiting (about 6 episodes), malaise, and a low-grade fever. She describes her onset of symptoms as sudden.

Physical examination shows that Nancy is afebrile with a supine blood pressure of 123/74 mm Hg. She has a diffusely tender abdomen and is dehydrated. Stool examination is negative for occult blood.

What is the possible differential diagnosis for her chief complaint?

- Infectious gastroenteritis
- Food intoxication (noninfectious gastroenteritis)
- Inflammatory bowel disease
- Appendicitis
- Pelvic inflammatory disease

What additional information would assist with the diagnosis?

- Did anyone in her household experience similar illness within the week prior to onset of symptoms?
- Has she been in contact with anyone outside her household with similar symptoms within the previous week?
- Has she had such symptoms before?
- Does she know if anyone else became ill?
- Has she traveled outside the United States within the last month?
- Has she previously had a sexually transmitted diseases or does she have multiple sex partners?

Nancy reports that she rarely has diarrhea or vomiting. She also reports no contact with anyone who was ill in the past week, nor has she been out of the country in the past month. Her boyfriend, who does not live with her, has similar symptoms with an almost identical onset time. Both attended a wedding 2 days ago. The meal at the wedding reception, which was held at a local reception hall, was the only meal they shared in the past several days. Nancy does not know if anyone else who attended the wedding became ill. Nancy reports that she has no history of a sexually transmitted disease and that she and her boyfriend have a monogamous sexual relationship.

How does this information assist with the diagnosis?

Based on the rapid onset of symptoms, Nancy's reported past history of good health, and the fact that her boyfriend has an almost identical history, inflammatory bowel disease, appendicitis, and pelvic inflammatory disease are the least likely diagnoses.

Food intoxication is also not very likely. Assuming that the wedding reception was the source of the toxin, and this was their most recent common meal, the time from exposure to onset of symptoms is too long. Toxins usually cause illness within minutes to hours after ingestion.

The most likely diagnosis is infectious gastroenteritis. There is a possibility that Nancy's and her boyfriend's illness may be associated with an outbreak of gastroenteritis.

What additional information would assist with the identification of the etiologic agent?

- What sorts of foods were served at the wedding reception?
- When did the couple last share a meal prior to the wedding reception?
- Has an outbreak of gastroenteritis associated with this reception has been reported to the local health department?. The health department may be able to aid in determining what the etiologic agent was if it is currently investigating the outbreak.

At the wedding, the couple had a choice of meal. Nancy had lobster tail and filet mignon. Her boyfriend had chicken. They both consumed stuffed mushrooms, salad, and hors d'oeuvres preceding the main meal. For dessert they both had wedding cake and fresh fruit. Both drank wine or beer during the reception.

The couple attended a barbecue the previous week. This outing was a function sponsored by Nancy's employer. Nancy tells you that none of her co-workers have been ill with vomiting and diarrhea.

You place an inquiry with the local health department about the possible outbreak. The health department notifies you that an investigation is currently under way. Illness has also been reported among 75% of attendees at a wedding the day before the one Nancy attended, at the same reception hall. The only common food between the 2 weddings is the salad, and the health department currently suspects a food handler who worked during both weddings who was experiencing diarrhea. Most patients have reported nausea, vomiting (about 90%), and diarrhea (70%), with some fever, malaise, headache, chills, and abdominal pain. The mean incubation period for those who have reported illness is 28.6 hours, with a mean duration of 31.8 hours.

The health department suspects viral gastroenteritis caused by a norovirus. A norovirus is suspected because of the rapid onset of symptoms, the short 36-hour incubation period and relatively short duration of illness, the absence of bloody diarrhea, and the high percentage of vomiting. Bacterial cultures are negative for enteric pathogens on stool samples collected thus far.

What are the complications of norovirus infection?

Noroviruses are common causes of self-limiting acute gastroenteritis, with illness frequently lasting no longer than 60 hours. They commonly cause outbreaks in such settings as restaurants, catered events, cruise ships, schools, and nursing homes. The viruses can be spread person to person through the fecal-oral route, through contaminated food or water, or by raw or undercooked shellfish.

How should norovirus infections be managed?

There is no antiviral agent that can be used to treat norovirus infections. Supportive care such as oral or intravenous fluids for rehydration should be provided.

To reduce the spread of illness, patients should be educated to use good hand washing practices, particularly after using the bathroom and before preparing and handling food.

The health department requests that a stool sample be collected. The sample should be collected in a sterile container without transport media, and kept at 4°C (40°F) until shipped. The sample should be shipped on ice packs to the local health department laboratory for testing. The health department also asks you to encourage Nancy's boyfriend to submit a stool sample.

How could this norovirus infection have been prevented?

The food handler with diarrhea should not have returned to work for at least 24–48 hours after symptoms subsided.

Proper hand washing procedures can prevent the spread of the virus between persons. Hands should be washed under warm water with soap for approximately 15 seconds to prevent fecal-oral transmission.

Antibiotic-Resistant Salmonellosis: A Patient Scenario

Andrea brings her 3-year-old son, Marcus, to your office with a 2-day history of low-grade fever, nausea, and 6–8 watery stools per day. Marcus has also been complaining of abdominal pain and feeling tired. He has been eating and drinking less than usual. His medical history is remarkable for recurrent otitis media, for which he was prescribed oral antibiotics 10 days prior to this visit.

Physical examination reveals a well-developed boy who appears fatigued. Vital signs are remarkable for low-grade fever (99.5°F/37.5°C). He does not have signs of dehydration. His otitis appears resolved and he has a normal cardiopulmonary exam. The abdominal exam reveals hyperactive bowel sounds, mild diffuse tenderness, and stool negative for occult blood.

What is the differential diagnosis for Marcus' chief complaint?

- Infectious gastroenteritis
- Appendicitis
- Celiac disease
- Inflammatory bowel disease
- Antibiotic-associated colitis

What additional information would assist with the diagnosis?

- Has he had similar symptoms before?
- Does he attend child care? If yes, have other children attending the same care facility been ill with similar symptoms?
- Has the child recently consumed a meal outside his home; eg, at a birthday party or restaurant?
- Do other members of the household or close acquaintances have diarrhea or bloody diarrhea?

- Has he traveled in the month prior to the onset of illness? If yes, where?
- Has he had contact with pet reptiles or farm animals or visited petting zoos in the week prior to his symptom onset?

Marcus has not had similar episodes of diarrhea in the past. He attends preschool and is cared for by his grandmother after school in her home. He last visited a petting farm 3 months prior to this illness. Their family returned the previous day from a 5-day Caribbean cruise. Marcus was diagnosed with otitis media 4 days prior to their departure and was prescribed a 1-week course of oral antibiotics. Andrea has had nausea and 3–4 loose stools per day for the previous 2 days. She has not had any fever, abdominal pain, or vomiting. Marcus' father and two sisters also traveled on the cruise and are asymptomatic. None of the family members took prophylactic antibiotics for travelers' diarrhea during the cruise.

How does this information assist with the diagnosis?

The additional history suggests that Marcus' and Andrea's illness may be an infectious gastroenteritis related to their recent travel. Antibiotic-associated colitis caused by *Clostridium difficile* infection must be considered since the child was prescribed antibiotics for otitis 8 days prior to this illness. Given the recent onset, travel history, and his mother's symptoms, it is unlikely that appendicitis, celiac disease, or inflammatory bowel disease are the etiologies of Marcus' illness.

The most likely diagnosis is infectious gastroenteritis.

What additional historical information will assist in the identification of the etiologic organism?

- What foods did Marcus and Andrea consume in the previous week? In particular, which foods/ beverages did they consume that the other family members did not?
- Did either Marcus or Andrea consume undercooked meats, runny eggs, unpasteurized milk, raw shellfish, or untreated water?
- Is there a reptile in the home?
- Marcus was prescribed antibiotics for otitis media 1 week prior to the onset of his gastrointestinal symptoms. Has Andrea been prescribed antibiotics during the month prior to the onset of her diarrheal illness?

• Have there been other cases of diarrhea recognized in the cruise ship travelers, in their community, or at Marcus' school?

An open-ended food history reveals multiple common meals eaten by Andrea and Marcus. Andrea denies the consumption of unpasteurized milk, raw shellfish, and undercooked meats. She does report that, unlike the rest of the family, she and Marcus used to wake up early enough to enjoy the breakfasts served on board the cruise. Breakfast served on the cruise consisted of a choice of French toast or pancakes with fruit compote, scrambled eggs or omelets made to order, potatoes, and fresh fruit along with a choice of beverages, including milk, coffee, and tea. Andrea complained that the eggs were occasionally runny. Several fellow passengers told Andrea at breakfast that they were experiencing vomiting and diarrhea. Andrea and Marcus ate the remainder of their meals with the entire family. They did not drink any untreated water or eat items purchased from street vendors at ports of call. In response to your other questions, Marcus does not have a reptile at home. Andrea has not been prescribed antibiotics for more than 1 year. The family lives in a city and has access to municipal water.

Based on the additional historical details, it appears that many people on board the cruise were experiencing symptoms of vomiting and diarrhea. This suggests an outbreak of infectious gastroenteritis that may be related to a common food or water source on the ship. The etiologic agent may be bacterial, viral, or parasitic. The most likely bacterial organisms causing this diarrheal illness are Campylobacter jejuni, Escherichia coli, Shigella species, and Salmonella. C. jejuni is the most common bacterial cause of diarrheal illness in the United States. Outbreaks of C. jejuni have been linked to raw milk, poultry, eggs, and water. Enterotoxigenic E. coli (ETEC) is recognized as the most common cause of "travelers' diarrhea" and can be transmitted via food or water. Salmonella is an important bacterial cause of foodborne illness, ranking just behind C. jejuni in its frequency. Vehicles most commonly implicated in foodborne outbreaks of salmonellosis include beef, poultry, produce, eggs, pork, and dairy products. Large waterborne outbreaks of salmonellosis have occurred rarely.

Why is identification of the cause of the diarrhea important?

Identification of the cause of diarrhea in these two cases is important because of the impact on treatment, identification of related cases, and detection of an outbreak and identification of the responsible vehicle. Stool cultures should be performed to detect common bacterial pathogens such as Campylobacter, Salmonella, Shigella, or E. coli O157:H7. Antimicrobial susceptibility results can guide antibiotic therapy if a resistant organism is detected. Additional testing may be conducted to detect nonbacterial organisms. Stool examination for ova and parasites (O&P) will reveal parasitic causes of foodborne and waterborne illness such as Cyclospora cayetanensis. Rotavirus infection, one of the most common etiologies of pediatric diarrhea, may be diagnosed with enzyme immunoassay (EIA). The presence of fecal leukocytes suggests bacterial infection but may be found in other infectious or inflammatory states. Testing for the presence of Shiga toxin to detect infection with enterohemorrhagic E. coli (EHEC) would be appropriate if Marcus or Andrea had bloody diarrhea.

What approaches would you take to treating Marcus' and Andrea's illness? Are antibiotics indicated for both Marcus and Andrea? What other therapeutic measures are useful for the management of diarrheal illness?

Because Andrea's symptoms are mild, she does not wish to receive antibiotics. For Marcus, you prescribe trimethoprim-sulfamethoxazole at appropriate doses. You encourage Andrea to monitor for worsening fever, diarrhea, vomiting, and dehydration. You obtain stool specimens for culture and O&P from both Marcus and Andrea to confirm the etiologic agent.

The primary goal of therapy for Marcus and Andrea is the maintenance of adequate hydration and electrolyte balance. A commercial oral rehydration solution (ORS) may be used, particularly for Marcus, to provide glucose and salts. You encourage Andrea to give Marcus ORS to prevent dehydration. Bismuth subsalicylate or loperamide may be used to decrease the number of unformed stools and shorten the duration of diarrhea, although neither is available over the counter for children of Marcus' age. Loperamide should not be used in those patients who develop fever or dysentery.

Finally, empiric antibiotic therapy can be used to treat "travelers' diarrhea," which is most commonly caused by ETEC, after obtaining the stool samples but prior to obtaining results of stool cultures. Three days after the initial visit, Andrea feels better with fewer stools per day, but Marcus has had worsening vomiting and diarrhea. He has had several episodes of high fever and has not been drinking ORS adequately. In the office, Marcus is febrile (102°F/38.8°C) and appears dehydrated with dry mucous membranes and decreased skin turgor. No significant change is noted in the abdominal examination. You admit Marcus for intravenous hydration and encouragement of oral rehydration and consider a change in antibiotic therapy. Because of the progressive systemic nature of his illness, you also obtain blood cultures at this time.

What information will guide your therapy at this time?

The use of intravenous fluids to improve volume status is reasonable given Marcus' inability to maintain hydration with ORS. However, during hospitalization, he should be encouraged to resume drinking ORS as early as possible. The decision to change from oral to intravenous antibiotics may be based on Marcus' increased vomiting and on his clinical decline. The choice of antibiotics should reflect the results of stool culture and antimicrobial sensitivities.

The laboratory reports the growth of Salmonella Typhimurium from Marcus' stool cultures. Susceptibility testing reveals an organism resistant to multiple antibiotics, including ampicillin and sulfamethoxazole. Multidrugresistant S. Typhimurium has been on the rise in the United States since the early 1990s and now accounts for at least 25% of these isolates. Definitive type 104 (DT 104), the most common phage type of multidrug-resistant S. Typhimurium, may be responsible for more invasive disease than other phage types. In an outbreak, resistant organisms appear to cause more cases than do sensitive strains. Marcus' recent exposure to antibiotics for otitis media likely increased his susceptibility to Salmonella infection, perhaps by decreasing the usual protection offered by normal bowel flora, and thus decreasing the infectious dose necessary to cause illness. In addition, he was placed at increased risk for infection with a resistant strain of S. Typhimurium if he was exposed while still taking the antibiotic.

Treatment of *Salmonella* gastroenteritis with antibiotic therapy is controversial because of the resulting increase in asymptomatic carriage, particularly among children less than 5 years of age. However, given the systemic nature of his illness, you choose to treat Marcus with several days of an intravenous third-generation cephalosporin. This is a reasonable choice in light of the antimicrobial resistance and the reluctance to use fluoroquinolones in the pediatric population.

Should these cases be reported to the local health department? What are the public health implications of these two cases of salmonellosis?

Salmonellosis is a nationally notifiable disease, and most states require clinicians to report cases to local or state public health agencies. The health department and its public health partners can conduct studies to determine whether these cases indicated an outbreak of salmonellosis aboard the cruise ship. If an outbreak is confirmed, additional investigation is necessary to identify the contaminated food or the ill food worker infected with Salmonella, and whether there were correctable food-handling errors. If a food vehicle is identified, traceback and recall may be necessary to remove it from the market and prevent the occurrence of other cases. Given the increasing prevalence of drugresistant strains of S. Typhimurium, public health laboratories may perform bacteriophage typing or pulsed-field gel electrophoresis (PFGE) to further characterize the drugresistance patterns of these organisms. Reporting of these cases will contribute to essential nationwide surveillance of salmonellosis, foodborne outbreaks, and antimicrobial resistance.

What prevention measures will you recommend to Marcus and Andrea? Are repeat stool cultures necessary?

To prevent Salmonella infections, all meat and egg dishes should be fully cooked. Andrea can purchase eggs that are pasteurized in the shell, and irradiated ground beef and poultry to reduce the risk of contamination. Basic food safety practices in the kitchen can also help prevent such infections, such as refrigerating leftovers promptly, washing hands and utensils after contact with raw meat and poultry, and keeping raw meat and poultry separate from ready-to-eat foods. Marcus and Andrea should be reminded to wash their hands with warm running water and soap after using the bathroom and before and after meals to avoid transmitting the infection to others. Marcus is likely to have prolonged carriage of Salmonella in the intestines. While he may return to preschool as soon as he is feeling well enough to do so because direct spread from one child to another is rare, clinicians should defer to their local health departments regarding their clearance policies for convalescing children attending preschool.

With adequate hydration and your chosen antimicrobial therapy, Marcus will likely recover fully from this diarrheal illness without residual complications.

Unexplained Illness: A Patient Scenario

You have been a primary care practitioner in Manhattan, New York, for several years. Jack, a 29-year-old otherwise healthy male, has been your patient for the past year. At 8:00 a.m. he calls your triage nurse complaining of a very sudden onset of nausea, cramps, coughing, and sweating. The nurse is concerned about the suddenness of onset and wants to know what you would like to do.

Should you have him call again later if he does not improve? Should you have him make an acute-visit appointment, or should you send him to the emergency room?

You are concerned about the suddenness of the onset of symptoms but not the severity, so you decide to have him come to the office immediately.

Jack presents in your office 30 minutes later. In addition to nausea, cramps, coughing, and sweating, his eyes have begun to tear uncontrollably and he complains of having had difficulty breathing while en route to the office. Upon arrival, he immediately asks to use the bathroom.

Jack reports that he started his morning routine as usual with a run. Upon returning home, he finished drinking the bottle of water he had purchased earlier from the local deli and began to get ready for work. By the time he had finished showering and dressing, he began to feel sick to his stomach. He then developed cramping but no diarrhea. Shortly thereafter, he began to have bouts of coughing uncontrollably. He does not know when the sweating started. He states that he had difficulty breathing while en route to the office, and that the tearing just started. He denies vomiting, hemoptysis, hematuria, bright red blood per rectum (BRBPR), chills, fever, headache, myalgia, arthralgia, or diarrhea. Jack also denies the use of any medication, other drugs or alcohol. "That stuff rots your gut."

Jack reports that he finished his run at about 7:00 a.m. It is now 9:00 a.m..

Despite having just urinated, he states that he must go again and immediately. However, Jack experiences incontinence on his way to the bathroom. Upon his return to the exam room, you notice a slight tremor in his left arm. He states that this has only just begun.

What preliminary diagnosis can you make at this point?

- An anxiety attack
- A viral syndrome
- A potential foodborne illness
- Anticholinergic poisoning

You are not ready to reach a conclusion at this point, so you move to a physical exam and observe the following: Objective:

, Respiration rate: 20 BP: 92/60 mm Hg. Heart rate: 50 Temperature: 98.6°F (37°C)

You note that Jack is anxious but oriented to time, place, and person. His head, ears, eyes, nose, throat (HEENT) examination shows bilateral miosis and decreased reactivity. There are no signs of trauma or bleeding. His heart has regular rate and rhythm, no murmur, and good perfusion. Radial and dorsal pulses are 2+. His lung examination reveals scattered wheezing. His abdomen is soft, nontender, not distended, with increased bowel sounds, and no mass. Extremities appear within normal limits. The neurologic exam reveals the slight tremor in his left arm, slightly slurred speech, excessive salivation, and transient fasciculations in both upper extremities. You note negative Babinski and his cranial nerves (CN) 2-11 appear intact, while CN 12 appears slightly abnormal.

What other information would assist with the diagnosis?

More history from Jack, including most recent activity and diet.

You now seek additional history. Jack lives alone and does not believe that he has been in contact with anyone who is ill. He works in an office as a lawyer. His run takes him up 5th Avenue and then over to 3rd Avenue, then back home. He does not run through Central Park. He does not have plants and does not garden as a hobby. His most recent meal was the night before, about 10 hours prior to the onset of his symptoms. It consisted of boiled pasta, steamed broccoli, and olive oil. He prepared the meal himself. He states that he carefully washed the broccoli, the oil was from a bottle he opened last week, and the pasta was from a box he had already used 2 days before. All he had to drink was tap water with dinner last evening and the bottled water from this morning.

Jack's presentation appears to involve which of the following systems?

- Autonomic nervous system
- Lymphatic system
- Central nervous system

The signs and symptoms in Jack's presentation predominantly involve increased autonomic responses, and are perhaps progressing to include the central nervous system as well. You decide that immediate treatment is called for and order oxygen, atropine, and pralidoxime (2-PAM). Given that Jack does not appear to have been exposed dermally, the most likely route appears to have been oral. Therefore, you also appropriately begin an IV with normal saline

What is the initial diagnosis?

This presentation is not consistent with bacterial, viral, or parasitic food poisoning. While the signs and symptoms indicate acute organophosphate poisoning, the history provides no indication, and indeed seemingly contradicts this theory because of the lack of exposure. There has been no exposure to places where organophosphates are typically used, such as on lawns, house plants, and parks. Nevertheless, Jack has presented with a fairly classic case of organophosphate poisoning. Therefore, ingestion must be considered. Since you have no suggestion of deliberate ingestion on Jack's part, it must be assumed that he has consumed it unintentionally.

Organophosphate poisoning has an onset of 30 minutes to 2 hours. Jack has actually made it easy to identify the most likely source: the only thing he has consumed in 10 hours is water. The broccoli could have had pesticides on it that may not have been removed when Jack washed it, but then he would have developed his symptoms during the night. Taking into account the temporal relationship between his ingestion of the bottled water and the onset of his symptoms, the bottled water seems the most likely candidate.

Given this information, what are key questions you should consider?

- Is the water truly contaminated?
- If it is, how did it become contaminated?
- Who else may have ingested it?
- Who else is at risk?
- What action should be taken?

You realize that if your diagnosis and conclusions are correct then a public health hazard may exist. Two things need to be done. First, the health department must be contacted, and second, tests need to be done that will confirm your diagnosis. While the usual work-up for organophosphate poisoning is clinical diagnosis, there are assays available to measure cholinesterase activity in plasma and red blood cells. It is also possible to detect some pesticides in urine. You decide to order both tests as this will provide the greatest insight into what the possible exposure is for other people in Jack's building, neighborhood, or even his city.

When communicating with the local public health department, whom should you ask to speak to concerning this situation?

- The medical epidemiologist?
- The medical director?
- The infectious disease officer?

You ask to speak with the medical director. You present Jack's case, making careful note of the time course, and also inform the medical director of your suspicions of the source. The medical director takes this information and agrees with your concerns. She then asks you to speak with the chief epidemiologist so that an investigation can begin.

In many large cities, there is a city health department; in smaller cities or towns, it will usually be necessary to contact the local or state health department. Try to match the level with the greatest number of people who may become affected. Other persons who may be of immediate help if you cannot reach the medical officer are the epidemiologist or even an environmental health officer. These people will most likely know what to do with the information you have.

Most health departments across the country have been working to increase their knowledge or at least their awareness of the possibility of intentional contamination. Many have also created positions solely devoted to this task. Therefore, it is possible that you will be directed to such an individual.

The health department initiates an investigation that includes testing the water; looking for other cases of organophosphate poisoning; interviewing the patient; notifying other parts of the public health system, including law enforcement, CDC, and the state health department. They may even issue a public notice.

There is another possible cause for the case you have just seen: sarin gas can cause a similar presentation. If sarin gas had been sprayed into the air, it is possible that Jack could have respiratory exposure to the nerve gas.

If this were true, how would it change what you did?

Persons exposed to sarin, and possibly other nerve agents, will have a clinical presenation similar to those with organophosphate poisoning. Hence, medical management will likely be similar.

Finally, you are gratified to have helped detect a possible act of contamination that could potentially harm or even kill a great many people. Afterward, while making rounds in the hospital that day you are told by a colleague that a number of runners from a 5K race in Central Park this morning and tourists visiting the Empire State Building were brought to the emergency room complaining of sudden onset of nausea, cramps, and coughing. It was reported that all had been drinking bottled water.

Clinical Vignettes: What's Your Call?

The following clinical vignettes are provided for your selfevaluation. All are possible situations that may present at your practice. The "Diagnostic Considerations" section and the tables of etiologic agents that are also part of this primer will provide the information necessary for you to adequately address these clinical situations. Note that these vignettes include both infectious and noninfectious forms of foodborne illness.

For the following clinical vignettes, choose the best answer from the choices listed at the end of the vignettes:

A—likely diagnosis; choose the best possible answer listed on "answer selections" page under **A** selections.

 \mathbf{B} — most appropriate choice to confirm the diagnosis (there may be more than one correct answer — list all of them). Choose from the possible answers listed on "answer selections" page under the \mathbf{B} section.

Finally, decide whether the situation warrants reporting to the local or state health department.

Clinical Vignettes

I. You receive a long-distance call from a patient who is an outdoorsman. He is with a group that collected and ate some wild mushrooms less than 2 hours ago. Several members of the group have since developed vomiting, diarrhea, and some mental confusion.

A — likely diagnosis: _

B — most appropriate test to confirm etiology/follow-up action:

Report to the health department? ____Yes ____No

II. A newborn child has symptoms of sepsis. Cerebrospinal fluid studies are consistent with meningitis. The mother had a flu-like syndrome prior to delivery.

A — likely diagnosis: _

B — most appropriate test to confirm etiology/follow-up action:

Report to the health department? ____Yes ____No

III. This patient has just returned today from Latin America following a 2-day business trip. He reports having eaten several meals of fish that he bought from street vendors around his hotel. He feels very ill with profuse, watery diarrhea, and vomiting.

A — likely diagnosis: _

B — most appropriate test to confirm etiology/follow-up action:

Report to the health department? ____Yes ____No

IV. An 18-month-old child is brought to your office with fever, bloody diarrhea, and some vomiting. She has been drinking unpasteurized milk in the last 48 hours. No other family members are ill.

A — likely diagnosis: _____ B — most appropriate test to confirm etiology/follow-up action: _____

Report to the health department? ____Yes ____No

V. A patient calls and states that he and several family members are ill with severe vomiting. They ate at a church picnic 4 hours earlier.

A — likely diagnosis: ___

B — most appropriate test to confirm etiology/follow-up action:

Report to the health department? ____Yes ____No

VI. A patient calls and states that most family members have developed severe vomiting, about 1 hour after eating at a picnic. They ate barbecued beef, chips, potato salad, and homemade root beer. Some are complaining of a metallic taste.

A — likely diagnosis: _____ B — most appropriate test to confirm etiology/follow-up action: _____ Report to the health department? ___Yes ___No

VII. A patient has had chronic intermittent diarrhea for about 3 weeks. There is no fever or vomiting and no blood in the stool. The patient travels to Latin America and Eastern Europe frequently, most recently 2 weeks ago.

A — likely diagnosis: _____

B — most appropriate test to confirm	etiology	/follow-up
action:		
Report to the health department? _	Yes _	No

VIII. The parents of a 6-month-old infant are concerned because she is listless and weak. The infant is feeding poorly, has poor head control, and is constipated. There is no fever or vomiting.

A — likely diagnosis: _

B — most appropriate test to confirm etiology/follow-up action:

Report to the health department? ____Yes ____No

IX. A businessman who travels frequently is ill with fatigue, jaundice, abdominal pain, and diarrhea. About 1 month ago, he returned from an international trip during which he consumed raw oysters.

A — likely diagnosis: ___

В —	most	app	orop	oriate	test	to conf	irm	etiology	/follow-up
action	1:								

Report to the health department? ____Yes ____No

X. Several members of a single family are ill with abdominal cramps and watery diarrhea. They just returned from visiting friends on the East Coast of the United States, where they consumed raw oysters 48 hours ago.

A — likely diagnosis: _

B — most appropriate test to confirm etiology/follow-up action:

Report to the health department? ____Yes ____No

XI. A minister at a local church calls to report that many members began experiencing watery diarrhea on the morning after the annual turkey dinner fundraiser. Some people also reported nausea and abdominal cramps, but no one has fever or bloody stools.

A — likely diagnosis: _

B — most appropriate test to confirm etiology/follow-up action:

Report to the health department? ____Yes ____No

XII. You receive a long-distance call from a patient on a fishing vacation off the coast of Belize. Her family has been eating a variety of local fish and shellfish that they caught. She reports that several family members developed abdominal pain, severe diarrhea, and weakness the morning after they consumed the seafood for dinner. One family member began having difficulty speaking later on that same night.

A — likely diagnosis: _

B — most appropriate test to confirm etiology/follow-up action:

Report to the health department? ____Yes ____No

XIII. A family in a rural community is worried that their father may be having a stroke. He is complaining of double vision and is having trouble swallowing. They have a large garden and eat home-canned vegetables.

A — likely diagnosis: _____ B — most appropriate test to confirm etiology/follow-up action: _____

Report to the health department? ____Yes ____No

XIV. A 2-year-old child who attends day care presents with abdominal cramps and severe bloody diarrhea, which has been present for 2 days. He has no fever.

A — likely diagnosis: _____

B — most appropriate test to confirm etiology/follow-up action:

Report to the health department? ____Yes ____No

XV. Susan tells you that she has had diarrhea, nausea, and abdominal cramping for almost 12 hours now. She also presents with malaise and a low-grade fever and informs you that as far as she can tell, the symptoms developed very suddenly. Stool examination is negative for occult blood. Susan informs you that her good friend is also sick and they both attended a company picnic less than 2 days ago.

A — likely diagnosis:

B — most appropriate test to confirm etiology/follow-up action:

Report to the health department? ____Yes ____No

XVI. Sally arrives at your office with acute gastrointestinal illness characterized by diarrhea, abdominal cramps, chills, fever, and body aches. She also informs you that about 3 days before she started getting sick, she had consumed raw ground beef that was seasoned with onions and an herb mix.

A — likely diagnosis: _

B — most appropriate test to confirm	etiolog	y/follow-up
action:		
Report to the health department?	Yes	No

XVII. James presents to the emergency room with a lowgrade fever and complaining of fatigue and nausea for the past 24 hours. He also describes his urine as being dark and states that he has had 4 bowel movements in the past 24 hours, all of which were light colored. Upon further questioning, James says that he has no history of jaundice and that he returned from a business trip to the Philippines a month ago.

A — likely diagnosis: _

B — most appropriate test to confirm etiology/follow-up action:

Report to the health department? ____Yes ____No

XVIII. You are halfway through your shift in the ER. There are four patients, two adults and two children, with a history of nausea, vomiting, abdominal pain, and profuse (especially in the children) watery diarrhea in the absence of fever. They each report that these symptoms began 5 days ago and resolved after 1 day. They had all been symptom free for 3 days, but now the symptoms have returned. There is also a new onset of jaundice and bloody diarrhea. Lab results indicate elevated LFTs. The patients do not know each other, but all report eating hamburgers several hours before the initial onset of symptoms.

A — likely diagnosis:

B — most appropriate test to confirm etiology/follow-up action:

Report to the health department? ____Yes ____No

XIX. A mother has brought in a 5-month-old child with apparent blindness. She reports that the child had been healthy until the past month when the vision problems appeared. The mother states that she had been well during the pregnancy, but further questioning reveals that the mother had two young cats at home for which she was the sole care provider. The cats were given away just before the birth of the child because of concerns about the child being smothered by the cats.

A — likely diagnosis:_

B — most appropriate test to confirm etiology/follow-up action:

Report to the health department? ____Yes ____No

Answer Choices

A: Choose from any of these possible etiologies:

- 1. Intoxication from preformed toxins of *Staphylococcus aureus* or *Bacillus cereus*
- 2. Intoxication from toxins produced *in vivo* by *Clostridium perfringens*
- 3. Salmonella or Campylobacter are possible.
- 4. E. coli O157:H7
- 5. Noroviruses, *Vibrio parahaemolyticus*, and other *Vibrio* infections
- 6. Vibrio cholerae infection
- 7. Botulism must be ruled out
- 8. Listeria monocytogenes sepsis
- 9. Cryptosporidium parvum
- 10. Cyclospora cayetanensis
- 11. A form of metal poisoning
- 12. A form of mushroom poisoning
- 13. Likely fish/shellfish toxin
- 14. Giardia lamblia
- 15. Trichinella spiralis
- 16. Hepatitis A virus
- 17. Congenital toxoplasmosis
- 18. Intentional amanitin poisoning

B: Choose from any of these following tests/actions

- 1. Clinical diagnosis; laboratory tests may not always be indicated.
- 2. Generally detected on routine stool cultures.
- 3. Generally, a reference laboratory is needed to identify the toxin from food, stool, or vomitus.
- 4. Important to identify causative organism for public health reasons.
- 5. Send stool samples to health department (*Vibrio cholerae*, other vibrios, *E. coli* O157:H7, special toxin tests, *Clostridium perfringens*, *Clostridium botulinum*).
- 6. Not detected by routine stool cultures (*E. coli* O157:H7, *Vibrio cholerae*, other vibrios).
- 7. Should test for viral agents.
- 8. For cysts, ova, and parasite detection, at least 3 stool samples must be collected. Sometimes the organism may still be missed.
- 9. Test for appropriate metal.
- 10. Special test needed to identify a fish toxin.
- 11. Consult a mycologist to identify the mushroom.
- 12. Blood culture is the best source for diagnosis.
- 13. Blood test helpful to identify the agent.
- 14. May need acute and convalescent serum or viral cultures.

- 15. Isolation of *T. gondii* from infant blood. PCR of white blood cells or CSF, or IgM and IgA serology, performed by a reference laboratory.
- 16. Rapid and aggressive antitoxin therapy. There is no single effective antidote at this time, but silibinin (with penicillin G) and N acetyl cysteine are showing promise. Plan for hepatic and renal failure.

Answers

Question number	Choice(s) for A	Choice(s) for B	Report to Health Dept.?
I	12	11	Yes
11	8	12	Yes
	6	5,6	Yes
IV	3,4	2	Yes
V	1	1,3	Yes
VI	11	9	Yes
VII	14	8	Yes
VIII	7	5	Yes
IX	16	13,7,14	Yes
Х	5	5,6,7	Yes
XI	2	1,5	Yes
XII	13	10	Yes
XIII	7	3,5	Yes
XIV	4	5,6	Yes
XV	3,5	5,6,7	Yes
XVI	3,4	2	Yes
XVII	16	13,7,4	Yes
XVIII	18	16,	Yes (intentional contamination?)
XIX	17	15,13	Yes

Suggested Resources

General Information

CDC Food Safety Information http://www.cdc.gov/foodsafety Continuing Medical Education (CDC) http://www2.cdc.gov/mmwr/cme/conted.html US Government Food Safety Information Gateway http://www.foodsafety.gov Fight BAC!TM Education Campaign http://www.fightbac.org Foodborne Illness Education Information Center http://www.nal.usda.gov/fnic/foodborne/foodborn.htm Public Health Partners - Networks and Resources http://www.cdc.gov/other.htm Bad Bug Book (FDA) http://www.cfsan.fda.gov/~mow/intro.html Travelers' Health Information (CDC) http://www.cdc.gov/travel

Listing of foodborne diseases, pathogens and toxins (CDC) http://www.cdc.gov/foodsafety/disease.htm Searchable database: U.S. Foodborne Disease Outbreaks,

1990–1995 www2.cdc.gov/ncidod/foodborne/fbsearch.asp Terrorism and Public Health (CDC) http://www.bt.cdc.gov/

Professional Organizations

American Academy of Family Physicians http://www.aafp.org American Medical Association (AMA) http://www.ama assn.org Infectious Diseases Society of America http://www.idsociety.org American Academy of Pediatrics http://www.aap.org American Nurses Association (ANA) http://www.nursingworld.org American Association for Health Education http://www.aahperd.org American Dietetic Association http://www.eatright.org

State and Local Organizations

Association of Food and Drug Officials http://www.afdo.org Association of State and Territorial Directors of Health Promotion and Public Health Education http://www.astdhpphe.org Association of Public Health Laboratories (APHL) http://www.aphl.org Association of State and Territorial Health Officials (ASTHO) http://www.astho.org Council of State and Territorial Epidemiologists (CSTE) http://www.cste.org National Public Health Information Coalition (NPHIC) http://www.nphic.org National Association of County and City Health Officials (NACCHO) http://www.naccho.org

Government

US Department of Agriculture (USDA) Food Safety and Inspection Service http://www.fsis.usda.gov US Department of Health and Human Services (DHHS): Centers for Disease Control and Prevention http://www.cdc.gov US Food and Drug Administration http://www.fda.gov Center for Food Safety and Applied Nutrition (CFSAN) Information for Health Professionals http://www.cfsan.fda.gov/~dms/hpro-toc.html State and local government agencies http://www.foodsafety.gov/~fsg/fsggov.html Role of Government Agencies in Food Safety http://vm.cfsan.fda.gov/~lrd/foodteam.html Gateway to government food safety information http://www.foodsafety.gov

Reports and Journals

CDC, Morbidity and Mortality Weekly Report http://www.cdc.gov/mmwr CDC, Emerging Infectious Diseases Journal http://www.cdc.gov/eid

Food Safety Education Resources

An Ounce of Prevention Keeps the Germs Away http://www.cdc.gov/ncidod/op Attention Pregnant Women: What you can do to keep germs from harming you and your baby http://www.cdc.gov/foodsafety/edu.htm Consumer Advice from CFSAN http://www.cfsan.fda.gov/~lrd/advice.html Cooking for Groups: A Volunteer's Guide to Food Safety www.fsis.usda.gov/OA/pubs/cfg/cfg.htm Fight BAC: Keep Food Safe From Bacteria http://www.fightbac.org Food Safety Resources for Kids, Teens and Educators http://www.foodsafety.gov/~fsg/fsgkids.html For Kids, Teens, and Educators http://www.cfsan.fda.gov/~dms/educate.html Hand Hygiene in Healthcare Settings http://www.cdc.gov/handhygiene Healthy Pets, Healthy People http://www.cdc.gov/healthypets Healthy Schools, Healthy People — It's a SNAP http://www.ItsASnap.org Listeriosis and Pregnancy: What is Your Risk? http://www.fsis.usda.gov/OA/pubs/lm_tearsheet.htm National Food Safety Education Month http://www.nraef.org/nfsem ThermyTM Campaign http://www.fsis.usda.gov/thermy

Thinking Globally, Working Locally: A Conference on Food Safety Education

http://www.fsis.usda.gov/Orlando2002

To Your Health: Food Safety for Seniors

http://www.foodsafety.gov/%7Efsg/sr2.html

Toxoplasmosis: An important message for pregnant women http://www.cdc.gov/ncidod/dpd/parasites/toxoplasmosis/ ToxoWomen.pdf

Food Safety Education Partnerships

Clean Hands Coalition Email to: cleanhands@cdc.gov Food Safety Training and Education Alliance http://www.FSTEA.org National Coalition for Food Safe Schools http://www.FoodSafeSchools.org Partnership for Food Safety Education http://www.fightbac.org Canadian Partnership for Consumer Food Safety Education http://www.canfightbac.org

Toll-free Information Phone Numbers

USDA Meat and Poultry Hotline: 1-800-535-4555 FDA Safe Food Hotline: 1-888-SAFE FOOD (723-3366) CDC Voice Information System: 1-888-CDC-FAXX (232-3299)

Bioterrorism/Food Bioterrorism Informational Web Sites

AMA Resources on Disaster Preparedness and Emergency Response

http://www.ama-assn.org/go/disasterpreparedness ANA Bioterrorism and Disaster Response

http://www.ana.org/news/disaster

DHHS/CDC Bioterrorism Resources http://www.bt.cdc.gov

DHHS/FDA Counterterrorism Resources http://www.fda.gov/oc/opacom/hottopics/bioterrorism.html DHHS/FDA/CFSAN Food Safety and Terrorism Resources

http://www.cfsan.fda.gov/~dms/fsterr.html

USDA-FSIS Biosecurity Resources http://www.fsis.usda.gov/oa/topics/biosecurity.htm

Suggested Reading List

General Reading

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Continuing Education Activity Sponsored by CDC **Diagnosis and Management of Foodborne Illnesses** EXPIRATION — April 16, 2007

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- Read this MMWR (Vol. 53, RR-4), which contains the correct answers to 1. the questions beginning on the next page.
- Go to the MMWR Continuing Education Internet site at <http://www. 2. cdc.gov/mmwr/cme/conted.html>.
- Select which exam you want to take and select whether you want to register for CME, CEU, CNE, or CHES credit.
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- Submit your answers no later than April 16, 2007. 6.
- Immediately print your Certificate of Completion for your records. 7.

charged for participating in this continuing education activity.

By Mail or Fax

- 1. Read this MMWR (Vol. 53, RR-4), which contains the correct answers to the questions beginning on the next page.
- Complete all registration information on the response form, including your name, mailing address, phone number, and e-mail address, if available.
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- 4. Select your answers to the questions, and mark the corresponding letters on the response form. To receive continuing education credit, you must answer all of the questions. Questions with more than one correct answer will instruct you to "Indicate all that apply."
- Sign and date the response form or a photocopy of the form and send no later than April 16, 2007, to

Fax: 404-639-4198 Mail: MMWR CE Credit Office of Scientific and Health Communications Epidemiology Program Office, MS C-08 Centers for Disease Control and Prevention 1600 Clifton Rd, N.E. Atlanta, GA 30333

6. Your Certificate of Completion will be mailed to you within 30 days.

ACCREDITATION

Continuing Medical Education (CME). This activity has been planned and implemented in accordance with the Essential Areas and Policies of the Accreditation Council for Continuing Medical Education through joint sponsorship of CDC; the Food Safety and Inspection Service, U.S. Department of Agriculture; and the Center for Food Safety and Applied Nutrition, Food and Drug Administration. CDC is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians. CDC designates this educational activity for a maximum of 2.75 hours in category 1 credit toward the AMA Physician's Recognition Award. Each physician should claim only those hours of credit that he/she actually spent in the educational activity.

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MMWR

Goal and Objectives

This *MMWR* provides recommendations for physicians and other health-care professionals who have a critical role in diagnosing, treating, and reporting food-related disease outbreaks. These recommendations were developed by the American Medical Association, the American Nurses Association-American Nurse Foundation, the Centers for Disease Control and Prevention, the Food and Drug Administration's Center for Food Safety and Nutrition, and the United States Department of Agriculture's Food Safety and Inspection Service. The goal of this report is to provide health-care providers with guidance and patient-education materials regarding foodborne illness. After completing this continuing education activity, the reader should be able to 1) differentiate between the six etiologic agents that should be considered regarding manifestations of foodborne illness; 2) describe four criteria to consider when treating a diagnosed foodborne illness; 3) summarize the reporting requirements for foodborne illness; and 4) identify three groups of persons who are at higher risk for foodborne illnesses.

To receive continuing education credit, please answer all of the following questions:

- 1. Which of the following provide important clues to the possible etiology of a food-associated illness?
 - A. Incubation period.
 - B. Duration of illness.
 - C. Predominant clinical signs and symptoms (e.g., vomiting, diarrhea, and abdominal pain).
 - D. Travel history.
 - E. All of the above.

2. Which group is at higher risk for complications from foodborne illness?

- A. Persons with weakened immune systems.
- B. Persons with liver disease.
- C. Pregnant women.
- D. Older adults.
- E. All of the above.

3. Which of the following is not a safe food-handling behavior?

- A. Using the same cutting board for raw foods and cooked foods.
- B. Using a food thermometer to check the internal temperature of food before eating it.
- C. Rinsing raw produce with water.
- D. Washing hands before and after handling food.
- 4. What is the appropriate method to use in determining if a hamburger is cooked to a proper temperature?
 - A. Cooking it until it is brown inside.
 - B. Using a food thermometer to ensure that the internal temperature reaches 160°F.
 - C. Determining if a hamburger is cooked to a proper temperature is not necessary because it is too small.
 - D. Taking a bite of the hamburger to ensure that it tastes cooked.

5. When a foodborne outbreak is suspected, who would be a helpful contact at the health department?

- A. Medical officer.
- B. Epidemiology officer.
- C. Environmental health officer.
- D. Any of the above would be helpful.

6. Which of the following is not consistent with inflammatory diarrhea?

- A. Presence of fecal leukocytes.
- B. Grossly bloody stool.
- C. Infection with invasive or cytotoxigenic bacterial and protozoan species.
- D. Involvement of the small intestine.
- 7. If a foodborne illness is suspected, which of the following should be considered?
 - A. Submission of appropriate specimens for laboratory testing.
 - B. Contacting the state or local health department.
 - C. Initiating oral rehydration therapy.
 - D. All of the above.

- 8. Intentional contamination of food is uncommon, but which of the following would make you suspect that such an act had occurred (i.e., the unusual nature of the situation would induce suspicion of intentional contamination)?
 - A. An unusual agent or pathogen in a common food.
 - A common agent or pathogen affecting an unusually large number of persons.
 - C. A common agent or pathogen that is uncommonly seen in clinical practice.
 - D. All of the above.

9. Multidrug-resistant Salmonella typhimurium cases . . .

- A. have been on the rise in the United States since the 1990s.
- B. might be responsible for more invasive disease than other types.
- C. often are resistant to ampicillin and sulfamethoxazole.
- D. cause more cases in an outbreak than do sensitive strains.
- E. all of the above.

10. Norovirus infection, which often results in nausea, vomiting, and watery/large-volume diarrhea within 24-48 hours, can be caused by ...

- A. inadequately cooked shellfish.
- B. inadequately cooked hamburger.
- C. ready-to-eat foods (e.g. salads).
- D. iced drinks.
- E. A, C, and D are correct.

11. Indicate your work setting.

- A. State/local health department.
- B. Other public health setting.
- C. Hospital clinic/private practice.
- D. Managed care organization.
- E. Academic institution.
- F. Other.

12. Which best describes your professional activities?

- A. Physician.
- B. Nurse.
- C. Health educator.
- D. Office staff.
- E. Other.

13. I plan to use these recommendations as the basis for . . . (*Indicate all that apply.*)

- A. health education materials.
- B. insurance reimbursement policies.
- C. local practice guidelines.
- D. public policy.
- E. other.

- 14. Each month, approximately how many patients with a foodborne illness do you treat?
 - A. None.
 - B. 1–5.
 - C. 6–20.
 - D. 21-50.
 - E. 51-100.
 - F. >100.
- 15. How much time did you spend reading this report and completing the exam?
 - A. <2.0 hours.
 - B. >2.0 hours but <3.0 hours.
 - C. >3.0 hours but <4.0.
 - D. >4.0 hours.
- 16. After reading this report, I am confident I can differentiate between the six etiologic agents that should be considered regarding manifestations of foodborne illness.
 - A. Strongly agree.
 - B. Agree.
 - C. Neither agree nor disagree.
 - D. Disagree.
 - E. Strongly disagree.

17. After reading this report, I am confident I can describe four criteria to consider when treating a diagnosed foodborne illness.

- A. Strongly agree.
- B. Agree.
- C. Neither agree nor disagree.
- D. Disagree.
- E. Strongly disagree.

- 18. After reading this report, I am confident I can summarize the reporting requirements for foodborne illness.
 - A. Strongly agree.
 - B. Agree.
 - C. Neither agree nor disagree.
 - D. Disagree.
 - E. Strongly disagree.

19. After reading this report, I am confident I can identify three groups of persons who are at higher risk for foodborne illnesses.

- A. Strongly agree.
- B. Agree.
- C. Neither agree nor disagree.
- D. Disagree.
- E. Strongly disagree.

20. The objectives are relevant to the goal of this report.

- A. Strongly agree.
- B. Agree.
- C. Neither agree nor disagree.
- D. Disagree.
- E. Strongly disagree.

21. The teaching strategies used in this report (text, figures, and tables) were useful.

- A. Strongly agree.
- B. Agree.
- C. Neither agree nor disagree.
- D. Disagree.
- E. Strongly disagree.

(Continued on pg CE-4)

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22. Overall, the presentation of the report enhanced my ability to understand the material.

- A. Strongly agree.
- B. Agree.
- C. Neither agree nor disagree.
- D. Disagree.
- E. Strongly disagree.

23. These recommendations will affect my practice.

- A. Strongly agree.
- B. Agree.
- C. Neither agree nor disagree.
- D. Disagree.
- E. Strongly disagree.

24. The content of this activity was appropriate for my educational needs.

- A. Strongly agree.
- B. Agree.
- C. Neither agree nor disagree.
- D. Disagree.
- E. Strongly disagree.

25. The availability of continuing education credit influenced my decision to read this report.

- A. Strongly agree.
- B. Agree.
- C. Neither agree nor disagree.
- D. Disagree.
- E. Strongly disagree.

26. How did you learn about this continuing education activity?

- A. Internet.
- B. Advertisement (e.g., fact sheet, MMWR cover, newsletter, or journal).
- C. Coworker/supervisor.
- D. Conference presentation.
- E. MMWR subscription.
- F. Other.

Correct answers for questions 1–10. 1. E; 2. E; 3. A; 4. B; 5. D; 6. D; 7. D; 8. D; 9. E; 10. E. The Morbidity and Mortality Weekly Report (MMWR) Series is prepared by the Centers for Disease Control and Prevention (CDC) and is available free of charge in electronic format and on a paid subscription basis for paper copy. To receive an electronic copy each week, send an e-mail message to *listserv@listserv.cdc.gov*. The body content should read *SUBscribe mmwr-toc*. Electronic copy also is available from CDC's World-Wide Web server at *http://www.cdc.gov/mmwr* or from CDC's file transfer protocol server at *ftp://ftp.cdc.gov/pub/publications/mmwr*. To subscribe for paper copy, contact Superintendent of Documents, U.S. Government Printing Office, Washington, DC 20402; telephone 202-512-1800.

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The Evolving Challenge of Foodborne Illness Outbreak Response

CHAPTER SUMMARY POINTS

- Foodborne illness strikes tens of millions, hospitalizes more than 100,000, and kills an estimated 3,000 people in the United States each year.
- The U.S. diet has changed in response to numerous factors creating new foodsafety challenges.
- Important advances in clinical laboratory techniques and public health approaches to detect and investigate clusters of illness are being used to better define the scope and nature of foodborne illness.
- Information systems and food-supply investigation techniques are developing to enhance our ability to trace contaminated foods, identify and control contamination sources, and remove contaminated food from circulation.
- Industry-driven and regulatory food-safety standards are being changed to better address risks identified by foodborne illness outbreak investigations to prevent similar outbreaks.

URLs in this chapter are valid as of July 11, 2019.

1.0 Introduction

Outbreaks of foodborne illness and their detection, investigation, and control are functions of several constantly changing factors. The U.S. diet has changed in response to public health recommendations; economics of food production and distribution; and the growing demands for convenience in food service, as well as diversity and freshness of foods in the marketplace. Important advances have been made in clinical laboratory techniques to diagnose foodborne illnesses and in public health approaches to detect and investigate clusters of illness. Information systems are developing to enhance our ability to trace contaminated food and eliminate it from circulation and to glean lessons learned from these investigations to prevent similar outbreaks. In addition, industry-driven and regulatory food-safety standards are being changed to better address risks identified by foodborne illness outbreak investigations to prevent similar outbreaks.

This chapter provides an overview of these ever-changing factors. Subsequent chapters detail specific approaches used by investigators.

1.1 The Burden of Foodborne Illness in the United States

1.1.1 In 2011, the Centers for Disease Control and Prevention (CDC) estimated that each year in the United States 47.8 million illnesses, resulting in 128,000 hospitalizations and 3,000 deaths, were attributable to contaminated food (1, 2). Of these illnesses, 9.4 million are caused by 31 known agents of foodborne illness, and the remaining 38.4 million by unspecified agents. Tracking overall changes in the burden of foodborne illness from year to year is not currently possible, but trends are evident in known foodborne illnesses tracked by FoodNet (https://wwwn.cdc.gov/ foodnetfast). Most notably, the incidence of Escherichia coli O157:H7 infections dropped from approximately 2.5 cases per 100,000 population during the mid-1990s to fewer than 1 case per 100,000 by the mid-2000s, accomplishing a goal of Healthy People 2010. Following early declines in the incidence of Listeria and Campylobacter infections, rates remained stable throughout the 2000s, whereas the incidence of Vibrio infections increased. Overall rates of Salmonella infections remained stable; the incidence of infection by serotypes Typhimurium and Heidelberg decreased; and infection by serotypes Enteritidis, Javiana, and

the monophasic variant of Typhimurium, serotype I 4,[5],12:i:-, increased (3).

Because not all illnesses caused by foodborne pathogens are individually reportable, recognition of other pathogen-specific trends relies on surveillance of foodborne illness outbreaks. CDC's National Outbreaks Reporting System (NORS) logged 20,854 outbreaks comprising 403,110 illnesses, 16,517 hospitalizations, and 392 deaths during 1998–2017 (https://wwwn.cdc.gov/ norsdashboard/). Reporting of foodborne illness outbreaks caused by norovirus increased during 1998-2004, but since 2010, annual totals have varied little, hovering around 300 per year. A comparison of etiologies causing single-agent outbreaks during 2012-2017 with those during 2002-2011 showed that outbreaks caused by agents associated with poor foodholding practices in commercial food-service establishments decreased: Bacillus cereus, down from an average of 17 outbreaks per year to 10 per year; Clostridium perfringens, from 40 to 32 per year; scombroid or histamine, from 23 to 17 per year; and Staphylococcus aureus, from 27 to 12 per year. These changes most likely represent actual reductions in outbreak

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1.1 The Burden of Foodborne Illness in the United States

occurrence because the percentage of reported outbreaks for which no etiologic agent was identified dropped from 59% in 1998 to 23% in 2017 (4).

1.1.2 In 2014, the U.S. Department of Agriculture's Economic Research Service (USDA-ERS) estimated the average annual economic burden of foodborne illness at \$15.5 billion (5). USDA-ERS based this burden on cost estimates of foodborne illness caused by 15 major pathogens in the United States (Table 1.1). These 15 pathogens account for 95% of illnesses and deaths from foodborne illness acquired in the United States for which a pathogen was identified. These estimates include costs associated with medical treatment of acute and chronic illness, lost wages of persons who recovered, and costs associated with premature deaths.

Table 1.1. Estimated Annual Cost of Foodborne Illness, Estimated Total Foodborne Cases, and Average Cost per Case Identified, United States, 2013

PATHOGEN	TOTAL COST	ESTIMATED TOTAL FOODBORNE CASES	COST PER CASE
Vibrio vulnificus	\$319,900,000	96	\$3,332,000
Listeria monocytogenes	\$2,834,400,000	1,591	\$1,782,000
Toxoplasma gondii	\$3,304,000,000	86,686	\$38,100
Vibrio spp. (other noncholera)	\$72,800,000	17,564	\$8,100
Shiga toxin-producing Escherichia coli O157	\$271,400,000	63,153	\$4,300
Salmonella spp. (nontyphoidal)	\$3,666,600,000	1,027,561	\$3,600
Yersinia enterocolitica	\$278,000,000	97,656	\$2,900
Campylobacter spp.	\$1,928,800,000	845,024	\$2,300
Vibrio parahaemolyticus	\$40,700,000	34,664	\$1,200
Shigella (all species)	\$138,000,000	131,254	\$1,100
Cryptosporidium parvum	\$51,800,000	57,616	\$900
Norovirus	\$2,255,800,000	5,461,731	\$410
Clostridium perfringens	\$342,700,000	965,958	\$360
Non-O157 Shiga toxin-producing E. coli	\$27,400,000	112,752	\$240
Cyclospora cayetanensis	\$2,300,000	11,407	\$200

Source: U.S. Department of Agriculture. Cost estimates of foodborne illnesses. <u>https://www.ers.usda.gov/data-products/cost-estimates-of-foodborne-illnesses</u>

1.1 The Burden of Foodborne Illness in the United States

1.1.3 The impact of foodborne illness on the food industry varies greatly, and the costs seldom are limited to one company. This impact is evident when the distribution network of the food supply is considered. The impacts of recalls on the food industry are farreaching, in some cases topping \$10 million in direct costs.

Direct costs of recalls include notification of regulators, supply chain, and consumers; product retrieval, storage, and destruction; unsalable product; and the additional labor associated with these activities. These direct costs do not include litigation, increased regulatory compliance, and the impact to the company's market value and brand reputation.

The outbreak of *E. coli* O157:H7 infection associated with romaine lettuce grown in the Yuma, Arizona, growing region in April 2018 provides a good example of the indirect costs to the industry associated with lost sales and brand damage (6). This outbreak sickened 210 people in 36 states. During the week that followed the initial news of the outbreak, sales of romaine lettuce fell 20% (7). In addition, data from Nielsen also showed marked drops in sales of iceberg lettuce, red leaf lettuce, and endive. The impact of a second, although unrelated, outbreak of *E. coli* O157:H7 associated with romaine lettuce in November 2018 (8) was even more dramatic because CDC advised consumers to avoid eating romaine lettuce from any source in an effort to remove potentially contaminated romaine from commercial distribution channels.

With a more comprehensive accounting of potential costs, researchers at the Johns Hopkins Bloomberg School of Public Health suggested that the cost to a restaurant for a single foodborne illness outbreak can range from \$4,000 to \$2.6 million, depending on the pathogen, type of restaurant involved, and size of the outbreak. For example, a foodborne illness outbreak in which five people became sick in a fast food restaurant would result in costs of approximately \$4,000 if there was no loss in revenue and no lawsuits, legal fees, or fines. In contrast, a single outbreak of listeriosis involving 250 persons in a fine dining restaurant could cost upwards of \$2.6 million in lost sales, lawsuits, legal fees, fines, and higher insurance premiums (9).

1.2 Growing Complexity of the Food Supply

U.S. food-consumption patterns change continuously. Changes in diets and food preferences have resulted in a greater demand for a broader variety of fruits, vegetables, and other foods. Moreover, Americans expect to consume these foods year-round, driving importation from areas of the world with the growing seasons necessary to meet U.S. demand. Meeting global supply-chain demands also has increased the complexity and logistics of how food is transported from farm to fork. **1.2.1 A major indicator of changing diets is the consumption of fresh fruits and vegetables.** From 1996 to 2017, loss-adjusted per capita availability of fresh fruit increased 7% from 55 to 59 pounds (10). Consumption of fresh vegetables increased only marginally from 68 to 70 pounds per person. During the same time, per capita consumption of chicken increased 30% from 40 to 52 pounds, whereas that of beef declined 17% from 49 to 41 pounds (10). Within the arena of fresh produce, consumption of head lettuce declined 34% from 12 to 8 pounds per capita, whereas consumption of romaine and leaf

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1.2 Growing Complexity of the Food Supply

lettuce doubled from 3 to 6 pounds per capita, and consumption of fresh spinach nearly tripled from 0.3 to 0.9 pounds per capita. Consumption of fresh berries also increased substantially. The general pattern of these dietary changes reflects public health recommendations toward healthier eating (10).

The food industry has met this demand through routine importation of items once considered out of season or exotic. According to reports by USDA-ERS (11), the proportion of imported fresh fruits increased from 39% in 1996 to 53% in 2016. Excluding bananas, for which there is no domestic production, the share of imported fruits increased from 16% to 38%. Similarly, the percentage of imported fresh vegetables increased from 14% to 31%. Although a high proportion of some fresh produce items, such as mango and papaya, always have been imported, an increasingly more conventional produce items are also imported. For example, the percentage of imported avocadoes increased from approximately 14% in 1996 to 89% in 2016, and that of blueberries increased from 24% to 57% during that same period (11).

The safety of imported food products depends largely on the public health and food-safety systems of other countries. Recent analyses of foodborne illness outbreaks reported to CDC support the existence of food-safety problems in other countries. During 1996–2014, the number of confirmed foodborne illness outbreaks associated with imported foods increased from 3 per year to 18 per year. *Salmonella* and *Cyclospora* accounted for about one third of the outbreaks and 75% of cases, most due to contaminated produce from Latin America (11).

1.2.2 Culinary preferences for undercooked or raw foods also contribute to more frequent infections and outbreaks caused by the microorganisms associated with these foods. These include classical outbreaks of Shiga toxin-producing *E. coli* (STEC), *Salmonella*, *Campylobacter*, and *Listeria* infections associated with raw milk and raw milk cheeses; *Salmonella* associated with raw tuna in sushi; and *Campylobacter* and *Salmonella* in minimally processed liver pates. A corresponding trend for raw pet foods made from meat and poultry products also has led to outbreaks among people from handling the raw pet food, exposure to ill animals, or environmental contamination in the household.

Foodborne illnesses also can be associated with ingestion of products not typically thought of as food. During 2017-2018, kratom, a tree leaf with stimulant and opioid properties, caused illness by a variety of Salmonella serotypes. Smoking marijuana caused an outbreak of salmonellosis in 1981 (12); and a cannabisassociated toxidrome among four persons who attended the August 2014 Denver County Fair was associated with consumption of chocolate bars obtained at the "LoveAll" booth at the fair's "Pot Pavilion" (13). The full legalization of cannabis products in at least nine other states and the District of Columbia since 2014 and associated sales of cannabisinfused edibles could lead to more foodborne illness outbreaks. However, no outbreaks from cannabis products were reported to NORS from 2015 to 2018.

1.2.3 Changes in how food is cultivated or raised, processed, and distributed and where, how, and by whom food is prepared also contribute to changing patterns of foodborne illness. The demand for processed and ready-to-eat foods has led to the industrialization of food production with increasingly intense agricultural practices and broadening distribution of food products. Changes in agricultural, processing, or packaging methods might facilitate bacterial contamination or growth. Large multistate STEC outbreaks associated with leafy green vegetables reflect the challenges of intensive

1.2 Growing Complexity of the Food Supply

animal and fresh produce production in a shared environment. The scale of these operations magnifies the impact of foodsafety system failures, resulting in thousands of exposures and potential illnesses across multiple states, and even multiple countries.

Increasingly complex food-distribution systems span the globe. Products move from farm to fork through a network of farms, processors, manufacturers, packers, importers, brokers, storage facilities, distribution centers, and retail outlets. In some instances, food from a farm can change hands more than 10 times before it reaches a consumer. These complex supply chains are maintained by a wide variety of record-keeping systems; outbreak investigators charged with tracing foods back through the supply chain are left to decode these systems and piece together, step by step, how a food reached its final destination.

At the same time, a counter-trend promoting local food sources and small-scale farm-to table distribution networks (sometimes termed the "locavore movement" or "communitysupported agriculture") has emerged. The number of small food producers and directto-consumer marketing avenues (e.g., farmer's markets, farm stands, farm-to-school programs, and "pick-your-own" operations) also has risen. According to national agriculture census data, from 1997 to 2017, direct sales of agricultural products to the public increased by 374%, compared with an increase of 93% for all agricultural sales. During the same period, the number of farms selling directly to consumers increased by 18%, compared with an 8% decrease in the total number of farms (14). In addition, most states have "cottage food" laws, allowing small producers to cook, can, or pickle outside of licensed kitchens certain foods that are typically considered low-risk.

The effect of increased consumption of locally produced foods is yet to be determined,

but the consequences of eating unsafe food apply to both small and large producers. For an individual, it is equally as bad to get STEC infection from farm-fresh strawberries harvested from a local field frequented by wild deer as it is to get STEC infection from romaine lettuce shipped hundreds of miles after contamination with runoff from a cattle feed lot. Although a small producer's limited distribution system might affect fewer people, implementing improved food-safety measures might be more challenging for the small producer. In addition, farm direct sales (i.e., farmers selling produce, eggs, and other foods they produced directly to retail customers, such as through farmers' markets and farm stands) are not included among food facilities in the 2011 Food Modernization and Safety Act (FMSA) (15). In some states and local jurisdictions, these sales have been exempted from food-safety regulations that pertain to other food facilities.

By whom and where our food is prepared also plays a role in foodborne illness occurrence and outbreaks. Americans increasingly eat away from home, spending more than 50% of food dollars away from home, since 2010 (16). During this period, there was considerable growth in limited service "fast casual" restaurants that featured more complex food handling than traditional fast-food restaurants. The increased number of meals eaten away from home most likely influenced the increase in foodborne illness. In an analysis of foodborne illness outbreaks reported to CDC during 2009-2017, 62% were associated with restaurants (4, 17). In addition, studies of sporadic and outbreak-associated foodborne illness, including infection with STEC O157, Salmonella enterica serotypes Enteritidis and Typhimurium, and Campylobacter jejuni suggest that commercial food-service establishments, such as restaurants, play an important role in foodborne illness in the United States (18).

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1.2 Growing Complexity of the Food Supply

Finally, the growing e-commerce in delivery of groceries and restaurant food directly to consumers' homes provides foodborne illness investigators with opportunities for verifying food purchases and dates. Whether an increased risk for illness accompanies these means of food distribution remains to be determined.

1.3 Enhanced U.S. Foodborne Illness Surveillance Systems

A variety of surveillance systems have been developed to identify foodborne illness and detect outbreaks. Some systems focus on specific pathogens likely to be transmitted through food and have been used extensively for decades. More recently, new surveillance methods have emerged that provide data on food vehicles, settings, pathogens, contributing factors, and environmental antecedents. Effective surveillance to track cases of foodborne illness and outbreaks is critical to developing effective control strategies.

1.3.1 Changes in surveillance for human illness have affected how outbreaks are detected (Chapter 4) and investigated (Chapter 5). All states and territories have legal requirements for the reporting of certain illnesses and conditions, including illnesses likely to be foodborne (e.g., salmonellosis, campylobacteriosis, and STEC infection), by healthcare providers and laboratories to the local, state, or territorial public health agency (Chapter 2). Local and state agencies also receive and respond to complaints of illness directly from the public. The adoption of new testing methods in clinical and public health laboratories, as well as improved information management systems and social media, are transforming surveillance activities.

 Molecular subtyping by public health laboratories has been the basis for national pathogen-specific surveillance since the initiation of PulseNet in 1996. The use of pulsed-field gel electrophoresis (PFGE) increased the ability to link isolates from distant locations and thereby to infer epidemiologic relatedness; PFGE revolutionized the detection and investigation of foodborne illness outbreaks and led to prevention of illnesses. However, PFGE provided limited information about the organism itself. Rapid bacterial sequencing technology and the informatics tools needed to accommodate whole-genome sequencing (WGS) have been developed and in 2019 rapidly deployed to public health laboratories across the United States. On July 15, 2019, WGS replaced PFGE as the primary molecular subtyping tool for pathogen-specific surveillance.

· Concurrent with the development of WGS to improve molecular subtyping, clinical laboratories have moved away from traditional fecal culture in favor of cultureindependent diagnostic tests (CIDTs). These methods can rapidly identify pathogens and expedite treatment decisions, but they do not yield the bacterial isolates required by public health officials. Many public health jurisdictions require submission of CIDTpositive specimens for subsequent culture and subtyping—but this shifts the burden of isolation from the clinical laboratory to the public health laboratory and delays cluster recognition. Conversely, CIDTs may be more sensitive and offer the prospect of detecting pathogens (e.g., enterotoxigenic *E. coli*) that may elude detection by culture. FoodNet, the 10-site active surveillance program for infections often transmitted through foods, has increased collection of data on use of CIDTs and on the frequency and results of reflex cultures.

1.3 Enhanced U.S. Foodborne Illness Surveillance Systems

- Newer technologies are likely to lead to recognition of more clusters and reduced cluster sizes than with PFGE. They also take longer, delaying cluster recognition by this means.
- Improved epidemiologic investigation practices have been developed. These include the standardization of common data elements for interviewing case-patients, use of standardized hypothesis-generating questionnaires, increased use of consumer product purchase (e.g., "shopper card") data, aggregation of case-patient exposures and comparison with population reference standards, and improved subcluster investigation and informational traceback methods to improve the specificity of exposure assessments.
- The principles of foodborne illness complaint surveillance are being standardized (Chapter 4). The value of using electronic databases to review and analyze complaints and to link complaints with pathogen-specific surveillance systems has been demonstrated. Numerous social media platforms have been evaluated to assess their potential utility to enhance conventional complaint surveillance. To the extent these can facilitate linking illnesses with exposure, rather than just reinforcing the "last meal eaten" bias, they may warrant attention from public health agencies.
- Standards and procedures for outbreak reporting have been developed for NORS. NORS supports outbreak reporting from state, local, and territorial health departments in the United States. NORS Dashboard is a public-facing, web-based tool containing limited and cleaned NORS data that can be filtered using an interactive interface that produces summary data, statistics, and a variety of graphs based on user preferences (<u>https://wwwn.cdc.</u> gov/norsdashboard). CDC, USDA's Food

Safety and Inspection Service (FSIS), Food and Drug Administration (FDA), and other investigating agencies analyze these data to improve understanding of the impact of foodborne illness outbreaks on human health and of the pathogens, foods, and settings involved in these outbreaks.

 Specialized surveillance networks have been developed for specific pathogens. For example, CaliciNet is a norovirus outbreak surveillance network of local, state, and federal public health laboratories. Network partners perform viral sequencing and upload sequences into CaliciNet to monitor circulating strains, and identify newly emerging norovirus strains. CaliciNet outbreak lab data are linked to matching outbreak data in NORS. CryptoNet, the first U.S. national molecular tracking system for a parasitic infection, was formally launched in 2015 to collect specimens and to characterize the molecular epidemiology of infection by Cryptosporidium spp., only some of which are pathogenic for humans but which are typically indistinguishable morphologically.

1.3.2 Surveillance for food-preparation hazards and environmental assessments of outbreaks have been developed to identify root causes (Chapter 5) and improve preventive controls (Chapter 6). Routine food-safety inspections are conducted for all licensed food-service establishments by approximately 3,000 local and 75 state and territorial agencies. Although traditionally conducted to ensure that food-service establishments were operating within the provisions of state food codes, many of which are adopted from the FDA Model Food Code (19), inspection results are being increasingly displayed at the point of service or online to provide information to consumers about potential food-safety risks. A growing body of evidence suggests that such public disclosure of inspection results might improve restaurant

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1.3 Enhanced U.S. Foodborne Illness Surveillance Systems

inspection results and reduce the risk for illness transmission to patrons.

- To standardize assessment of retail food risk factors, FDA initiated the Retail Food Risk Factor Study to measure practices and behaviors commonly identified as contributing factors in foodborne illness outbreaks (20). Data from the initial study, collected during 1998, 2003, and 2008, documented progress toward the goal of reducing contributing factors (https://www. cdc.gov/nceh/ehs/nears/cf-definitions.htm) at retail establishments: five of the nine facility types showed a statistically significant improvement in compliance for all 42 contributing factors during the study period. A second round of the Retail Food Study was initiated in 2013 to assess food-protection manager certification and food-safety management systems. One important finding from the study was that fewer food-safety items were out of compliance in restaurants having well-developed and documented food-safety management systems (20).
- The Environmental Health Specialists Network (EHS-Net) of environmental health specialists and epidemiologists from local and state health departments, FDA, FSIS, USDA's Food and Nutrition Service, and CDC developed the National Environmental Assessment Reporting System (NEARS) to systematically monitor and evaluate root causes of foodborne illness outbreaks, including contributing risk factors and environmental antecedents. This system is cross-referenced with NORS and collects information from detailed environmental assessments on factors contributing to the outbreak and the underlying conditions that led to it. The information collected through NEARS can inform hypothesis generation about antecedents to foodborne illness outbreaks and strengthen the ability of foodcontrol authorities to formulate and evaluate the effectiveness of food-safety actions.

1.3.3 The food supply and associated environments are tested by local, state, and federal regulatory officials and the food industry. Food testing is a tool used to assess whether an establishment's foodsafety system is functioning adequately to address hazards in food production and manufacturing and prevent foodborne illnesses. Food and environmental testing data, including molecular subtyping data, can be used to inform hypothesis generation during outbreaks. Food testing data also can be used to estimate the fraction of selected foodborne illnesses caused by specific food sources, to assess changes in food contamination over time, and to assess the success of regulatory measures. Foodborne pathogens of interest that are isolated from food or from animal or environmental sources during various government testing programs are being characterized by WGS and the sequence data added to FDA's GenomeTrakr BioProjects housed at NIH NCBI, where they can be compared with data from human isolates directly on NCBI Pathogen Browser and/or in the CDC-PulseNet National Database. No formal framework exists to link industrywide testing to public health surveillance data. Mechanisms have been discussed that would provide access to aggregated, or blinded industry data to avoid regulatory penalties to individual companies.

To ensure technical competence and the ability to generate reliable data, food testing laboratories within FDA and FSIS maintain accreditation in the International Organization for Standardization/International Electrotechnical Commission 17025 standard—the main international standard used by testing and calibration laboratories. Additionally, FDA is leading an effort to bring state human and animal food testing laboratories into International Organization for Standardization/International Electrotechnical Commission 17025 accreditation to enhance efforts to protect the food supply. Data **H**

1.3 Enhanced U.S. Foodborne Illness Surveillance Systems

generated by accredited laboratories will be made available for consideration during FDA enforcement actions, as well as for surveillance purposes and during local, state, or federal response to foodborne illness outbreaks. Laboratory accreditation also will assist state manufactured food-regulatory programs in achieving conformance with the Manufactured Food Regulatory Program Standards.

1.4 Foodborne Illness Outbreak Response and System Change

1.4.1 Although foodborne illness surveillance and response are rooted in individual states' laws, the growing trend in multistate outbreaks associated with widely distributed foods requires increasing standardization of methods, integration of activities, and federal support and oversight. In response to the emergence of E. coli O157:H7 and other foodborne pathogens during the 1990s, CDC developed the active surveillance network FoodNet, with funding assistance from FSIS and FDA, to conduct comprehensive surveillance of diagnosed illnesses within defined populations to assess and monitor trends in the burden of illness associated with specific agents. Simultaneously, CDC established the national molecular subtyping network PulseNet to improve laboratory-based surveillance for bacterial pathogens routinely detected by clinical laboratories. PulseNet increased detection of multistate outbreaks, and FoodNet provided a framework to interpret the impact of food system changes in response to improved outbreak detection and regulatory activity.

In 2005, CIFOR was established to identify barriers to effective surveillance and investigation of foodborne illnesses and outbreaks. One of the first CIFOR projects was to develop guidelines for outbreak detection and response. The First Edition of the CIFOR Guidelines, published in 2009, established model practices for foodborne disease surveillance at local and state levels, with specific reference to coordination of multijurisdictional outbreaks investigations and development of performance indicators to measure the effectiveness of surveillance activities. The Second Edition of the Guidelines was published in 2014.

During this time, CDC began providing dedicated funding to support state-level foodborne illness outbreak response through Epidemiology and Laboratory Capacity cooperative agreements. This led to development of several CDC programs: OutbreakNet, CDC's Foodborne Diseases Centers for Outbreak Response Enhancement (FoodCORE), and the Integrated Food Safety Centers of Excellence and OutbreakNet Enhanced (OBNE). The CDC Integrated Food Safety Centers of Excellence were created by FSMA. These programs are intended to work together to enhance the development and evaluation of foodborne illness surveillance and outbreak response activities across the United States.

In conjunction with CDC's investments in the performance of public health agencies, FDA has used additional resources provided by FSMA to develop a network of Rapid Response Teams (RRT) to enhance coordination between public health and food-regulatory agencies at the state level and formed a Coordinated Outbreak Response and Evaluation (CORE) Network to centralize coordination of outbreak response activities within FDA. FSIS has developed parallel outbreak response capacity (Chapter 3).

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1.4 Foodborne Illness Outbreak Response and System Change

With a stated goal of building an Integrated Food Safety System, FDA established the Partnership for Food Protection in 2008, bringing together local, state, territorial, tribal, and federal representatives with expertise in food; feed; epidemiology; laboratory; and animal, environmental, and public health. The Partnership for Food Protection (PFP) brings the collective expertise of the above stakeholders to work on projects that enhance human and animal food safety in the United States.

Coordination of activities on the federal level is accomplished through mutual liaisons between agencies, and joint participation in the Intergovernment Food Safety Analytics Collaboration (IFSAC) which seeks to improve the use of outbreak surveillance in foodborne illness attribution models and thus better guide food-safety regulation. Chapter 3 details the agencies currently involved in foodborne illness outbreak response, along with their respective roles and responsibilities. Issues posed in the response to multijurisdictional outbreaks are discussed in Chapter 7.

1.4.2 Food-safety standards are changing to better control food-safety risks identified by foodborne illness outbreak investigations. Both industry-driven standards (e.g., from the Global Food Safety Initiative, https://www.mygfsi.com/about-us/ about-gfsi/what-is-gfsi.html) and governmentdriven regulatory requirements are being updated to identify and manage food-safety hazards more rapidly. Examples of noteworthy regulatory changes in the United States include

• The 2011 FSMA—the first major reform of the FDA's food-safety authority since the 1938 enactment of the Food, Drug, and Cosmetic Act. Since the Second Edition of the CIFOR Guidelines, some key provisions of FSMA have been rolled out in seven federal regulations (Chapter 2), which provide FDA with additional legal authorities and resources to strengthen food-safety systems. They enable FDA and its foodsafety partners, to focus on preventing foodsafety problems and to address food-safety risks more rapidly when they are identified. FSMA and its associated regulations grant FDA substantial new authority to protect food all along the farm-to-fork line, covering preventive controls, inspections, laboratory testing, product tracing, mandatory recall authority, importer accountability, authority to deny entry to the U.S. market, state and local capacity building, and other areas.

• Since enactment of its Pathogen Reduction, Hazard Analysis and Critical Control Point Systems rule to reduce risks associated with meat and poultry in 1996, FSIS has continued to address food-safety hazards. In 2011, FSIS established raw poultry performance standards for Campylobacter and updated existing ones for Salmonella. In 2012, FSIS added six non-O157 STEC serogroups as "adulterants" in raw beef. In 2015, after agency investigators noted they often were impeded in efforts to trace ground beef to its source during outbreak investigations and in response to STEC-positive sample results, FSIS required its regulated establishments and retail stores to maintain detailed records to identify all ground-beef source materials.

In summary, the foods we eat and the processes by which they are produced, distributed, and prepared; the means for diagnosing illness and detecting outbreaks; the methods whereby outbreaks are investigated; and the response of government and private partners are always changing. The following chapters provide updated guidance to responders with these changes in mind. The final chapter (Chapter 8) provides and references metrics for evaluating an agency's progress toward optimizing its response to foodborne illness outbreaks.

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Outbreaks of Acute Gastroenteritis Transmitted by Person-to-Person Contact — **United States, 2009–2010**





U.S. Department of Health and Human Services Centers for Disease Control and Prevention

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Front cover photo: Two images, which include 1) norovirus and 2) hand washing.

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Outbreaks of Acute Gastroenteritis Transmitted by Person-to-Person Contact — United States, 2009–2010

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Abstract

Problem/Condition: Approximately 179 million cases of acute gastroenteritis (AGE) occur in the United States each year, and outbreaks of AGE are a substantial public health problem. Although CDC has conducted national surveillance for waterborne and foodborne AGE outbreaks since 1971 and 1973, respectively, no national surveillance existed for AGE outbreaks resulting primarily from person-to-person transmission before implementation of the National Outbreak Reporting System (NORS) in 2009.

Reporting Period: 2009–2010.

Description of System: NORS is a national surveillance system launched in 2009 to support the reporting of all waterborne outbreaks and enteric disease outbreaks from foodborne, person-to-person, animal contact, environmental, and unknown modes of transmission. State and local public health agencies in the 50 U.S. states, the District of Columbia, five U.S. territories, and three Freely Associated States report these outbreaks to CDC via NORS using a standardized online data entry system. Data are collected on general outbreak characteristics (e.g., dates, number of illnesses, and locations), demographic characteristics of cases (e.g., age and sex), symptoms, case outcomes, and laboratory testing information and results. Only outbreaks reported in NORS with a primary mode of transmission of person-to-person contact are included in this report.

Results: During 2009–2010, a total of 2,259 person-to-person AGE outbreaks were reported in NORS from 42 states and the District of Columbia. These outbreaks resulted in 81,491 reported illnesses, 1,339 hospitalizations, and 136 deaths. No etiology was reported in approximately 40% (n = 840) of outbreaks. Of the remaining 1,419 outbreaks with a reported etiology, 1,270 (89%) were either suspected or confirmed to be caused solely by norovirus. Other reported etiologies included *Shigella* (n = 86), *Salmonella* (n = 16), Shiga toxin-producing *Escherichia coli* (STEC) (n = 11), and rotavirus (n = 10). Most (82%) of the 1,723 outbreaks caused by norovirus or an unknown etiology occurred during the winter months, and outbreaks caused by *Shigella* or another suspected or confirmed etiology most often occurred during the spring or summer months (62%, N = 53 and 60%, N = 38, respectively). A setting was reported for 1,187 (53%) of total outbreaks. Among these reported settings, nursing homes and other long-term–care facilities were most common (80%), followed by childcare centers (6%), hospitals (5%), and schools (5%).

Interpretation: NORS provides the first national data on AGE outbreaks spread primarily through person-to-person transmission and describes the frequency of this mode of transmission. Norovirus is the most commonly reported cause of these outbreaks and, on the basis of epidemiologic characteristics, likely accounts for a substantial portion of the reported outbreaks of unknown etiology. In the United States, sporadic and outbreak-associated norovirus causes an estimated 800 deaths and 70,000 hospitalizations annually, which could increase by an additional 50% during epidemic years. During 2009–2010, norovirus outbreaks accounted for the majority of deaths and health-care visits in person-to-person AGE outbreaks reported to NORS.

Public Health Action: Prevention and control of person-to-person AGE outbreaks depend primarily on appropriate hand hygiene and isolation of ill persons. NORS surveillance data can help identify the etiologic agents, settings, and populations most often involved in AGE outbreaks resulting primarily from person-to-person transmission and guide development of targeted interventions to avert these outbreaks or mitigate the spread of infection. Surveillance for person-to-person AGE outbreaks via NORS also might be important in clarifying the epidemiology and role of certain pathogens (e.g., STEC) that have been traditionally considered

Corresponding author: Mary Wikswo, Division of Viral Diseases, National Center for Immunization and Respiratory Diseases, CDC, 1600 Clifton Rd., NE, MS A-34, Atlanta, GA 30333. Telephone: 404-639-0881; Fax: 404-235-7861; E-mail: ezq1@cdc.gov. foodborne but can also be transmitted person-to-person. As ongoing improvements and enhancements to NORS are introduced, participation in NORS has the potential to increase, allowing for improved estimation of epidemic person-to-person AGE and its relative importance among other modes of transmission.

Introduction

Acute gastroenteritis (AGE) is a major cause of illness in the United States, with approximately 179 million episodes occurring each year (1,2). Outbreaks of AGE contribute to this substantial public health problem. Data on waterborne and foodborne AGE outbreaks have been collected nationally by various methods since the 1920s (3,4). These methods were formalized in 1971 and 1973 when the Waterborne Disease and Outbreak Surveillance System (WBDOSS) and the Foodborne Disease Outbreak Surveillance System (FDOSS), respectively, were created as national surveillance systems to provide complete and accurate data for waterborne and foodborne disease outbreaks. However, no national system existed for reporting AGE outbreaks caused by direct personto-person contact or other modes of transmission.

In 2006, the Council for State and Territorial Epidemiologists passed a resolution recommending that all outbreaks of AGE in the United States be nationally reported. The National Outbreak Reporting System (NORS) addresses this resolution by integrating WBDOSS and FDOSS with the first national reporting system for person-to-person AGE outbreaks, as well as AGE outbreaks caused by contact with animals or contaminated environments or by unknown modes of transmission.

The information collected through NORS can help guide the development of appropriate strategies to prevent and control AGE outbreaks resulting primarily from person-toperson transmission. This information is vital to improving understanding of these outbreaks, their frequency, and population-level risk factors for severe illness and death. To characterize the frequency and characteristics of person-toperson AGE outbreaks, CDC analyzed 2009–2010 data (the inaugural years that data were available) from NORS. This report summarizes those findings and is intended to be used by health departments and regulatory agencies to identify settings and populations for interventions likely to yield the greatest public health benefits.

Methods

Data Source

NORS is a voluntary national surveillance system designed to support the reporting of all waterborne outbreaks and enteric disease outbreaks from foodborne, person-to-person, animal contact, environmental, and unknown modes of transmission. State and local public health agencies in the United States and its territories report these outbreaks to CDC via NORS using a standard online data entry system. The NORS web-based data entry system was launched in February 2009 to all 59 NORS reporting sites comprised of the 50 U.S. states, the District of Columbia, five U.S. territories (American Samoa, Guam, the Commonwealth of the Northern Mariana Islands, Puerto Rico, and the U.S. Virgin Islands), and three Freely Associated States (the Federated States of Micronesia, the Republic of the Marshall Islands, and the Republic of Palau). Sites were encouraged to report outbreaks occurring since January 1, 2009, as well as those occurring prospectively.

Case Definition and Classification

All cases included in each NORS report were assumed to have met the case definition used for that outbreak investigation. For this analysis, the etiology of the outbreaks were categorized as norovirus, *Shigella*, other/multiple, and unknown on the basis of state reports. The term "no etiology reported" also is sometimes referred to as an "unknown etiology." The term "any etiology" refers to all reported outbreaks, including those with no etiology reported. NORS allows reporting sites to edit, add, or delete reports at any time. To reduce this fluidity in the data, only reports marked as "finalized" by the reporting site administrators were included in the analyses. Data also are subjected to basic logic checks at the conclusion of each calendar year to improve data quality.

An outbreak from person-to-person transmission is defined as ≥ 2 cases of a similar enteric illness associated with a common exposure in which the primary mode of transmission is reported as person-to-person contact, as determined by each reporting site. The source or index case of each outbreak, defined as the patient with the earliest illness onset, is included among the outbreak-associated cases. Case definitions or classification schemes might not be consistent across all sites. The date of earliest illness onset is defined in NORS as the date of outbreak occurrence. Data are reported on general outbreak characteristics (e.g., dates, number of illnesses, and locations), general demographic characteristics of cases (e.g., age and sex), symptoms, case outcomes, and laboratory testing information and results. A reported etiology was considered "confirmed" if ≥ 2 laboratory-confirmed cases were reported, consistent with CDC guidelines for confirmation of etiologies in foodborne disease outbreaks (5). If a reported etiology was associated with <2 laboratory-confirmed cases, it was classified as a suspected etiology. Symptoms were classified and reported in NORS according to the definitions used during each outbreak investigation.

To calculate attack rates in certain settings in which exposure occurred, states were asked to classify cases into two groups: residents and guests or staff. The former group is intended to capture the number of persons who did not work in the major setting of exposure, such as children attending childcare centers, residents of a long-term–care facility (LTCF), or guests of a hotel. The latter group is intended to capture the number of persons who work in the major setting, such as health-care providers, teachers, childcare center employees, and hotel staff.

The following outbreak reports were excluded from analysis: all outbreak reports in which the total number of cases was not entered, the total number of cases was <2, the reported etiology does not cause AGE (e.g., measles, scabies, or Hepatitis A), and outbreaks with a different primary mode of transmission (e.g., foodborne) with secondary person-to-person transmission.

Analysis

Person-to-person AGE outbreaks with a first illness onset during January 2009–December 2010, reported in NORS, marked as finalized by a state administrator, and meeting the inclusion criteria as stated in the methods are included in this summary. Data were extracted from NORS on January 24, 2012. Outbreak incidence in each reporting site was calculated using national data from the U.S. Census Bureau for 2009 for each state and expressed per 1 million population per year (6). If a state reported outbreaks in both 2009 and 2010, the average number of outbreaks over the 2 years was used to calculate the incidence per 1 million population per year. If a state only reported outbreaks for 1 year, the incidence per 1 million population for that single year was used.

During 2009–2010, NORS only allowed reporting of the percentage of the number of cases in each age and sex category. An age group category "unknown" is included in the analysis because states were allowed to enter the percentage of cases that were of unknown age as part of the total. Only outbreaks with complete information were included in each analysis; reports in which the total age or sex percentages were not entered or did not add to 100% (+/- 2% to account for rounding errors) were excluded from that analysis.

Comparisons of symptoms and case outcomes by etiology were performed using Pearson's chi square test. Comparisons of the mean number of cases reported in outbreaks from different etiologies were performed between each pair of etiologies using the Tukey method in conjunction with one-way analysis of variance. Comparisons of median attack rates among the different etiologies were performed using the Kolmogorov-Smirnov test, and comparisons of the staff and guest/resident group were performed using the signed rank test. All analyses were performed using SAS v 9.2 (SAS Institute, Inc.; Cary, North Carolina). Significance was determined by p<0.05 for all analyses.

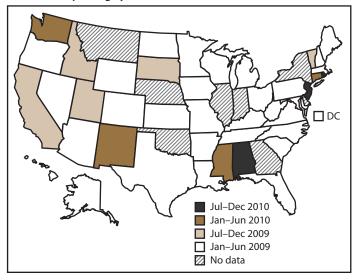
Results

Reporting Sites

As of January 24, 2012, a total of 2,340 enteric person-toperson outbreaks occurring during 2009-2010 were reported to CDC through NORS. Of these, 2,259 outbreak reports were marked finalized by a state administrator and met the inclusion criteria (972 for 2009 and 1,287 for 2010). These outbreaks were reported in 43 reporting sites representing 42 states and the District of Columbia. Eight states (Delaware, Georgia, Illinois, Indiana, Montana, Nebraska, New York, and Oklahoma), five U.S. territories (American Samoa, Guam, the Commonwealth of the Northern Mariana Islands, Puerto Rico, and the U.S. Virgin Islands), and three Freely Associated States (the Federated States of Micronesia, the Republic of the Marshall Islands, and the Republic of Palau) reported no person-to-person AGE outbreaks occurring during 2009–2010 that met the inclusion criteria. Several of these sites, primarily the territories (excluding Puerto Rico) and Freely Associated States, have not reported any outbreaks in NORS or reported only one or two outbreaks of any type during 2009-2010. Puerto Rico and the eight U.S. states have regularly reported foodborne and waterborne outbreaks in NORS but have not reported any or have only reported very few outbreaks from person-to-person transmission.

Of the 43 reporting sites with at least one finalized personto-person AGE outbreak report, 36 either began entering data in early 2009 (during the launch period) or reported these outbreaks retrospectively (Figure 1). Of the 36 states reporting outbreaks occurring in 2009, five did not enter reports for

FIGURE 1. Date of first reported acute gastroenteritis outbreak transmitted by person-to-person contact, by state — National Outbreak Reporting System, United States, 2009–2010

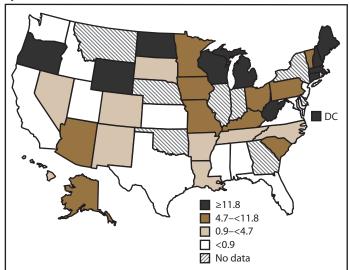


outbreaks occurring before July 2009. Forty-one states reported at least one outbreak occurring in 2010. Of the 43 sites that reported at least one outbreak occurring during 2009–2010, a total of 16 sites reported <10 outbreaks each, 18 reported 10–99 outbreaks each, and nine reported >100 outbreaks each (range: 1–276 outbreaks per site). Across all sites, the median outbreak incidence was 4.7 per million population per year (mean: 7.9; range: 0.03–41.9) (Figure 2).

Etiologies

Of the 2,259 person-to-person AGE outbreaks, 840 (37%) had an unknown etiology, 1,410 (62%) had a single suspected or confirmed etiology, and nine (<1%) had multiple etiologies (Figure 3). Norovirus was the only suspected or confirmed etiology reported in 1,270 (56%) outbreaks. Shigella was the second most commonly reported etiology, although it accounted for only 86 (4%) of all reported outbreaks. Other single-etiology outbreaks were suspected or confirmed to be caused by Salmonella (n = 16), rotavirus (n = 10), Shiga toxinproducing *E. coli* (STEC) (n = 11), *Giardia lamblia* (n = 5), *Cryptosporidium spp.* (n = 9), *Clostridium difficile* (n = 4), sapovirus (n = 2), and Campylobacter jejuni (n = 1). Nine outbreaks had multiple etiologies: norovirus and *Clostridium spp.* (n = 5), norovirus and rotavirus (n = 1), Salmonella enterica and STEC (n = 1), norovirus and *Bacillus sp.* (n = 1), and norovirus, rotavirus, and *Clostridium difficile* (n = 1).

FIGURE 2. Rate* of outbreaks of acute gastroenteritis transmitted by person-to-person contact, by state[†] — National Outbreak Reporting System, United States, 2009–2010



^{*} Incidence of outbreaks per state, per million population, on the basis of U.S. Census Bureau population estimates.

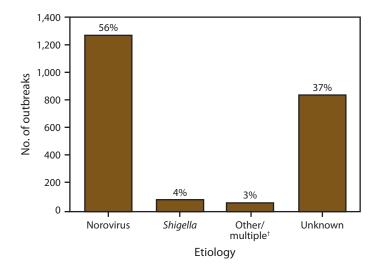


FIGURE 3. Number* and percentage of outbreaks of acute gastroenteritis transmitted by person-to-person contact, by etiology —

National Outbreak Reporting System, United States, 2009–2010

* N = 2,259.

⁺ Includes Salmonella (n = 16), rotavirus (n = 10), Shiga toxin-producing *E. coli* (STEC) (n = 11), *Giardia lamblia* (n = 5), *Cryptosporidium spp.* (n = 5), *Clostridium difficile.* (n = 4), sapovirus (n = 2), *Campylobacter jejuni* (n = 1), norovirus and *Clostridium spp.* (n = 5), norovirus and rotavirus (n = 1), *Salmonella enterica* and STEC (n = 1), norovirus and *Bacillus sp.* (n = 1), and norovirus, rotavirus, and *Clostridium difficile* (n = 1).

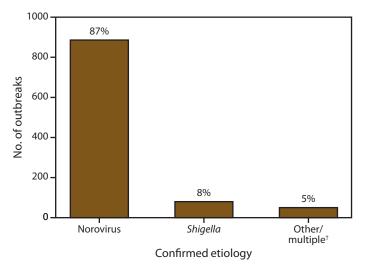
Of the 1,419 outbreaks with at least one reported etiology, 1,016 (72%) reported at least one laboratory confirmed etiology and 403 (28%) outbreaks reported only a suspected etiology. Of the 1,016 outbreaks with at least one laboratory confirmed etiology, 886 (87%) reported norovirus as the only confirmed etiology, and 80 (8%) listed *Shigella spp*. (Figure 4). Other laboratory confirmed single-etiology outbreaks were caused by *Salmonella enterica* (n = 16), STEC (n = 9), rotavirus (n = 7), *Giardia lamblia* (n = 4), *Cryptosporidium spp*. (n = 4), *Clostridium difficile* (n = 3), sapovirus (n = 2), and *Campylobacter jejuni* (n = 1). Four outbreak reports listed ≥ 2 confirmed etiologies: norovirus and *Clostridium difficile* (n = 1).

Seasonality

Most (79%) of the 2,259 outbreaks occurred during winter months (Figure 5). This pattern is attributed to the substantial number of suspected and confirmed norovirus outbreaks, which exhibit strong winter seasonality. An estimated 83% of norovirus outbreaks occurred during October–March. Approximately 80% of outbreaks of unknown etiology also occurred during the winter season. In contrast, outbreaks caused by suspected or confirmed *Shigella* were relatively more

⁺ For states reporting outbreaks in both 2009 and 2010, the average number of outbreaks per year was used to calculate the incidence. For states reporting outbreaks for 1 year, the incidence per 1 million population for that single year was used.

FIGURE 4. Number* and percentage of outbreaks of acute gastroenteritis transmitted by person-to-person contact, by confirmed etiology — National Outbreak Reporting System, United States, 2009–2010



* N = 1,016.

⁺ Includes Salmonella enterica (n = 16), STEC (n = 9), rotavirus (n = 7), Giardia lamblia (n = 4), Cryptosporidium spp. (n = 4), Clostridium difficile (n = 3), sapovirus (n = 2), Campylobacter jejuni (n = 1), norovirus and Clostridium difficile (n = 2), norovirus and Bacillus sp. (n = 1), and norovirus, rotavirus, and Clostridium difficile (n = 1).

frequent during summer months, although an insufficient number of these outbreaks existed to determine a consistent seasonal pattern. Only 38% of *Shigella* and 40% of other or multiple etiology outbreaks occurred during October–March.

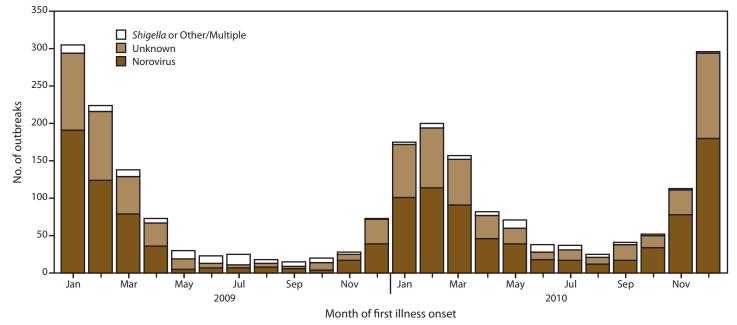
Outbreak-Associated Cases

In the 2,259 person-to-person AGE outbreaks reported in NORS, 81,491 cases were identified, with a mean of 36 cases per outbreak (median: 26; range: 2–394) (Table 1). The mean number of cases per outbreak was significantly higher for suspected or confirmed norovirus outbreaks (44 cases) than for outbreaks caused by *Shigella* (15 cases), multiple or other etiology (22 cases), or an unknown etiology (27 cases). Outbreaks suspected or confirmed to be caused by *Shigella* had a significantly lower mean number of cases per outbreak (15 cases) than that of outbreaks caused by an unknown etiology (27 cases).

Of the 1,038 outbreak reports of any etiology with complete information on distribution of cases by sex, 69% of cases occurred in females (Figure 6). Of the 627 outbreaks with a suspected or confirmed etiology of norovirus, 71% of cases occurred among females. Cases reported in outbreaks suspected or confirmed to be caused by *Shigella* were as likely to occur among males as females. In outbreaks with another etiology or unknown etiology, 66% of cases were in females, following a similar pattern to those associated with norovirus outbreaks.

Of the 936 outbreak reports containing information on age group distribution of cases, most cases (54%) occurred among those aged >49 years. This pattern can be largely explained by the substantial number of suspected and confirmed norovirus outbreaks (N = 550), in which 58% of cases occurred among those aged >49 years (Figure 7). Conversely, 76% of cases in suspected or confirmed *Shigella* outbreaks occurred in children

FIGURE 5. Number* of outbreaks of acute gastroenteritis transmitted by person-to-person contact, by month of first illness onset and etiology — National Outbreak Reporting System, United States, 2009–2010

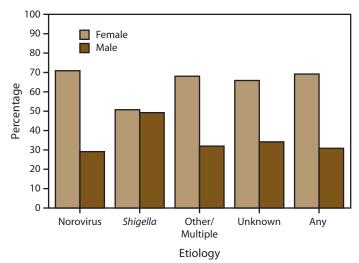


* N = 2,259.

Etiology	Total cases	No. of outbreaks	Mean cases	Median cases	Range
Norovirus	56,053	1,270	44	33	2–357
Shigella	1,305	86	15	7	2-105
Other/multiple	1,375	63	22	10	2-394
Unknown	22,758	840	27	20	2–246
Any	81,491	2,259	36	26	2-394

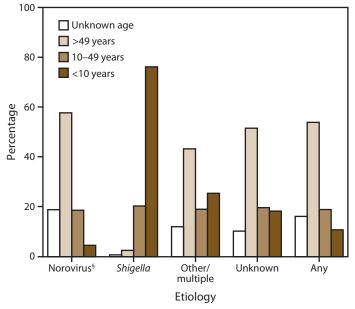
TABLE 1. Number of cases in outbreaks of acute gastroenteritis transmitted by person-to-person contact, by etiology — National Outbreak Reporting System, United States, 2009–2010

FIGURE 6. Percentage* of cases in outbreaks of acute gastroenteritis transmitted by person-to-person contact,⁺ by sex and etiology — National Outbreak Reporting System , United States, 2009–2010



* Percentages might not total 100% because of rounding. † N = 1,038.

FIGURE 7. Percentage* of cases in outbreaks of acute gastroenteritis transmitted by person-to-person contact,[†] by age group distribution and etiology — National Outbreak Reporting System, United States, 2009–2010



* Percentages might not total 100% because of rounding. $^{\dagger}_{1}$ N = 936.

[§] N = 550.

aged <10 years. In outbreaks caused by another or unknown etiology, 43% and 52% of cases, respectively, occurred in persons aged >49 years, similar to those associated with norovirus outbreaks. However, these outbreaks also involved relatively larger proportions (26% and 18%, respectively) of children aged <10 years than norovirus outbreaks (5%).

Settings

The age distribution of cases by etiology largely relate to the settings in which these outbreaks occurred. Overall, of the 1,187 person-to-person AGE outbreak reports containing information on setting, most were commonly identified in LTCFs, schools, childcare centers, and hospitals (Table 2). Outbreaks caused by norovirus or an unknown etiology occurred most frequently in LTCFs (86% and 77%, respectively); thus, most cases occurred in older adults. Outbreaks caused by Shigella occurred almost exclusively (97%) in childcare centers; thus, the majority of cases were reported in children aged <10 years. Other settings reported included other health-care facilities (n = 26), camp (eight), private settings (seven), prison or detention facilities (three), restaurants (three), athletic facilities (three), youth centers (two), hotels (two), adult day care facility (one), a harbor (one), and communitywide (e.g., not limited to a single setting) (one).

Symptoms and Clinical Outcomes

Information on the proportion of cases reported with at least one symptom was available for 1,156 (51%) outbreaks, although not all cases or all outbreaks provided information on each of the four symptoms analyzed in this report (Figure 8). Among cases for which information was available, diarrhea and vomiting were the most common symptoms reported (80% of 39,055 cases and 65% of 37,464 cases, respectively). Outbreaks caused by norovirus or unknown etiology involved vomiting in a significantly higher percentage of cases (65% and 68%, respectively) than in outbreaks caused by any other suspected or confirmed etiology (43% of cases). Fever and bloody stools were each reported significantly more often in outbreaks caused by *Shigella* (32%), multiple, or other etiologies (15%) than

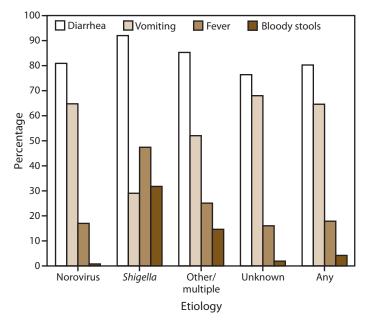
	Total	LT	CF	Childca	re center	Sch	lool	Hos	oital	Oth	ner*
Etiology	outbreaks	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)
Norovirus	714	617	(86)	13	(2)	21	(3)	32	(5)	31	(4)
Shigella	30	0	(0)	29	(97)	0	(0)	0	(0)	1	(3)
Other/multiple	30	11	(37)	10	(33)	3	(10)	0	(0)	6	(20)
Unknown	413	320	(77)	15	(4)	37	(9)	22	(5)	19	(5)
Any	1,187	948	(80)	67	(6)	61	(5)	54	(5)	57	(5)

TABLE 2. Number and percentage of outbreaks of acute gastroenteritis transmitted by person-to-person contact, by setting and etiology — National Outbreak Reporting System, United States, 2009–2010

Abbreviation: LTCF = long-term-care facilities.

* Other settings reported included other health-care facilities (N=26), camp (8), private settings (7), prison or detention facilities (3), athletic facilities (3), restaurants (3), youth centers (2), hotels (2), an adult day care facility (1), a harbor (1), and communitywide (1).

FIGURE 8. Percentage*of outbreaks of acute gastroenteritis transmitted by person-to-person contact, by symptom and etiology — National Outbreak Reporting System, United States, 2009–2010



* Percentage calculations do not include missing data; different denominators are used for each category (e.g., etiology by reported system).

with outbreaks caused by norovirus or an unknown etiology (1% and 2%, respectively).

During 2009–2010, a total of 136 deaths were reported among 1,670 outbreaks with information on death, and 1,339 hospitalizations were reported among 1,576 outbreaks with information on hospitalizations. Of the 94 outbreak reports with an associated death, 61 outbreaks occurred in an LTCF, three in a hospital or other health-care setting, and one in a childcare center; setting was not reported for 29 outbreaks. In 80 (85%) of these 94 outbreak reports, which accounted for 118 (87%) deaths, norovirus was identified as the only suspected or confirmed etiology. *Cryptosporidium sp.* and *E. coli* O157:H7 were the confirmed etiology in one outbreak each, and each was associated with one reported death. A total of 16 reported deaths occurred in 12 outbreaks for which an etiology was not reported.

Patients reported in outbreaks caused by *Shigella* were significantly more likely to seek health care (i.e., outpatient health-care provider visits, emergency department visits, and hospitalizations combined) than cases associated with norovirus outbreaks (odds ratio: 10.2; 95% confidence interval = 9.3-11.2); however, norovirus outbreaks contributed to the largest number of deaths and health-care visits (Table 3).

Attack Rates

Unique to person-to-person AGE outbreak surveillance, data are collected on the total number of persons exposed, categorized as either guests and residents or staff, thereby allowing for calculation of attack rates. The median attack rate for guests and residents was significantly higher in suspected or confirmed norovirus outbreaks (35%) than in *Shigella* outbreaks (12%) (Table 4). Likewise, the median attack rate for guests and residents in outbreaks of unknown etiology (21%) also was significantly higher than in *Shigella* outbreaks. The median staff attack rate in norovirus outbreaks (19%) was significantly higher than the median staff attack rate in *Shigella* outbreaks (8%). For each outbreak etiology, the median attack rates were significantly lower among staff than among guests and residents.

Discussion

NORS is a novel reporting system that provides the first national data on AGE outbreaks spread through personto-person transmission, highlighting the frequency of these outbreaks. During 2009–2010, a total of 2,259 person-toperson AGE outbreaks and 81,491 outbreak-related illnesses were reported to CDC via NORS from 43 reporting sites. The actual frequency of these outbreaks is likely underestimated, as evidenced by the inconsistent levels of reporting in NORS of outbreaks from different modes of transmission. In 2009, a total

	Dea	iths	Hospitaliz	ed patients		vho visited ED	Patients who sought health care [†]		
Etiology	No.	(%)	No.	(%)	No.	(%)	No.	(%)	
Norovirus	118	(0.3)	919	(2)	373	(2)	1,639	(10)	
Shigella	0	(0.0)	50	(6)	97	(16)	551	(69)	
Other/multiple	2 [§]	(0.2)	57	(4)	20	(5)	116	(25)	
Unknown	16	(0.1)	313	(2)	129	(2)	369	(6)	
Any	136	(0.2)	1,339	(2)	619	(2)	2,675	(11)	

TABLE 3. Number and percentage* of cases in outbreaks of acute gastroenteritis transmitted by person-to-person contact, by clinical outcome and etiology — National Outbreak Reporting System, United States, 2009–2010

Abbreviation: ED = emergency department.

* Percentages might not round to 100% because each percentage was calculated using a different denominator.

[†] Excludes ED visits and hospitalizations.

[§] Confirmed Cryptosporidium sp. and E. coli O157:H7 outbreaks were responsible for one death each.

TABLE 4. Attack rates among guests and residents or staff in outbreaks of acute gastroenteritis transmitted by person-to-person contact, by etiology — National Outbreak Reporting System, United States, 2009–2010

Etiology	Median guest/resident attack rate* %	Range (%)	Median staff attack rate [†] %	Range (%)	p-value [§]
Norovirus	35	(1.0–100)	19	(0-100)	p<0.001
Shigella	12	(3–51)	8	(0-38)	0.001
Other/multiple	31	(0-78)	12	(0-75)	0.03
Unknown	21	(1-100)	8	(0-100)	<0.0001
Any	30	(0–100)	13	(0–100)	<0.0001

* N = 976 outbreaks.

[†] N = 768 outbreaks.

§ As calculated using the signed rank test.

of 972 person-to-person AGE outbreaks and 33,085 illnesses were reported in NORS by only 36 sites. In comparison, during the same year, 668 foodborne outbreaks and 13,497 illnesses were reported by 45 sites (7). This further illustrates that person-to-person transmission is an important cause of AGE outbreaks in the United States, although surveillance for these outbreaks has only recently been prioritized and has not yet been fully implemented in all states.

Norovirus was the most frequently reported cause of personto-person AGE outbreaks. It was reported as the only suspected or confirmed etiology in 56% of all person-to-person AGE outbreaks and in 89% of outbreaks with an etiology reported. These data are consistent with other studies indicating that norovirus is the leading cause of AGE outbreaks and that person-to-person transmission is the most common mode of transmission (8-13). Consistent with previous reports (9-16), LTCFs were the most frequent setting of person-to-person norovirus outbreaks (86%) reported in NORS, which might explain, in part, the predominance of cases among females and older adults. The 2004 National Nursing Home Survey indicated that 71% of nursing home patients were female (17), which is a similar proportion of males and females in norovirus outbreaks (86%) reported in NORS. However, among the 936 outbreak reports that include complete information

by age, only 58% of cases in norovirus outbreaks occurred among persons aged >50 years. This might be explained, in part, by outbreaks in which not all ages of persons with cases were known; age was unknown in 19% of cases in norovirus outbreaks for which at least some age information was provided for the outbreak report. In addition, information on patient age was reported in fewer than half of all outbreaks reported, possibly introducing some bias. Nursing home outbreaks also included cases among staff members, which might have shifted the age of cases downward relative to the age of the resident populations.

The median attack rates reported for suspected and confirmed person-to-person norovirus outbreaks were significantly higher than those reported for suspected and confirmed *Shigella* outbreaks. This high norovirus attack rate is consistent with other attack rates calculated for outbreaks in LTCFs and previous reports in the literature (10,13). The lower attack rates among staff might result from several factors including better hand hygiene practices, immunity acquired from more frequent exposures to these pathogens from working in a high-risk setting (18, 19), reluctance to report illness (20, 21) or, in settings such as LTCFs, staff members who are younger than their residents and therefore less likely to experience symptomatic or severe disease (12, 13, 16, 22-24).

The overall case-hospitalization and case-fatality rates for person-to-person norovirus outbreaks also were consistent with other studies conducted during norovirus outbreaks in LTCFs (10, 12-15). Although hospitalizations and deaths represent a relatively small fraction of all person-to-person norovirus outbreak-associated cases (2% and 0.3%, respectively), the high frequency of these outbreaks resulted in 118 reported deaths and 919 hospitalizations during 2009–2010. Norovirus infection often results in a mild, self-limiting illness; however, consequences of norovirus disease in elderly and immunosuppressed populations could be especially severe and have been previously reported to include hospitalization and death (12, 13, 16, 22-24).

Furthermore, other studies have demonstrated that all-cause gastroenteritis-associated hospitalizations and deaths appear to be increasing, which might be attributable at least in part to norovirus. In the United States, sporadic and outbreakassociated norovirus causes an estimated 800 deaths and 70,000 hospitalizations each year. These numbers increase by up to 50% during epidemic years associated with emergent strains (25,26). The highest rates of norovirus-associated hospitalizations occurred among adults aged ≥ 65 years; within this group, rates increased with advancing age (25). The elderly also accounted for 83% of AGE-associated deaths, of which norovirus was the second leading infectious cause after Clostridium difficile (26). A literature review of norovirus outbreaks during 1993-2011 indicated that hospitalizations and deaths were significantly more likely when outbreaks occurred in health-care settings, including LTCFs (27). The findings in this report affirm that norovirus can account for substantial morbidity and mortality among persons with acute gastrointestinal illness in the United States, particularly among the elderly.

Person-to-person AGE outbreaks of unknown etiology reported through NORS had epidemiologic characteristics consistent with norovirus outbreaks. Norovirus illnesses tend to peak during winter months, and many enteric bacterial illnesses tend to predominate in summer (9,11,25,26,28-30). Both the confirmed and suspected norovirus outbreaks and outbreaks of unknown etiology reported through NORS exhibited a strong winter seasonality, whereas outbreaks caused by other etiologies demonstrated slight peaks during spring and summer months. Norovirus and unknown etiology outbreaks also exhibited similarly high frequencies of diarrhea and vomiting and low frequencies of fever and bloody stools. A high proportion of patients with vomiting $(\geq 50\%)$ and a relatively low proportion with fever are characteristics that have been previously demonstrated as helpful in differentiating norovirus outbreaks from AGE outbreaks caused by other etiologies (31,32). However, bloody stools are more commonly associated with

bacterial infections than with norovirus infections (8,33-36). Other characteristics common among norovirus and unknown etiology outbreaks reported through NORS included a higher frequency of cases among older adults and females, a high number of cases in LTCFs, and high attack rates. These findings suggest that many of the outbreaks of unknown etiology might have been caused by norovirus.

Although surveillance for person-to-person AGE outbreaks is predominated by norovirus, other etiologies were identified as important contributors. Shigella was suspected or confirmed to have caused 86 person-to-person AGE outbreaks and 1,305 outbreak-related illnesses reported through NORS during the first 2 years of the system. Similar to norovirus, Shigella has a low infectious dose and is commonly transmitted person-toperson (37-39). However, the profile of Shigella outbreaks was distinct from norovirus and unknown etiology outbreaks, likely in part because of the difference in setting. Almost all (97%) Shigella outbreaks for which setting was reported occurred in a childcare center, thus explaining why approximately 75% of reported outbreak-associated cases occurred among children aged <10 years. Most cases of shigellosis are identified in children aged <5 years, and Shigella is a well-recognized cause of AGE outbreaks in childcare facilities (29,37,40-45). Shigella outbreaks tend to affect more females than males, but outbreaks in young children tend to have an equal distribution of male and female patients, as is reflected in the findings of this report (29,41).

Cases involved in *Shigella* outbreaks were more likely to include fever and bloody stools than cases in norovirus outbreaks, and substantially more patients in *Shigella* outbreaks sought health care than those involved in norovirus outbreaks (69% and 10%, respectively). These outbreaks might have even broader implications because transmission to household contacts is common during *Shigella* outbreaks (44–46), and adults, particularly those aged ≥65, have much higher hospitalization rates for shigellosis than children (37). Furthermore, antibiotics are widely used during shigellosis outbreaks in the United States, yet multidrug resistance is common among *Shigella* bacteria, limiting antibiotic treatment options (41,43,44).

Some reported etiologies in person-to-person AGE outbreaks, including STEC and *Salmonella*, are primarily considered foodborne pathogens, and before 2009, national surveillance systematically captured only foodborne outbreaks of these pathogens. In 2009, a total of 119 foodborne outbreaks of *Salmonella* were reported via NORS; five additional *Salmonella* outbreaks were reported through person-to-person AGE outbreak surveillance. An estimated 40 foodborne outbreaks of STEC were reported in 2009, and five additional STEC outbreaks were reported as transmitted by person-to-person

contact (7). In 2010, six additional STEC person-toperson outbreaks were reported, including one outbreak of STEC O157:H7 in a childcare facility that resulted in a death. Studying these outbreaks is especially important because person-to-person transmission of STEC is a recognized cause of outbreaks in childcare settings, and young children are most at risk for STEC infection and the complication hemolytic uremic syndrome (HUS) (47). Surveillance for person-to-person AGE outbreaks via NORS also might be important in clarifying the epidemiology and role of non-O157 STEC, which has been underrecognized because of limitations in surveillance and diagnostics (48–50).

Limitations

The findings in this report are subject to at least two limitations. First, similar to many other passive reporting systems, NORS is subject to underreporting. NORS ultimately relies on health-care providers and facilities reporting potential outbreaks to state and local health departments, which often depends on the general public seeking medical care. However, only 15%-20% of persons with an acute diarrheal illness seek medical care and only 17%–19% of those submit a stool sample for testing (51, 52). This type of underreporting is influenced by behavioral patterns and nuances of the health-care system in the United States. In addition, underreporting to NORS probably occurs because state and local health departments, which often have limited resources and competing responsibilities, might not have the resources available to investigate each potential outbreak or submit a completed report in NORS. Furthermore, NORS is a new reporting system with variable adoption and use, which might vary by modes of illness transmission. For example, Puerto Rico and the eight U.S. states that did not have any NORS reports that met the inclusion criteria for this summary have been regularly reporting foodborne and waterborne outbreaks in NORS but have not reported any or have only reported very few outbreaks from person-to-person transmission. Only 36 sites reported person-to-person AGE outbreak data in 2009, compared with 45 sites that reported foodborne outbreaks during the same year (7). The primary mode of illness transmission also is determined by each reporting site and case definitions or classification schemes might not be consistent across all sites. Notably, the most populous states reported no or disproportionately low numbers of person-to-person AGE outbreaks, suggesting that the number of outbreaks in this report is an underestimate, and the findings reported might not be generalizable. System coverage could improve as sites become more familiar with NORS and as features are enhanced or added to facilitate reporting.

Second, only four variables (outbreak identification, date of first illness, reporting site, and total ill) are required to submit an outbreak report via NORS. Although this affords flexibility to sites that would like to report outbreaks for which they have only limited information available, it results in variable levels of completeness of other variables collected in the system (e.g., age, sex, etiology, and setting).

Conclusion

The findings in this report enable a better understanding of the frequency, causes, and patient outcomes of AGE outbreaks in the United States, especially those caused by person-toperson transmission. During 2009–2010, norovirus was the most frequently reported cause of person-to-person AGE outbreaks. On the basis of epidemiologic characteristics, norovirus also might be responsible for a substantial portion of the 840 reported outbreaks of unknown etiology. An additional 86 outbreaks were reported to be caused by *Shigella*. No vaccines exist for either norovirus or *Shigella* in the United States, and recommendations for prevention and control of person-to-person AGE outbreaks depend primarily on appropriate hand hygiene and isolation of ill persons.

Although norovirus and *Shigella* were associated with the majority of AGE outbreaks, they were not the only reported cause of person-to-person AGE outbreaks. Approximately 3% of outbreaks were caused by other or multiple etiologies, many of which are considered primarily foodborne pathogens but can be transmitted through multiple routes, such as *Salmonella* and STEC. Further study of these person-to-person AGE outbreaks should provide a better understanding of these pathogens and how they can be spread. Similarly, further examination of outbreaks of unknown etiology could help identify barriers to making an etiologic determination, to analyze clinical and epidemiologic clues suggestive of a probable etiology, and to discover new and emerging etiologic agents.

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Outbreak-associated Salmonella enterica Serotypes and Food Commodities, United States, 1998–2008

Brendan R. Jackson, Patricia M. Griffin, Dana Cole, Kelly A. Walsh, and Shua J. Chai

Salmonella enterica infections are transmitted not only by animal-derived foods but also by vegetables, fruits, and other plant products. To clarify links between Salmonella serotypes and specific foods, we examined the diversity and predominance of food commodities implicated in outbreaks of salmonellosis during 1998–2008. More than 80% of outbreaks caused by serotypes Enteritidis, Heidelberg, and Hadar were attributed to eggs or poultry, whereas >50% of outbreaks caused by serotypes Javiana, Litchfield, Mbandaka, Muenchen, Poona, and Senftenberg were attributed to plant commodities. Serotypes Typhimurium and Newport were associated with a wide variety of food commodities. Knowledge about these associations can help guide outbreak investigations and control measures.

Salmonella enterica is estimated to cause 1.2 million Sillnesses each year in the United States and to be the leading cause of hospitalizations and deaths from foodborne disease (1). Because of the major public health role of Salmonella infections, the US Department of Health and Human Services has made decreasing the nationwide incidence of these infections by 25% a Healthy People 2020 national goal (2). Overall, salmonellosis incidence has not decreased in the past decade; the incidence has substantially increased for some serotypes and decreased for others (2,3). Focused attention on determining sources of Salmonella infections will be vital to reach the 25% target reduction in these infections.

Salmonella serotypes differ in their natural reservoirs and ability to cause human infections (4-6); only a small proportion of >2,500 serotypes cause most human infections

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(4,7). In 2009, only 20 serotypes comprised >82% of the \approx 36,000 serotyped human-derived *Salmonella* isolates in the United States that were reported to the Centers for Disease Control and Prevention (3). A few serotypes have been associated with specific animal reservoirs. For example, serotype Dublin, which caused 103 laboratory-confirmed human infections in 2009 (3), is found predominantly in cattle (5). However, reservoir sampling alone has limited use in predicting the contribution of a reservoir to the incidence of human illness (8).

Outbreak data and case–control studies have linked some serotypes to certain foods or exposures (e.g., serotype Enteritidis to eggs and chicken) (9-11). Information obtained during outbreak investigations is a key tool in understanding which foods are common sources of pathogens contributing to foodborne infections. During outbreak investigations, illnesses can be linked to a particular food by using epidemiologic or laboratory evidence (12). To our knowledge, no systematic examination of *Salmonella* serotypes and food vehicles implicated in outbreaks has been reported. We analyzed foodborne disease outbreak data to determine associations between food commodities and serotypes to help inform future outbreak investigations, foodborne illness source attribution analyses, and control measures.

Methods

State, local, and territorial health departments voluntarily submit reports of foodborne disease outbreak investigations to the Foodborne Disease Outbreak Surveillance System (FDOSS) of the Centers for Disease Control and Prevention. A foodborne disease outbreak is defined as ≥ 2 cases of a similar illness resulting from ingestion of a common food. Submitted reports include a description of the pathogen, the implicated food(s), the main ingredients of

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the food, and the contaminated ingredient, if known (13). When a *Salmonella* sp. is the etiologic agent, public health laboratories serotype the isolate. A *Salmonella* sp. is considered the confirmed etiology of an outbreak when the same serotype is isolated from ≥ 2 ill persons or when the bacterium is isolated from an epidemiologically implicated food (13).

To standardize the analysis of foods, we used a modified version of an existing classification scheme (14) to categorize reported foods into 1 of 20 mutually exclusive food commodities. Foods were classified into a single food commodity if a single ingredient was implicated or if all ingredients in a food product belonged to a single food commodity. We then combined the individual food commodities into 3 broad food commodities (crustaceans, fish, and mollusks); 2) land animal–derived food commodities (dairy, eggs, beef, game, pork, chicken, turkey, and duck); and 3) plant-derived food commodities (grains–beans, oils–sugars, fruit, nuts, fungi, sprouts, leafy vegetables, root vegetables, and vine–stalk vegetables).

We reviewed all reports of foodborne outbreaks of *Salmonella* infections to FDOSS during 1998–2008 and included in the analysis those outbreaks caused by a single, laboratory-confirmed serotype. We excluded outbreaks in which multiple etiologies were reported, that had an unknown serotype, or that could not be assigned to 1 of the 20 food commodities.

Among all salmonellosis outbreaks and for each *Salmonella* serotype, we calculated the frequency and percentage of outbreaks associated with each food commodity. For each serotype, we also determined the percentage of outbreaks associated with animal-derived food commodities (land and aquatic) and plant-derived food commodities. We calculated the Gini coefficient as a descriptive measure of the magnitude of food commodity diversity, or inequality (*15*) among outbreaks caused by a particular serotype. The Gini coefficient was chosen as a measure of diversity because it provides an easily interpretable range of values from 0 to 1. A Gini coefficient of 0 indicates an equal distribution of outbreaks caused by a serotype across all food commodities, and a value of 1 indicates that all outbreaks were attributed to a single food commodity.

Results

During 1998–2008, a total of 1,491 outbreaks of *Salmonella* infections were reported to FDOSS, and 1,193 (80%) were caused by a single serotype. Of the single-serotype outbreaks, 595 (50%) had an implicated food, and 403 (34%) could be assigned to a single food commodity. Among these 403 outbreaks, 47 serotypes were reported; 23 serotypes caused \geq 3 outbreaks. Of the 47

serotypes reported, the 4 most common caused 66% of the 403 outbreaks (Enteritidis 144 [36%], Typhimurium 58 [14%], Newport 40 [10%], and Heidelberg 24 [6%]). Overall, eggs were the most commonly implicated food commodity (112 outbreaks, 28%), followed by chicken (64 outbreaks, 16%), pork (37 outbreaks, 9%), beef (33 outbreaks, 8%), fruit (33 outbreaks, 8%), and turkey (28 outbreaks, 7%) (Table 1, Appendix, wwwnc.cdc.gov/EID/ article/19/8/12-1511-T1.htm).

The most commonly implicated food commodity differed by Salmonella serotype (Table 1). Eggs were the most common food commodity for outbreaks caused by serotypes Enteritidis (93 [65%] of 144 outbreaks) and Heidelberg (10 [42%] of 24 outbreaks). Egg-associated serotype Enteritidis outbreaks accounted for 23% of all single food commodity outbreaks. Chicken was the most common food commodity for serotypes I 4,[5],12:i- (3 [75%] of 4 outbreaks) and Typhimurium (15 [26%] of 58 outbreaks). Pork was the most common food commodity for serotypes Uganda (all 4 outbreaks) and Infantis (4 [57%] of 7 outbreaks). Fruit was the most common food commodity for serotypes Litchfield (all 5 outbreaks), Poona (all 4 outbreaks), Oranienburg (2 [50%] of 4 outbreaks), and Javiana (3 [30%] of 10 outbreaks). Turkey was the most common food commodity for serotypes Hadar (3 [38%] of 8 outbreaks) and Saintpaul (3 [33%] of 9 outbreaks). Sprouts were the most common food commodity for serotype Mbandaka (3 [75%] of 4 outbreaks). Food commodities in the aquatic animal group were the most common for serotype Weltevreden (2 [67%] of 3 outbreaks). Animalderived food commodities were implicated in >90% of outbreaks caused by serotypes Enteritidis, Heidelberg, Hadar, I 4,[5],12:i:-, Uganda, and Weltevreden, whereas plantderived food commodities were implicated in >50% of outbreaks caused by serotypes Javiana, Litchfield, Mbandaka, Muenchen, Poona, and Senftenberg.

Evaluation of the serotype diversity within food commodity categories (Table 2, Appendix, wwwnc.cdc.gov/ EID/article/19/8/12-1511-T2.htm) showed that the 112 egg-associated outbreaks were predominantly caused by Salmonella serotypes Enteritidis (83%) and Heidelberg (9%). Of the 64 chicken-associated outbreaks, 64% were caused by serotypes Enteritidis (28%), Typhimurium (23%), and Heidelberg (13%) combined. Among the 37 pork-associated outbreaks, serotypes Typhimurium (22%), Infantis (11%), Newport (11%), and Uganda (11%) were the most common etiology. The most common serotypes causing beef-associated outbreaks were Enteritidis (18%), Newport (18%), and Typhimurium (18%). Of the 33 fruitassociated outbreaks, 57% were caused by serotypes Newport (18%), Litchfield (15%), Enteritidis (12%), and Poona (12%) combined. Among the fruit-associated outbreaks, 17 (52%) were attributed to melons. The most common

serotypes causing melon-associated outbreaks were Litchfield (29%), Poona (24%), Newport (18%), and Javiana (12%). Of the 28 turkey-associated outbreaks, 53% were caused by serotypes Enteritidis (25%), Heidelberg (14%), and Typhimurium (14%) combined. Of the 21 vine-stalk vegetable-associated outbreaks, the most common serotypes were Newport (29%), Braenderup (14%), and Typhimurium (14%). Among the vine-stalk vegetable outbreaks, 19 (90%) were attributed to tomatoes. The most common serotypes causing tomato-associated outbreaks were Newport (32%), Typhimurium (16%), Braenderup (11%), Enteritidis (11%), and Javiana (11%). Of the 16 dairy-associated outbreaks, most were caused by serotypes Typhimurium (56%) and Newport (25%). Eleven outbreaks were associated with aquatic animal-derived food commodities, of which 5 (45%) were caused by serotype Enteritidis. Of the 10 leafy vegetable-associated outbreaks, 50% were caused by serotypes Newport (30%) and Javiana (20%).

Some serotypes were associated with a narrow range of food commodities. Among the 10 serotypes causing the most outbreaks in our study, *Salmonella* serotypes Enteritidis, Hadar, Heidelberg, and Infantis had the lowest diversity, or highest inequality (Gini coefficient ≥ 0.8), of implicated food commodities (Figure). Outbreaks caused by serotypes Enteritidis, Hadar, and Heidelberg were mostly attributed to eggs and poultry, and serotype Infantis outbreaks were mostly linked to pork. Serotypes Newport and Typhimurium had the greatest diversity (Gini coefficient <0.6), which reflected a wide range of implicated food commodities. Serotypes Braenderup, Javiana, Montevideo, and Saintpaul had modest diversity. Among them, serotype Montevideo outbreaks were mostly attributed to animal–derived food commodities (>80%); 30%–56% of outbreaks caused by serotypes Braenderup, Javiana, and Saintpaul were attributed to animal-derived food commodities.

Discussion

We found notable relationships between Salmonella serotypes and food commodities that point to major food reservoirs for different serotypes. Certain serotypes, in particular Enteritidis, Heidelberg, Hadar, and Infantis, caused outbreaks predominantly attributed to specific animal-derived food commodities, a finding that is consistent with results from animal reservoir sampling (6). We also identified serotypes that commonly caused outbreaks associated with plant-derived food commodities, particularly the fruit, vinestalk vegetable, sprouts, and leafy vegetable food commodities. These serotypes that cause plant-associated outbreaks are found relatively infrequently in Salmonella reservoir studies of livestock (6), which suggests that serotypes with non-livestock reservoirs (e.g., environmental, amphibian, or reptile reservoirs) may be more likely to cause outbreaks by plant-based food vehicles. For example, during an outbreak investigation of serotype Poona infections attributed to cantaloupe consumption, investigators suspected that melons might have been indirectly contaminated through packing equipment or wash water contaminated by reptiles (16). Our findings regarding plant-associated serotypes are particularly relevant given recent increases in Salmonella outbreaks attributed to fruits or vegetables and a concurrent increase in infections caused by serotype Javiana (3, 17), a serotype that compared with other common serotypes in this study, caused a higher percentage of plant-derived food commodity-associated outbreaks.

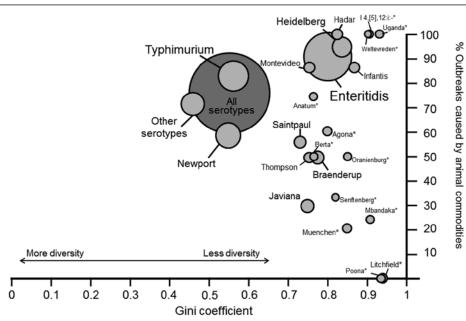


Figure. Gini coefficient and percentage of outbreaks attributed to animal commodities for each Salmonella enterica serotype, Foodborne Disease Outbreak Surveillance System, United States, 1998-2008. Size of circle indicates number of outbreaks for each serotype. Animal commodities include land animals (beef, chicken, eggs, game, pork, and turkey) and aquatic animals (crustaceans, fish, and mollusks). *Serotypes with ≤5 outbreaks. The Gini coefficient is a measure of diversity; a value of 0 indicates an equal distribution of outbreaks caused by a serotype across all commodities, and a value of 1 indicates that all outbreaks were attributed to a single commodity.

Our findings of predominant animal-derived food commodities for specific serotypes are supported not only by animal reservoir studies, but also by case-control studies of sporadic illness. Although the percentage of outbreaks attributed to a specific food commodity is not directly comparable to the population-attributable fraction estimated in case-control studies because the units of measure (outbreaks versus illnesses) and the method of estimating the sources of illnesses are different, our results and those of case-control studies show similar dominant food commodity reservoirs for some serotypes. For example, serotype Enteritidis was responsible for a high (83%) proportion of egg-associated outbreaks and $\approx 25\%$ of chicken and turkey outbreaks; these findings are supported by case-control studies that found eggs and poultry to be common sources of serotype Enteritidis infection (10,11).

The high percentages of serotype Heidelberg outbreaks attributed to eggs, chicken, and turkey are also supported by findings from case-control studies and previous reviews (18,19). These findings suggest that these products are common vehicles for this serotype. The link we found between serotype Hadar and turkey is consistent with historical data and animal surveillance data showing that serotype Hadar is now the most common serotype isolated from turkey (6). The link we found between serotype Infantis and pork is also consistent with animal surveillance data showing that this serotype is commonly isolated from swine but not poultry (6). Three of the 4 serotypes with the lowest food commodity diversity measured by the Gini coefficient (Enteritidis, Heidelberg, and Hadar) were predominantly associated with eggs and poultry, suggesting that these serotypes are well adapted to poultry reservoirs and are a well-defined target for control measures.

Two of the most common *Salmonella* serotypes, Typhimurium and Newport, had a wider range of implicated food commodities. Serotype Typhimurium has a wellcharacterized ability to infect various species (20) and can survive for a long time in the environment (21); these 2 factors enhance the ability of this serotype to be one of the most common causes of salmonellosis in the United States (2). Although we found serotype Typhimurium was associated with several animal commodities, the most common food commodity was chicken (26% of outbreaks), indicating that chicken is a major route of exposure. Among porkassociated outbreaks, Typhimurium was the most common serotype, which corroborates animal data showing that serotype Typhimurium has emerged as the predominant serotype in swine (6).

For *Salmonella* serotype Newport, diversity of implicated food commodities might be related to intraserotype genetic variation because several distinct clades have been identified (22). Antimicrobial drug resistance data might be helpful for differentiating serotype Newport infections transmitted through animal commodities versus those transmitted by plant-derived food commodities. A sporadic case-control study found associations between infection with multidrug-resistant strains of Salmonella serotype Newport and beef and egg consumption, whereas infection with pansusceptible strains was associated with direct or indirect exposure to frogs or lizards (23). In a similar manner, strains of serotype Newport causing several outbreaks attributed to beef or dairy products have been multidrug resistant (24, 25), whereas outbreaks attributed to produce have generally been pansusceptible (26,27). Therefore, pansusceptibility might be a marker for serotype Newport strains with environmental reservoirs and a greater potential for transmission though produce. Our findings support the hypothesis that Salmonella serotypes with environmental, amphibian, or reptile reservoirs might be more likely to be transmitted by fresh produce.

All outbreaks caused by *Salmonella* serotypes Litchfield and Poona were attributed to fruit. These 2 serotypes were responsible for 25% of fruit outbreaks despite representing only 2% of outbreaks caused by all serotypes in our study. Both serotypes have been established as reptile associated (28,29) and reptiles might play a role in fruit contamination (16). In a similar manner, 70% of outbreaks caused by serotype Javiana, a serotype associated with reptile and amphibian contact (30), were linked to plantderived food commodities.

Among Salmonella serotypes causing small numbers of outbreaks, several had particular animal reservoirs. This result is consistent with reported findings. For example, 2 of 3 serotype Weltevreden outbreaks were associated with aquatic animals, and serotype Weltevreden was the most common serotype found in a survey of imported seafood (31). Serotype Agona was responsible for 2 of the 3 outbreaks attributed to grains-beans, both traced to the same facility 10 years apart (32). This serotype was introduced into the United States in the 1970s by another dry food product, contaminated fishmeal used in livestock feed (33), which suggests good survival of this serotype in dry environments and products.

Salmonella serotype Agona also caused outbreaks attributed to chicken and turkey, consistent with animal surveillance data documenting its frequent isolation in swine, chicken, and turkey since its introduction in animal feed (6,34). All 4 serotype Uganda outbreaks were attributed to pork, and all 4 serotype I 4,[5],12:i:- outbreaks were linked to eggs or poultry, suggesting that these food products are reservoirs. Serotype I 4,[5],12:i:- emerged as a cause of human illness in the early 1990s and is now one of the 10 most common serotypes in humans in the United

States (35). Serotype Senftenberg is one of the most commonly isolated serotypes from turkeys and chickens (6) but was the cause of only a few outbreaks (all nonpoultry) in our study, suggesting that poultry is not the only food serving as a vehicle for transmission of serotype Senftenberg to humans.

Outbreak-associated illnesses represent only a small fraction of all Salmonella infections (1), and food vehicles responsible for outbreaks might differ from those causing sporadic infections. During the 11 years of our study, changes in product contamination frequency or consumption patterns might be associated with changes in the distribution of serotypes causing illness in the general population or the proportion of sporadic illnesses associated with specific food commodities. In a recent analysis of the distribution of serotypes causing foodborne disease outbreaks (36), the proportion of outbreaks caused by serotype Enteritidis decreased from 44% of Salmonella outbreaks during 1998-2000 to 24% during 2006-2008, and the percentage of outbreaks caused by S. enterica remained relatively constant. That study lacked the statistical power to detect changes over time in the percentages of outbreaks associated with most serotype-food commodity pairs, but found that the percentage of outbreaks caused by Salmonella and eggs decreased from 33% during 1998-1999 to 15% during 2006-2008.

Although outbreak data provide one of the only direct connections between food sources and infection, outbreak investigations are frequently unable to confirm the single contaminated food vehicle, limiting our ability to detect major changes over time. In our study, <33% of outbreaks had an implicated food that could be assigned a commodity. Investigators may also report suspected food vehicles on the basis of prior knowledge of the most likely foods associated with the serotype; this reporting technique would bias results toward these typical foods. Although genetic heterogeneity and differences in reservoirs exist within serotypes (22,37), our results demonstrate that serotyping provides helpful discrimination among certain serotypefood commodity pairs. Further subtyping of Salmonella serotypes could help identify major subtype-food commodity relationships, particularly for common serotypes like Enteritidis and Newport.

This systematic examination of foodborne disease outbreaks by *Salmonella* serotype and implicated food commodity provides major evidence linking serotypes to likely reservoirs and pathways of food contamination. Our analysis could have used outbreak-associated illnesses rather than outbreaks; the attributed sources would have been the same, but the percentages would have differed. However, the goal of this study was to describe major commodity sources by serotype, and this goal was not greatly influenced by the number of outbreak-associated illnesses. Using outbreaks or illnesses for analysis would not provide information about the proportion of sporadic illnesses that can be attributed to specific food commodities; more complex models are needed for such analyses (14). The results of our analysis can provide guidance to investigators when forming hypotheses about contaminated food sources during outbreak investigations, and in suggesting the likely contaminated ingredient in outbreaks associated with foods containing ingredients from multiple commodities. Investigators should also remain alert to uncommon or novel food vehicles, which are regularly being identified (38). Armed with knowledge of serotype– food commodity associations, public health officials may be able to more quickly form hypotheses, identify likely sources of contamination, and prevent illnesses.

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Dr Jackson is a medical epidemiologist at the Centers for Disease Control and Prevention and a lieutenant commander in the United States Public Health Service. His research interests include the epidemiology of enteric and environmental diseases.

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Outbreak-associated Salmonella enterica Serotypes and Food Commodities, United States, 1998–2008

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Research

Outbreak-associated Salmonella enterica Serotypes and Food Commodities, United States, 1998–2008

Commodity

Brendan R. Jackson , Patricia M. Griffin, Dana Cole, Kelly A. Walsh, and Shua J. Chai Author affiliations: Centers for Disease Control and Prevention, Atlanta, Georgia, USA Main Article

Table 1

Percentage of Salmonella enterica serotypes attributed to specific food commodities for 403 outbreaks, Foodborne Disease Outbreak Surveillance System, Unites States, 1998–2008*

Serotype (no. outbreaks)	Eggs	Chicken	Pork	Beef	Fruit	Turkey	Vine-stalk veg.	Sprouts	Dairy	Aquatic animals†	Leafy veg.	Game	Nuts	Grains-beans	Root veg.	Other‡
Enteritidis (144)	65	13	1	4	3	5	1	3	0	3	1	0	1	0	0	0
Typhimurium (58)	7	26	14	10	0	7	5	3	16	2	2	3	2	0	2	2
Newport (40)	0	13	10	15	15	8	15	0	10	0	8	3	0	0	5	0
Heidelberg (24)	42	33	0	4	4	17	0	0	0	0	0	0	0	0	0	0
Braenderup (10)	10	30	10	0	0	0	30	20	0	0	0	0	0	0	0	0
Javiana (10)	0	10	20	0	30	0	20	0	0	0	20	0	0	0	0	0

Table 1 - Outbreak-associated Salmonella enterica Serotypes and Food Commodities, United St...

Commodity

Serotype (no. outbreaks)	Eggs	Chicken	Pork	Beef	Fruit	Turkey	Vine–stalk veg.	Sprouts	Dairy	Aquatic animals†	Leafy veg.	Game	Nuts	Grains–beans	Root veg.	Other‡
Saintpaul (9)	11	0	0	11	11	33	11	22	0	0	0	0	0	0	0	0
Hadar (8)	13	38	13	0	0	38	0	0	0	0	0	0	0	0	0	0
Infantis (7)	0	0	57	29	0	0	0	0	0	0	0	0	0	14	0	0
Montevideo (7)	0	0	29	14	0	14	0	14	29	0	0	0	0	0	0	0
Thompson (6)	0	17	0	33	0	0	17	0	0	0	17	0	17	0	0	0
Agona (5)	0	20	0	0	0	20	0	0	0	0	0	20	0	40	0	0
Litchfield (5)	0	0	0	0	100	0	0	0	0	0	0	0	0	0	0	0
Muenchen (5)	0	20	0	0	40	0	0	40	0	0	0	0	0	0	0	0
Anatum (4)	0	0	25	25	25	0	0	0	0	0	0	0	0	0	0	25
Berta (4)	0	0	25	25	25	0	25	0	0	0	0	0	0	0	0	0
I 4,[5],12:i:- (4)	25	75	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Mbandaka (4)	0	0	25	0	0	0	0	75	0	0	0	0	0	0	0	0
Oranienburg (4)	0	25	0	25	50	0	0	0	0	0	0	0	0	0	0	0
Poona (4)	0	0	0	0	100	0	0	0	0	0	0	0	0	0	0	0
Uganda (4)	0	0	100	0	0	0	0	0	0	0	0	0	0	0	0	0
Senftenberg (3)	0	0	0	33	33	0	0	0	0	0	33	0	0	0	0	0
Weltevreden (3)	0	0	33	0	0	0	0	0	0	67	0	0	0	0	0	0
Other (31)	3	13	16	13	6	6	6	10	3	10	3	6	3	0	0	0
Total (403)	112	64	37	33	33	28	21	19	16	11	10	6	5	3	3	2

*veg, vegetables.

†Crustaceans, fish, and mollusks.

‡Duck (serotype Anatum) and fungus (serotype Typhimurium).

Main Article

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Table 1 - Outbreak-associated Salmonella enterica Serotypes and Food Commodities, United St...

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Table 2 - Outbreak-associated Salmonella enterica Serotypes and Food Commodities, United St...

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Figure

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Volume 19, Number 8-August 2013

Research

Outbreak-associated Salmonella enterica Serotypes and Food Commodities, United States, 1998-2008

Brendan R. Jacksone, Patricia M. Griffin, Dana Cole, Kelly A. Walsh, and Shua J. Chai Author affiliations: Centers for Disease Control and Prevention, Atlanta, Georgia, USA

Main Article

Table 2

Percentage of food commodity-associated outbreaks caused by specific Salmonella enterica serotypes for 403 outbreaks, Foodborne Disease Outbreak Surveillance System, United States, 1998-2008*

Commodity (no.												Seroty	pe											
outbreaks)	Enteritidis	Typhimurium	Newport	Heidelberg	Braenderup	Javiana	Saintpaul	Hadar	Infantis	Montevideo	Thompson	Agona	Litchfield	Muenchen	Anatum	Berta	I 4,[5],12:i:-	Mbandaka	Oranienburg	Poona	Uganda	Senftenberg	Weltevrede	n Other
Eggs (112)	83	4	0	9	1	0	1	1	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	1
Chicken (64)	28	23	8	13	5	2	0	5	0	0	2	2	0	2	0	0	5	0	2	0	0	0	0	6
Pork (37)	5	22	11	0	3	5	0	3	11	5	0	0	0	0	3	3	0	3	0	0	11	0	3	14
Beef (33)	18	18	18	3	0	0	3	0	6	3	6	0	0	0	3	3	0	0	3	0	0	3	0	12
Fruit (33)	12	0	18	3	0	9	3	0	0	0	0	0	15	6	3	3	0	0	6	12	0	3	0	6
Turkey (28)	25	14	11	14	0	0	11	11	0	4	0	4	0	0	0	0	0	0	0	0	0	0	0	7
Vine-stalk veg. (21)	10	14	29	0	14	10	5	0	0	0	5	0	0	0	0	5	0	0	0	0	0	0	0	10
Sprouts (19)	21	11	0	0	11	0	11	0	0	5	0	0	0	11	0	0	0	16	0	0	0	0	0	16
Dairy (16)	0	56	25	0	0	0	0	0	0	13	0	0	0	0	0	0	0	0	0	0	0	0	0	6
Aquatic animals† (11)	45	9	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	18	27
Leafy veg. (10)	10	10	30	0	0	20	0	0	0	0	10	0	0	0	0	0	0	0	0	0	0	10	0	10
Game (6)	0	33	17	0	0	0	0	0	0	0	0	17	0	0	0	0	0	0	0	0	0	0	0	33
Nuts (5)	40	20	0	0	0	0	0	0	0	0	20	0	0	0	0	0	0	0	0	0	0	0	0	20
Grains, beans (3)	0	0	0	0	0	0	0	0	33	0	0	67	0	0	0	0	0	0	0	0	0	0	0	0
Root veg. (3)	0	33	67	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Other‡ (2)	0	50	0	0	0	0	0	0	0	0	0	0	0	0	50	0	0	0	0	0	0	0	0	0
Total (403)	144	58	40	24	10	10	9	8	7	7	6	5	5	5	4	4	4	4	4	4	4	3	3	31

*veg., vegetables.

†Crustaceans, fish, and mollusks.

‡Duck (serotype Anatum) and fungus (serotype Typhimurium).

Main Article

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Foodborne Illness Outbreaks at Retail Establishments — National Environmental Assessment Reporting System, 16 State and Local Health Departments, 2014–2016



U.S. Department of Health and Human Services Centers for Disease Control and Prevention

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Foodborne Illness Outbreaks at Retail Establishments — National Environmental Assessment Reporting System, 16 State and Local Health Departments, 2014–2016

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Abstract

Problem/Condition: State and local public health departments report hundreds of foodborne illness outbreaks each year to CDC and are primarily responsible for investigations of these outbreaks. Typically, investigations involve epidemiology, laboratory, and environmental health components. Health departments voluntarily report epidemiologic and laboratory data from their foodborne illness outbreak investigations to CDC through the Foodborne Disease Outbreak Surveillance System (FDOSS); however, minimal environmental health data from outbreak investigations are reported to FDOSS.

Period Covered: 2014–2016.

Description of System: In 2014, CDC launched the National Environmental Assessment Reporting System (NEARS) to complement FDOSS surveillance and to use these data to enhance prevention efforts. State and local health departments voluntarily report data from their foodborne illness outbreak investigations of retail food establishments. These data include characteristics of foodborne illness outbreaks (e.g., agent), characteristics of establishments with outbreaks (e.g., number of meals served daily), food safety policies and practices of these establishments (e.g., glove use policies), and characteristics of outbreak investigations (e.g., timeliness of investigation activities). NEARS is the only available data source that includes characteristics of retail establishments with foodborne illness outbreaks.

Results: During 2014–2016, a total of 16 state and local public health departments reported data to NEARS on 404 foodborne illness outbreaks at retail establishments. The majority of outbreaks with a suspected or confirmed agent were caused by norovirus (61.1%). The majority of outbreaks with identified contributing factors had at least one factor associated with food contamination by a worker who was ill or infectious (58.6%). Almost half (47.4%) of establishments with outbreaks had a written policy excluding ill workers from handling food or working. Approximately one third (27.7%) had a written disposable glove use policy. Paid sick leave was available for at least one worker in 38.3% of establishments. For most establishments with outbreaks (68.7%), environmental health investigators initiated their component of the investigation soon after learning about the outbreak (i.e., the same day) and completed their component in one or two visits to the establishment (75.0%). However, in certain instances, contacting the establishment and completing the environmental health component of the investigation occurred much later (>8 days).

Interpretation: Most outbreaks reported to NEARS were caused by norovirus, and contamination of food by workers who were ill or infectious contributed to more than half of outbreaks with contributing factors; these findings are consistent with findings from other national outbreak data sets and highlight the role of workers in foodborne illness outbreaks. The relative lack of written policies for ill workers and glove use and paid sick leave for workers in establishments with outbreaks indicates gaps in food safety practices that might have a role in outbreak prevention. The environmental health component of the investigation for most outbreaks was initiated quickly, yet the longer initiation timeframe for certain outbreaks suggests the need for improvement.

Public Health Action: Retail establishments can reduce viral foodborne illness outbreaks by protecting food from contamination

Corresponding author: Laura Brown, Division of Environmental Health Science and Practice, National Center for Environmental Health, CDC. Telephone: 770-488-4332; E-mail: lrg0@cdc.gov. through proper hand hygiene and excluding workers who are ill or infectious from working. NEARS data can help prioritize training and interventions for state and local food safety programs and the retail food establishment industry by identifying gaps in food safety policies and practices and types of establishments vulnerable to outbreaks. Improvement of certain outbreak investigation practices (e.g., delayed initiation of environmental health investigations) can accelerate identification of the agent and implementation of interventions. Future analysis comparing establishments with and without outbreaks will contribute knowledge about how establishments' characteristics and food safety policies and practices relate to foodborne illness outbreaks and provide information to develop effective prevention approaches.

Introduction

Public health departments report hundreds of outbreaks each year to CDC. During 2009–2015, state, local, and territorial health departments reported 5,760 foodborne illness outbreaks to CDC (1). Most of these outbreaks occurred in retail food establishments (1).

State and local public health departments are typically responsible for regulating and ensuring food safety in retail food establishments. They do this primarily through inspecting establishments to ensure they comply with their jurisdictions' food safety regulations. The U.S. Food and Drug Administration (FDA) Food Code is the basis of most jurisdictions' food safety regulations. The FDA Food Code is a model set of science-based, comprehensive food safety regulations intended to reduce foodborne illness risk in retail food establishments (2). For example, the Food Code includes guidelines that

- limit opportunities for food workers to contaminate food, such as prohibiting workers who are ill or infectious from working with food and prohibiting workers from handling ready-to-eat food (i.e., foods that need no further preparation) with their bare hands (e.g., through glove use); and
- require kitchen managers to be certified in food safety (i.e., pass a food safety knowledge test administered by an accredited program).

State and local public health departments also investigate foodborne illness outbreaks. Data from these investigations provide insights into the epidemiology of foodborne illness, such as identifying the pathogens and foods that lead to illness (1). This information can be used to help prevent foodborne illness outbreaks and sporadic foodborne illnesses that can have the same epidemiologic profile as outbreaks.

State and local public health departments provide epidemiologic and laboratory data from their investigations to CDC through the Foodborne Disease Outbreak Surveillance System (FDOSS) (3). Typically, epidemiology or communicable disease control programs within health departments collect and report these data, which include the etiologic agent; food; setting; and number of illnesses, hospitalizations, and deaths associated with an outbreak. These data have led to discoveries of new and emerging foodborne illness agents and specific agent-food pairs (4).

Environmental health programs within state and local health departments also are involved in investigations of foodborne illness outbreaks. The environmental health component of the investigation, or environmental assessment, describes how the environment contributed to the introduction or transmission of agents that caused illness. During these assessments, environmental health investigators typically interview the manager of the establishment with an outbreak about characteristics such as food preparation policies and practices that might have contributed to the outbreak. Environmental health investigators also review the processes used in preparing food items suspected in the outbreak and observe workers' food preparation practices. After all investigation activities are completed, the epidemiologic, laboratory, and environmental health information is reviewed to determine the outbreak contributing factors, which are the conditions that enabled or amplified a foodborne illness outbreak. These factors can contribute to contamination of food with foodborne illness agents, proliferation of microbial agents in food, or survival of foodborne illness agents in food after a process that should have eliminated or reduced them.

Although FDOSS captures data on foodborne illness outbreak contributing factors, the system does not capture most other environmental assessment data and is not limited to retail food establishments. These data about the context in which outbreaks occur are important to outbreak prevention. For example, data on worker practices associated with outbreaks can provide information about interventions that encourage retail food establishments to improve worker practices. Because of the importance of these environmental assessment data, CDC developed the National Environmental Assessment Reporting System (NEARS) to capture data from health departments' environmental assessments of outbreaks (5). NEARS was designed to be a complementary surveillance system to FDOSS. The Environmental Health Specialists Network (EHS-Net), a CDC-funded network of environmental health specialists and epidemiologists from CDC, FDA, the U.S. Department of Agriculture, and multiple state and local health departments (6), helped develop NEARS.

This report summarizes selected data reported to NEARS for foodborne illness outbreaks that occurred during 2014–2016. The data describe the outbreaks, the establishments where the outbreaks occurred, including their food safety policies, and the outbreak investigations. State and local public health departments responsible for ensuring food safety and investigating foodborne illness outbreaks can use these data to help identify gaps in their outbreak investigation practices and in retail food establishment policies.

Methods

Description of the System and Case Definition

The majority of foodborne illness outbreaks occur in retail food establishments (i.e., places that prepare and serve food to consumers) (1). In 2014, NEARS was launched to collect data on outbreaks associated with such establishments (4). CDC defines a foodborne illness outbreak as an incident in which two or more persons experience a similar illness resulting from the ingestion of a common food (7); most state and local health departments have a similar definition. Outbreak agents were classified as confirmed if they were laboratory confirmed according to CDC laboratory and clinical guidelines (7); otherwise they were classified as suspected. During 2014–2016, a total of 16 state and local health departments (California; Coconino County, Arizona; Connecticut; Davis County, Utah; Fairfax County, Virginia; Harris County, Texas; Michigan; Minnesota; New York City; New York State; Rhode Island; South Carolina; Southern Nevada Health District; Tennessee; Washington; and Wisconsin) reported environmental assessment data to NEARS from at least one foodborne illness outbreak occurring in a retail food establishment. Supplementary data on foodborne illness outbreaks reported to NEARS (https://stacks.cdc.gov/view/cdc/61382) and retail establishments with outbreaks (https://stacks.cdc.gov/view/ cdc/61383) are available.

NEARS complements FDOSS by collecting data from state and local foodborne illness outbreak investigations that are not collected in FDOSS. Although some data points are collected in both systems (e.g., outbreak agent), this redundancy is designed to ensure that outbreaks can be matched accurately across the two systems.

Data Sources, Collection, and Variables

NEARS data sources include environmental health investigators and their epidemiology and laboratory counterparts, as well as interviews with establishment managers (Box 1). After each foodborne illness outbreak investigation in a retail food establishment is completed, participating health departments voluntarily report their environmental health investigation data to CDC through the NEARS online data management system on CDC's website. Environmental health investigators' epidemiologic and laboratory counterparts provide the data on outbreak characteristics. The environmental assessments provide data on characteristics and policies of establishments with outbreaks, primarily through interviews with managers. The environmental health investigators determine outbreak investigation characteristics. Not all data points are collected during all investigations; thus, denominators vary throughout the results.

Data are collected and presented on four sets of variables: characteristics of foodborne illness outbreaks, characteristics of establishments with outbreaks, policies of establishments with outbreaks, and characteristics of investigations.

- **Outbreak characteristics**. Characteristics include the outbreak agent and contributing factors. FDA and CDC have identified 32 outbreak contributing factors, divided into three groups (*8*):
 - contamination of food with a foodborne illness agent;
 - proliferation or growth of microbial agents in food (proliferation can mean an increase in the number of bacteria, the production of toxins, or both); and
 - survival of foodborne illness agents after a process, such as cooking, that should have eliminated or reduced them.
- Outbreak establishment characteristics. Characteristics that have been hypothesized or found to be associated with retail food establishment food safety. These include ownership (independent or chain [shares name and operation with at least one other establishment]) and number of meals served daily (9–12).
- Outbreak establishment policies. Policies recommended by FDA in the Food Code to reduce foodborne illness risk. These include limiting opportunities for food workers to contaminate food, such as prohibiting workers who are ill or infectious from working with food and prohibiting workers from handling ready-to-eat food (i.e., foods that need no further preparation) with their bare hands (e.g., through glove use), and requiring kitchen managers to be certified in food safety (i.e., pass a food safety knowledge test administered by an accredited program). Data also are included on the availability of paid sick leave for ill workers. Although the Food Code specifically does not recommend paid sick leave, the food service industry could explore this policy as a potential method to help keep ill workers from working (*13*).
- **Outbreak investigation characteristics.** Characteristics that have been hypothesized or found to be associated with investigation effectiveness, such as the timeliness of outbreak environmental assessments (*14,15*).

Data collected	Source
Outbreak characteristics	
Primary agent identification — confirmed (laboratory-confirmed by laboratory and clinical guidelines) or suspected (not confirmed by the guidelines) (In 2014, these data were obtained from the Foodborne Disease Outbreak Surveillance System; during 2015–2016, environmental health investigators reported these data to the National Environmental Assessment Reporting System)	Epidemiology and laboratory investigation counterparts
Contributing factor identification (factors that contribute to the contamination, proliferation, and survival of foodborne illness agents on food)	Investigation team determination
Outbreak also reported to the Foodborne Disease Outbreak Surveillance System	Epidemiology and laboratory investigation counterparts
Outbreak establishment characteristics	
Ownership — independent or chain (establishment shares name and operations with at least one other establishment)	Establishment manager interview
Establishment type — restaurant (fixed establishment that prepares and serves food to customers) or other (e.g., institutions, mobile food units, temporary food stands, or restaurants in supermarkets, etc.)	Environmental health investigator determination
Average number of meals served daily	Establishment manager interview
 Most complex food preparation process Complex — food item requires a pathogen kill step (a process, such as cooking or freezing, that reduces or eliminates pathogens) and holding beyond same-day service, or a kill step and some combination of holding, cooling, reheating, and freezing Complex-serve — food item is prepared for same-day service; at least one involves a kill step such as cooking Prep-serve — food item is prepared and served without a kill step 	Environmental health investigator determination
Menu type (e.g., American or Indian)	Environmental health investigator determination
Number of critical violations on previous inspection (i.e., violations of regulations that help eliminate or reduce hazards associated with foodborne illness; also called priority or priority foundation items)	Environmental health investigator determination

BOX 1. Data sources for characteristics of foodborne illness outbreaks, characteristics and policies of retail establishments with outbreaks, and characteristics of investigations — National Environmental Assessment Reporting System, 2014–2016

Box continued on next page.

Data Analysis

CDC calculated descriptive statistics on four sets of NEARS variables. These were characteristics of foodborne illness outbreaks, characteristics of establishments with outbreaks, policies of establishments with outbreaks, and characteristics of investigations.

Results

During 2014–2016, state and local health departments reported 404 foodborne illness outbreaks in retail establishments to NEARS. Of these, 111 (27.5%) occurred in 2014, 113 (28.0%) in 2015, and 180 (44.6%) in 2016. A total of 384 (95.0%) of these outbreaks occurred in one location, and

BOX 1. (Continued) Data sources for characteristics of foodborne illness outbreaks, characteristics and policies of retail establishments with
outbreaks, and characteristics of investigations — National Environmental Assessment Reporting System, 2014–2016

Data collected	Source
Outbreak establishment policies	
Policy requiring workers to tell their manager when they are ill	Establishment manager interview
Policy restricting or excluding ill workers from working	Establishment manager interview
Paid sick leave available for at least one worker	Establishment manager interview
Disposable glove use policy	Establishment manager interview
Disposable glove use policy requiring food workers to wear gloves at all times when working in the kitchen, when handling ready-to-eat food, and when they have cuts or other skin injuries	Establishment manager interview
Kitchen manager food safety certification requirement	Establishment manager interview
Outbreak investigation characteristics	
Number of visits to the establishment with an outbreak to complete environmental assessment	Environmental health investigator determination
Number of days between identification of establishment for an environmental assessment and first contact with the establishment, observation, and manager interview	Environmental health investigator determination
Number of critical violations on previous inspection (i.e., violations of regulations that help eliminate or reduce hazards associated with foodborne illness; also called priority or priority foundation items)	Environmental health investigator determination

20 (5.0%) occurred in multiple locations. Data were reported to NEARS on 415 establishments with outbreaks. Most (83.7%, 338 of 404) outbreaks reported to NEARS also were reported to FDOSS. This percentage is expected to increase in the future because of updates to the reporting system and improvements in linking processes. A supplementary summary report is available (https://www.cdc.gov/nceh/ehs/nears/ outbreak-investigations-restaurants-2014-16.html).

Outbreak Characteristics

Investigations identified an agent in 311 (77.0%) outbreaks (Table 1). Of these agents, 31.8% were suspected and 68.2% were confirmed. Most identified agents were viral (61.7%), followed by bacterial (34.4%) and toxic, chemical, or other (3.9%). Overall, norovirus was the most common agent, accounting for 61.1% of outbreaks where an agent was identified. The second most common agent was *Salmonella*, accounting for 16.1% of outbreaks with an identified agent.

Investigators identified at least one contributing factor in 251 (62.1%) outbreaks. Outbreaks can have more than one contributing factor, and 455 were identified. Of the 251 outbreaks with an identified contributing factor, 214 (85.3%) had at least one contamination factor, 69 (27.5%) had at least one proliferation factor, and 44 (17.5%) had at least one survival factor (Table 2). The top three contributing factors were related to food contamination by an ill worker; 147 (58.6%) outbreaks with an identified contributing factor had at least one of these factors.

All three types of contributing factors (i.e., contamination of food with agents, proliferation of agents, and survival of agents) were represented among the top 10 contributing factors to foodborne illness outbreaks (Box 2). The most common contributing factor (27.9%) was bare-hand contact by a food worker suspected to have an infectious illness, followed by contamination through a method other than hand contact by a food worker suspected to have an infectious illness (23.1%) and glove-hand contact by a food worker suspected to have an infectious illness (15.5%) (Table 2). The most common proliferation and survival contributing factors were improper or slow cooling of hot food (10.0%) and insufficient time or temperature during cooking or heat processing (10.8%).

Outbreak Establishment Characteristics

Most establishments with outbreaks were independently owned (72.9%, 237 of 325), were restaurants (80.2%, 333 of 415), and served complex food items (i.e., a food item required a kill step, which is a process, such as cooking, that reduces or eliminates foodborne illness pathogens, and other food preparation processes, such as cooling and reheating) (87.2%, 362 of 415) (Table 3). More than half of establishments with outbreaks (54.6%, 161 of 295) served ≤200 meals daily (upper range: 7,500). The most common menu type was American (nonethnic) (55.9%, 232 of 415), and most (65.8%) establishments with outbreaks received one or more critical violations on their last routine inspection before the outbreak.

Outbreak Establishment Policies

More than half of establishments with outbreaks (56.3%, 179 of 318) had a written policy and 36.2% (115) had a verbal policy requiring food workers to notify their manager when they were ill (Table 4). About half (47.4%, 144 of 304) of establishments had a written policy and 39.1% had a verbal policy that prevented ill workers from handling food (i.e., restriction) or prevented ill workers from working (i.e., exclusion). In 118 of 308 (38.3%) establishments, paid sick leave was available for at least one food worker. The majority of establishments with outbreaks (62.3%, 198 of 318) had a verbal policy concerning disposable glove use; an additional 27.7% had a written disposable glove use policy. Glove use policy requirements were varied. Most establishments required food workers to wear gloves when handling ready-to-eat foods (97.2%, 278 of 286) and when they had cuts or other skin injuries (98.6%, 278 of 282), and half (49.7%, 142 of 286) required food workers to wear gloves at all times when working in the kitchen. In 243 of 314 (77.4%) establishments, kitchen managers were required to have a food safety certification.

Outbreak Investigation Characteristics

Three fourths (74.6%, 306 of 410) of environmental assessments were completed in one or two visits to the establishment; the remaining assessments were completed in three or more visits (Table 5). Investigators contacted most (68.7%, 285 of 415) of the establishments with outbreaks the same day they were identified for an environmental assessment. The mode of contact varied (e.g., telephone, e-mail, or in person). For the remaining establishments, contact occurred 1–2 days (23.4%, 97 of 415) and ≥3 days (7.9%, 9 of 415) after identification. Half (49.6%, 175 of 353) of observations were conducted the same day the establishment was identified for an environmental assessment. The remaining observations

BOX 2. Top 10 contributing factors to foodborne illness outbreaks,* by type — National Environmental Assessment Reporting System, 2014–2016

Contamination of food with a foodborne illness agent Bare-hand contact by a food handler, worker, or preparer with a suspected infectious illness

Other mode of contamination (excluding crosscontamination) by a food handler, worker, or preparer with a suspected infectious illness

Glove-hand contact by a food handler, worker, or preparer with a suspected infectious illness

Cross-contamination of ingredients

Other source of contamination

Contaminated raw product — food was intended to be consumed raw or undercooked or underprocessed

Contaminated raw product — food was intended to be consumed after a kill step

Proliferation or growth of microbial agents in food (increase in number of bacteria or the production of toxins)

Improper or slow cooling

No attempt was made to control the temperature of implicated food or the length of time food was out of temperature control

Survival of foodborne illness agents after a process, such as cooking, that should have eliminated or reduced them

Insufficient time, temperature, or both during cooking or heat processing

* N = 251 outbreaks for which data were known; some outbreaks had more than one identified contributing factor.

were conducted 1–2 days after identification (28.0%) and ≥ 3 days after identification (22.4%). One fourth (25.8%, 82 of 318) of interviews with managers were conducted the same day the establishment was identified for an assessment. The remaining interviews were conducted 1–2 days (19.5%), 3–7 days (12.9%), and ≥ 8 days (41.8%) after identification.

Discussion

Approximately 60% of foodborne illness outbreaks in retail food establishments reported to NEARS were caused by norovirus, and contamination of food by workers who were ill

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or infectious contributed to more than half of outbreaks with contributing factors. These findings are similar to national outbreak data reported to FDOSS; a recent analysis found that approximately half of restaurant-associated foodborne illness outbreaks were caused by norovirus, and that workers who were ill or infectious contributed to about half of restaurant-associated outbreaks (16). NEARS and FDOSS data both highlight the role of workers in norovirus outbreaks (2,15,16). The data also indicate the need for continued focus on reducing viral foodborne illness outbreaks by protecting food from worker contamination through proper hand hygiene, including glove use, and preventing workers who are ill or infectious from working (16,17).

NEARS is the only available data source that includes characteristics of retail establishments with foodborne illness outbreaks. Because ill workers are a frequent contributor to outbreaks (1), of particular interest are NEARS data on establishment characteristics that might be related to ill worker behavior, such as requiring gloves and excluding workers who are ill from work. Most establishments with outbreaks had policies requiring food workers to wear gloves when handling ready-to-eat foods and preventing those who are ill from working. The FDA Food Code recommends these policies to protect against outbreaks (2), yet establishments with these policies still had outbreaks. One reason might be that existing policies are not enforced.

This report assessed whether the establishments had these policies but did not assess whether the policies were regulatory requirements in the areas where the establishments were located. If policies are not regulatory requirements, regulatory officials do not assess them in their inspections and establishments do not receive violations for a lack of policies. Lack of regulation might affect policy effectiveness.

Finally, the mode of the policy might have a role in effectiveness; research suggests that written policies are more effective than unwritten ones (11). Written food safety policies might indicate prioritization of food safety or institutionalized policies and practices. Approximately half, or fewer, of the establishments with outbreaks had these policies in writing.

Paid sick leave also might be relevant to outbreaks caused by ill workers; a study found an association between supportive paid sick leave regulations and decreased foodborne illness rates (18). Workers have reported that lack of paid sick leave factors into their decision to work while ill (19). The relative lack of sick leave for workers suggests this might be a risk factor for foodborne illness outbreaks.

Most outbreaks reported to NEARS occurred in establishments that engaged in complex food preparation processes, served American-style food, were independently owned, and received critical violations on their last inspection. These data can contribute to generating hypotheses about the context in which outbreaks occur. For example, the proportion of establishments with outbreaks engaging in complex food preparation processes (87%) is high compared with the proportion of establishments without outbreaks engaging in these processes (approximately 50%) found in other studies (EHS-Net restaurant cooling practices study, unpublished data, CDC, 2009) (20). This difference suggests that outbreaks might occur more often in establishments where complex food preparation occurs. On the other hand, comparisons of establishment ownership indicate that the proportion of independently owned establishments in the NEARS outbreak data set is similar to the proportion of independently owned restaurants nationwide (73% versus 66%) (21), which suggests that independent and chain restaurants might experience outbreaks with similar frequency. Although research comparing establishments with and without outbreaks is necessary to confirm these hypotheses, preliminary comparisons indicate the potential value of NEARS data to facilitate development and testing of hypotheses about the characteristics of outbreaks associated with retail food establishments.

NEARS also provides new data that might identify strengths and weaknesses of investigation practices. For example, for most outbreaks the investigators initiated an environmental assessment within a day of learning about the outbreak, which is a positive indicator because experts recommend initiating environmental assessments as quickly as possible (15). Research also indicates that timely and comprehensive environmental assessments are associated with identifying factors contributing to outbreaks, which is an important goal of outbreak investigations (14). On the other hand, for certain outbreaks, investigators took considerably longer (from 8 days to >14 days) to initiate contact, suggesting a need for improvement in timeliness of environmental assessments. CDC provides free, interactive training on outbreak environmental assessments, a first step for health departments seeking to improve investigation practices (22). The CDC-funded Integrated Food Safety Centers of Excellence also provide free resources for food safety professionals (23).

Limitations

The findings in this report are subject to at least four limitations. First, the findings are determined from data reported by a limited number of state and local health departments. Although these health departments represent geographically diverse areas, the foodborne illness outbreaks reported to NEARS are not representative of all U.S. outbreaks. Second, not all outbreaks are identified, reported, or investigated; therefore, the extent to which the outbreaks reported to NEARS represent all outbreaks that occurred in the reporting areas is unknown. Third, outbreak investigation procedures and practices vary across state and local health departments, possibly resulting in systematic differences in data collection. Finally, the manager interview data might be subject to social desirability bias, in which respondents overreport socially desirable conditions, such as the existence of food safety policies in their establishments.

Future Directions

Most (83.7%) foodborne illness outbreaks reported to NEARS also were reported to FDOSS. Therefore, the data from the two systems can be matched by outbreak to create a comprehensive outbreak data set with epidemiologic, laboratory, and environmental health data. Subsequent analyses of matched data can help guide and develop outbreak prevention efforts. For example, analysis of the relation between establishment policies (environmental health data) and outbreak size (epidemiologic data) can help identify effective policies. Future analyses also might focus on differences between outbreaks that are reported to both NEARS and FDOSS and outbreaks that only are reported to FDOSS. NEARS data also allow comparisons of establishments that have had bacterial outbreaks with those that have had viral outbreaks, which can identify characteristics and policies that might contribute to the likelihood of specific types of outbreaks.

Conclusion

NEARS provides unique data on establishments that have had foodborne illness outbreaks. These data increase knowledge about the environmental context of outbreaks and contribute to generating hypotheses about their causes and prevention. Use of NEARS data to compare characteristics of establishments with and without outbreaks, examine relations between establishments and epidemiologic characteristics, and compare bacterial and viral outbreaks will contribute to understanding the role of these factors in outbreaks. CDC is developing these analyses, and the information gained from them can help public health authorities develop data-based, effective approaches to prevention of foodborne illness outbreaks. Because NEARS data identify gaps in food safety policies and practices and types of establishments vulnerable to outbreaks, the data also can help target training and interventions for state and local food safety programs and the retail food establishment industry. (For example, the data suggest that outbreaks occur more often in establishments using complex food preparation.) Finally, NEARS data can identify gaps in environmental health investigation practices, such as delayed environmental assessments. Identifying these gaps can help investigators target their improvement efforts, which might include increasing communication among environmental health, epidemiologic, and laboratory programs, as well as implementing policies and training to support environmental assessments (24).

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Conflict of Interest

No conflicts of interest were reported.

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	Suspected	Confirmed	Total
Agent	No. (%)*	No. (%)*	No. (%)*
Virus			
Norovirus	66 (21.2)	124 (39.9)	190 (61.1)
Hepatitis A	0 (0.0)	2 (0.6)	2 (0.6)
fotal viral outbreaks	66 (21.2)	126 (40.5)	192 (61.7)
Bacteria			
almonella species	2 (0.6)	48 (15.4)	50 (16.1)
Clostridium perfringens	9 (2.9)	8 (2.6)	17 (5.5)
Campylobacter species	2 (0.6)	9 (2.9)	11 (3.5)
acillus cereus	5 (1.6)	1 (0.3)	6 (1.9)
scherichia coli O157:H7/STEC	0 (0.0)	10 (3.2)	10 (3.2)
taphylococcus aureus	5 (1.6)	2 (0.6)	7 (2.3)
higella species	0 (0.0)	2 (0.6)	2 (0.6)
<i>librio</i> species	1 (0.3)	2 (0.6)	3 (1.0)
isteria monocytogenes	0 (0.0)	1 (0.3)	1 (0.3)
<i>ersinia</i> species	0 (0.0)	0 (0.0)	0 (0.0)
otal bacterial outbreaks	24 (7.7)	83 (26.7)	107 (34.4)
oxin, chemical, and other [†]			
combroid toxin	4 (1.3)	1 (0.3)	5 (1.6)
iguatoxin	1 (0.3)	0 (0.0)	1 (0.3)
hemical	1 (0.3)	0 (0.0)	1 (0.3)
Other	3 (1.0)	2 (0.6)	5 (1.6)
fotal toxin outbreaks	9 (2.9)	3 (1.0)	12 (3.9)
otal outbreaks	99 (31.8)	212 (68.2)	311 (100.0)

TABLE 1. Foodborne illness outbreaks with a suspected or confirmed identified agent — National Environmental Assessment Reporting System, 16 state and local health departments, 2014-2016

Abbreviation: STEC = Shiga toxin-producing *Escherichia coli*. * All numbers are divided by the total number of outbreaks with a suspected or confirmed agent (denominator = 311) to obtain the percentage. Because of rounding, some percentages might not total 100%.

⁺ Toxins produced by bacteria are included in the bacteria category; natural toxins, such as marine and mushroom, are included in the toxin category.

TABLE 2. Factors contributing to foodborne illness outbreaks, by type of factor — National Environmental Assessment Reporting System, 16 state and local health departments, 2014–2016

iontributing factor	No. (%)*
ontamination of food with a foodborne illness agent	
are-hand contact by a food handler, worker, or preparer who was suspected to have an infectious illness (C10)	70 (27.9)
ther mode of contamination (excluding cross-contamination) by a food handler, worker, or preparer who was suspected to have an infectious illness (C12)	58 (23.1)
ove-hand contact by a food handler, worker, or preparer who was suspected to have an infectious illness (C11)	39 (15.5)
oss-contamination of ingredients (does not include ill food workers) (C9)	28 (11.2)
ntaminated raw product — food was intended to be consumed raw or undercooked or underprocessed (C7)	15 (6.0)
her source of contamination (C15)	24 (9.6)
ntaminated raw product — food was intended to be consumed after a kill step (C6)	14 (5.6)
cic substance part of the tissue (e.g., ciguatera) (C1)	5 (2.0)
ds contaminated by nonfood handler, worker, or preparer who was suspected to have an infectious illness (C13)	9 (3.6)
sonous substance accidentally or inadvertently added (C3)	1 (0.4)
ds originating from sources shown to be contaminated or polluted (C8)	4 (1.6)
sonous substance intentionally or deliberately added (C2)	0 (0.0)
dition of excessive quantities of ingredients that are toxic in large amounts (e.g., niacin poisoning in bread) (C4)	0 (0.0)
ic container (e.g., galvanized containers with acid foods) (C5)	0 (0.0)
rage in contaminated environment (C14)	13 (5.2)
al contamination factors	280 (100.0)
liferation or growth of microbial agents in food (increase in number of bacteria or the production of toxins)	
proper or slow cooling (P8)	25 (10.0)
attempt to control the temperature of implicated food or the length of time food was out of temperature control (during food service or isplay of food) (P2)	23 (9.2)
proper cold holding due to malfunctioning refrigeration equipment (P4)	13 (5.2)
roper hot holding due to an improper procedure or protocol (P7)	14 (5.6)
proper cold holding due to an improper procedure or protocol (P5)	18 (7.2)
d preparation practices that support proliferation of pathogens (during food preparation) (P1)	18 (7.2)
proper hot holding due to malfunctioning equipment (P6)	5 (2.0)
dequate modified atmosphere packaging (e.g., vacuum-packed fish) (P10)	2 (0.8)
roper adherence to approved plan for using time as a public health control (P3)	1 (0.4)
longed cold storage (P9)	0 (0.0)
dequate processing (e.g., acidification, water activity, or fermentation) (P11)	1 (0.4)
er situations that promoted or allowed microbial growth or toxin production (P12)	2 (0.8)
al proliferation factors	122 (100.0)
rvival of foodborne illness agents after a process, such as cooking, that should have eliminated or reduced them	
ufficient time, temperature, or both during cooking or heat processing (e.g., roasted poultry, canned foods, or pasteurization) (S1)	27 (10.8)
ufficient time, temperature, or both during reheating (S2)	12 (4.8)
ifficient time, temperature control, or both during freezing (S3)	0 (0.0)
ufficient or improper use of chemical processes designed for pathogen destruction (S4)	10 (4.0)
ner process failures that permit agent survival (S5)	4 (1.6)
tal survival factors	53 (100.0)
tal contributing factors	455 (100.0)

Source: CDC [Internet]. NORS guidance for contributing factors (CF) in foodborne outbreak reports. Atlanta, GA: US Department of Health and Human Services, CDC; 2018. https://www.cdc.gov/nors/downloads/appendix-d.pdf

Abbreviations: C = contamination; P = proliferation; S = survival. * Denominator = 251; some outbreaks had more than one identified contributing factor, so percentages sum to more than 100%. These designations (e.g., C1, P6, or S2) are used by outbreak investigators to refer to the type of contributing factor (e.g., contamination, proliferation, or survival) and its numerical position on the contributing factor list.

Establishment characteristic	No. (%)*
Ownership	
Independent Chain	237 (72.9)
Chain	88 (27.1)
Total	325 (100.0)
Establishment type	
Restaurant	333 (80.2)
Other	82 (19.8)
Total	415 (100.0)
Most complex food preparation process	
Complex — food item requires a pathogen kill step (a process, such as cooking or freezing, that reduces or eliminates pathogens) and holding beyond same-day service, or a kill step and some combination of holding, cooling, reheating, and freezing	362 (87.2)
Cook-serve — food item is prepared for same-day service; at least one involves a kill step such as cooking	39 (9.4)
Prep-serve — food item is prepared and served without a kill step	14 (3.4)
Total	415 (100.0)
Number of meals served daily	
≤100	88 (29.8)
101–200	73 (24.7)
201–300	48 (16.3)
301-400	29 (9.8)
401-500	14 (4.8)
501-7,500	43 (14.6)
Total	295 (100.0)
Menu	
American	232 (55.9)
Other (e.g., Mediterranean, Indian, or Spanish)	72 (17.3)
Mexican	38 (9.2)
Italian	30 (7.2)
Chinese	23 (5.5)
Japanese Thai	16 (3.9)
Total	4 (1.0) 415 (100.0)
Critical violations on last inspection	1 4 2 (2 4 2)
None	142 (34.2)
≥1 	273 (65.8)
Total	415 (100.0)

TABLE 3. Characteristics of retail establishments with foodborne illness outbreaks — National Environmental Assessment Reporting System, 16 state and local health departments, 2014–2016

* Denominators vary because of missing data. Because of rounding, some percentages might not total 100%.

Establishment policy	No. (%)*
Policy requiring food workers to tell their manage	ger when they are ill
Yes	115 (36.2)
Yes, and it's written	179 (56.3)
No	24 (7.5)
Total	318 (100.0)
Policy restricting or excluding ill workers from w	vorking
Yes	119 (39.1)
Yes, and it's written	144 (47.4)
No	41 (13.5)
Total	304 (100.0)
Paid sick leave available for at least one worker	
Yes	118 (38.3)
No	190 (61.7)
Total	308 (100.0)
Disposable glove use policy	
Yes	198 (62.3)
Yes, and it's written	88 (27.7)
No	32 (10.1)
Total	318 (100.0)
Glove use policy requiring food workers to wear ready-to-eat food [†]	r gloves when handling
Yes	278 (97.2)
No	8 (2.8)
Total	286 (100.0)
Glove use policy requiring food workers to wear cuts or other skin injuries [†]	r gloves when they have
Yes	278 (98.6)
No	4 (1.4)
Total	282 (100.0)
Glove use policy requiring food workers to wear working in the kitchen [†]	r gloves at all times when
Yes	142 (49.7)
No	144 (50.3)
Total	286 (100.0)
Kitchen manager food safety certification requi	rement
Yes	243 (77.4)
No	71 (22.6)
Total	314 (100.0)

TABLE 4. Policies of retail establishments with foodborne illness outbreaks - National Environmental Assessment Reporting System, 16 state and local health departments, 2014–2016 _

* Denominators vary because of missing data and interview skip patterns. Because of rounding, some percentages might not total 100%. † Only asked if the manager said they have a glove use policy.

TABLE 5. Characteristics of foodborne illness outbreak investigations — National Environmental Assessment Reporting System, 16 state and local health departments, 2014–2016

Investigation characteristic	No. (%)*
No. of visits needed to complete the enviro	onmental assessment
1	202 (49.9)
2	104 (25.1)
3	51 (12.3)
4	26 (6.3)
≥5 (up to 30 visits)	27 (6.5)
Total	410 (100.0)
Time interval between establishment iden assessment and first contact with the esta	
Same day [†]	285 (68.7)
1–2 days	97 (23.4)
3–7 days	24 (5.8)
8–14 days	6 (1.4)
>14 days (up to 36 days)	3 (0.7)
Total	415 (100.0)
Time interval between establishment iden assessment and establishment observation	
Same day	175 (49.6)
1–2 days	99 (28.0)
3–7 days	43 (12.2)
8–14 days	18 (5.1)
>14 days (up to 103 days)	18 (5.1)
Total	353 (100.0)
Time interval between establishment iden assessment and establishment manager	
Same day [†]	82 (25.8)
1–2 days	62 (19.5)
3–7 days	41 (12.9)
8–14 days	27 (8.5)
15–21 days	23 (7.2)
22–28 days	18 (5.7)
29–35 days	18 (5.7)
>35 days (up to 389 days)	47 (14.8)

Total

* Denominators vary because of missing data. Because of rounding, some percentages might not total 100%.

318 (100.0)

⁺ Includes one situation in which preliminary information led investigators to contact the establishment or conduct a manager interview before the establishment officially was identified for an environmental assessment.

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Surveillance for Foodborne Disease Outbreaks — United States, 2009–2015



U.S. Department of Health and Human Services Centers for Disease Control and Prevention

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Surveillance for Foodborne Disease Outbreaks — United States, 2009–2015

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Abstract

Problem/Condition: Known foodborne disease agents are estimated to cause approximately 9.4 million illnesses each year in the United States. Although only a small subset of illnesses are associated with recognized outbreaks, data from outbreak investigations provide insight into the foods and pathogens that cause illnesses and the settings and conditions in which they occur.

Reporting Period: 2009–2015

Description of System: The Foodborne Disease Outbreak Surveillance System (FDOSS) collects data on foodborne disease outbreaks, which are defined as the occurrence of two or more cases of a similar illness resulting from the ingestion of a common food. Since the early 1960s, foodborne outbreaks have been reported voluntarily to CDC by state, local, and territorial health departments using a standard form. Beginning in 2009, FDOSS reporting was made through the National Outbreak Reporting System, a web-based platform launched that year.

Results: During 2009–2015, FDOSS received reports of 5,760 outbreaks that resulted in 100,939 illnesses, 5,699 hospitalizations, and 145 deaths. All 50 states, the District of Columbia, Puerto Rico, and CDC reported outbreaks. Among 2,953 outbreaks with a single confirmed etiology, norovirus was the most common cause of outbreaks (1,130 outbreaks [38%]) and outbreak-associated illnesses (27,623 illnesses [41%]), followed by *Salmonella* with 896 outbreaks (30%) and 23,662 illnesses (35%). Outbreaks caused by *Listeria, Salmonella*, and Shiga toxin-producing *Escherichia coli* (STEC) were responsible for 82% of all hospitalizations and 82% of deaths reported. Among 1,281 outbreaks in which the food reported could be classified into a single food category, fish were the most commonly implicated category (222 outbreaks [17%]), followed by dairy (136 [11%]) and chicken (123 [10%]). The food categories responsible for the most outbreak-associated illnesses were chicken (3,114 illnesses [12%]), pork (2,670 [10%]), and seeded vegetables (2,572 [10%]). Multistate outbreaks comprised only 3% of all outbreaks reported but accounted for 11% of illnesses, 34% of hospitalizations, and 54% of deaths.

Interpretation: Foodborne disease outbreaks provide information about the pathogens and foods responsible for illness. Norovirus remains the leading cause of foodborne disease outbreaks, highlighting the continued need for food safety improvements targeting worker health and hygiene in food service settings. Outbreaks caused by *Listeria, Salmonella*, and STEC are important targets for public health intervention efforts, and improving the safety of chicken, pork, and seeded vegetables should be a priority.

Public Health Action: The causes of foodborne illness should continue to be tracked and analyzed to inform disease prevention policies and initiatives. Strengthening the capacity of state and local health departments to investigate and report outbreaks will assist with these efforts through identification of the foods, etiologies, and settings linked to these outbreaks.

Introduction

Approximately 800 foodborne disease outbreaks are reported in the United States each year, accounting for approximately 15,000 illnesses, 800 hospitalizations, and 20 deaths (1). Outbreak-associated foodborne illnesses are

Corresponding author: Daniel Dewey-Mattia, National Center for Emerging and Zoonotic Infectious Diseases, CDC. Telephone: 404-639-5164; E-mail: vvp6@cdc.gov. only a small subset of the estimated 9.4 million foodborne illnesses from known pathogens that occur annually in the United States (2). However, the food sources and exposure settings for illnesses that are not part of outbreaks can be determined only rarely. Outbreak investigations, on the other hand, often link etiologies with specific foods, allowing public health officials, regulatory agencies, and the food industry to investigate how foods become contaminated. Foodborne outbreak data also can be used to identify emerging food safety issues and to assess whether programs to prevent illnesses from particular foods are effective.

This report summarizes foodborne disease outbreaks reported in the United States in which the first illness occurred between January 1, 2009, and December 31, 2015. The report highlights a few large outbreaks as well as novel foods and food-pathogen pairs responsible for outbreaks during the reporting period.

Methods

A foodborne disease outbreak is defined as two or more cases of a similar illness resulting from ingestion of a common food (3). When exposure to a contaminated food occurs in a single state, the outbreak is classified as a single-state outbreak; when exposure occurs in two or more states, the outbreak is classified as a multistate outbreak. Local, state, and territorial health departments voluntarily report foodborne outbreaks to CDC through the Foodborne Disease Outbreak Surveillance System (FDOSS) (https://www.cdc.gov/fdoss/). CDC staff also report multistate foodborne disease outbreaks to FDOSS; these outbreaks are identified by PulseNet, the national molecular subtyping network (4). Initially a paperbased surveillance system, FDOSS reporting became electronic in 1998. In 2009, FDOSS was incorporated into the newly created National Outbreak Reporting System, a web-based platform that also includes reports of outbreaks attributable to waterborne, person-to-person, animal contact, environmental, and indeterminate or unknown modes of transmission.

Etiologies reported to FDOSS include bacterial, parasitic, and viral pathogens as well as chemicals and toxins. Outbreak etiologies are classified as unknown, suspected, or confirmed. Specific criteria (i.e., laboratory testing and clinical syndrome) are used to classify etiologies of outbreaks as suspected or confirmed (5). An outbreak is categorized as a multiple etiology outbreak if more than one agent is reported.

Foods and ingredients are identified as outbreak sources (i.e., implicated) using one or more of the following types of evidence: epidemiologic, laboratory, traceback, environmental assessment, or other data. Some outbreak investigations do not identify a source and in these instances the food is reported as unknown. CDC categorizes foods implicated in outbreak investigations on the basis of a hierarchical scheme (6). One of 24 food categories (e.g., mollusks) is assigned if a single contaminated ingredient (e.g., raw oysters) is reported as the source or if all implicated ingredients belong to the same category (e.g., raw oysters and raw clams). When a food or contaminated ingredient cannot be assigned to a single

category, the outbreak is classified as not attributed to a single food category (7). The place where the implicated food was prepared is reported as one of 23 locations (e.g., a camp, farm, grocery store, or private home).

Population-based reporting rates were calculated for each state by use of U.S. Census Bureau estimates of the mid-year state populations for 2009–2015 (8). This report includes all foodborne outbreaks with a date of first illness onset from January, 1, 2009, through December, 31, 2015, but reported to FDOSS and finalized as of April 10, 2017.

Results

During 2009–2015, FDOSS received reports of 5,760 outbreaks, resulting in 100,939 illnesses, 5,699 hospitalizations, and 145 deaths (Figure 1). Outbreaks were reported by all 50 states, the District of Columbia, Puerto Rico, and CDC (Figure 2). The single-state outbreak reporting rate was 2.6 outbreaks per 1 million population. The overall national reporting rate (which includes multistate outbreaks) during 2009–2015 was also 2.6 outbreaks per 1 million population. Single-state outbreaks accounted for 5,583 (97%) of all outbreaks with 89,907 cases (median: 8 cases per outbreak; range: 2–800 cases). Four percent of these ill persons (3,733) were reported as being hospitalized. Multistate outbreaks accounted for 177 (3%) of all outbreaks with 11,032 cases (median: 20 cases per outbreak; range: 2–1,939 cases). Eighteen percent of these ill persons (1,966) were hospitalized.

Etiologic Agents

A single confirmed etiology was reported for 2,953 (51%) outbreaks, resulting in 67,130 illnesses, 5,114 hospitalizations, and 140 deaths (Table 1). Among 2,953 outbreaks with a single confirmed etiology, norovirus was the most common cause of outbreaks (1,130 outbreaks [38%]) and outbreak-associated illnesses (27,623 illnesses [41%]). Salmonella was the second most common single confirmed etiology reported, with 896 outbreaks (30%) and 23,662 illnesses (35%), followed by Shiga toxin-producing Escherichia coli (STEC) (191 outbreaks [6%]), Campylobacter (155 [5%]), Clostridium perfringens (108 [4%]), scombroid toxin (95 [3%]), ciguatoxin (80 [3%]), Staphylococcus aureus (35 [1%]), Vibrio parahaemolyticus (35 [1%]), and Listeria monocytogenes (35 [1%]). Listeria, Salmonella, and STEC were the most common causes of hospitalizations (82%) and deaths (82%) reported among persons in outbreaks with a single confirmed etiology.

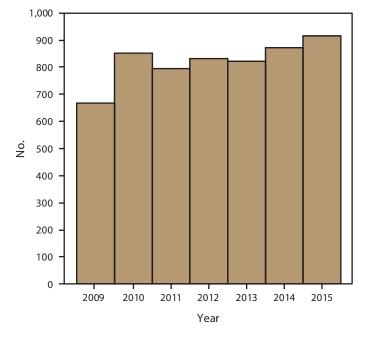


FIGURE 1. Number of foodborne disease outbreaks, by year — Foodborne Disease Outbreak Surveillance System, United States and Puerto Rico, 2009–2015

Location of Food Preparation

A location of preparation was provided for 5,022 outbreak reports (87%), with 4,696 (94%) indicating a single location (Table 2). Among outbreaks reporting a single location of preparation, restaurants were the most common location (2,880 outbreaks [61%]), followed by catering or banquet facilities (636 [14%]) and private homes (561 [12%]). Sitdown dining style restaurants (2,239 [48%]) were the most commonly reported type of restaurant. The locations of food preparation with the most outbreak-associated illnesses were restaurants (33,465 illnesses [43%]), catering or banquet facilities (18,141 [24%]), and institutions, such as schools (9,806 [13%]). The preparation location with the largest average number of illnesses per outbreak was institutions (46.5), whereas restaurants had the smallest (11.6).

Foods

Outbreak investigators identified a food in 2,442 outbreaks (42%). These outbreaks resulted in 51,341 illnesses (51%) (Table 3). The food reported belonged to a single food category in 1,281 outbreaks (22%). The food category most commonly implicated was fish (222 outbreaks [17%]), followed by dairy (136 [11%]) and chicken (123 [10%]). The food categories responsible for the most outbreak-associated illnesses were chicken (3,114 illnesses [12%]), pork (2,670 [10%]), and seeded vegetables (2,572 [10%]). Scombroid toxin in fish was

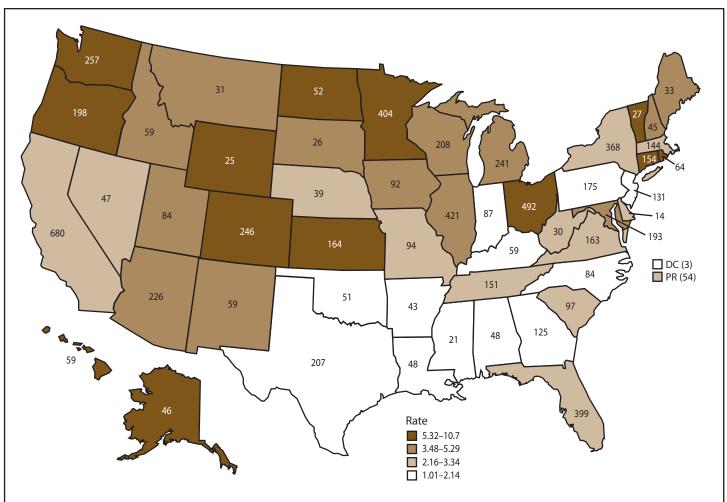
the single confirmed etiology and food category pair responsible for the most outbreaks (85), followed by ciguatoxin in fish (72) and *Campylobacter* in dairy (60) (Table 4). The pathogenfood category pairs that caused the most outbreak-associated illnesses were *Salmonella* in eggs (2,422 illnesses), *Salmonella* in seeded vegetables (2,203), and *Salmonella* in chicken (1,941). In comparison, scombroid toxin and ciguatoxin outbreaks from fish resulted in 519 outbreak-associated illnesses, an average of three illnesses per outbreak. Outbreaks of *Salmonella* infections from seeded vegetables resulted in an average of 88 illnesses per outbreak, and outbreaks of *Salmonella* infections from eggs resulted in an average of 78 illnesses per outbreak.

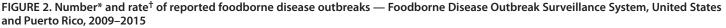
Several novel food vehicles caused outbreaks during the study period. In 2011, an outbreak of *Salmonella* serotype Enteritidis infections linked to pine nuts imported from Turkey resulted in 53 illnesses and two hospitalizations. In 2014, an outbreak of *Salmonella* serotypes Gaminara, Hartford, and Oranienburg in chia seed powder imported from Canada caused 45 illnesses and seven hospitalizations. An outbreak of STEC serogroups O26 and O121 infections that began in 2015 was linked to raw wheat flour produced in the United States; it resulted in 56 illnesses and 16 hospitalizations in 24 states. An outbreak of *Salmonella* serotype Virchow infections attributable to moringa leaf powder imported from South Africa began in 2015 and caused 35 illnesses and six hospitalizations in 24 states. It was an ingredient of an organic powdered shake mix branded to be used as a meal replacement.

Multistate Outbreaks

Multistate outbreaks comprised only 3% of outbreaks but were responsible for 11% of illnesses, 34% of hospitalizations, and 54% of deaths. Multistate outbreaks involved a median of seven states with a range of two to 45 states in which exposure occurred. The largest of the 177 multistate outbreaks was caused by Salmonella serotype Enteritidis and due to contaminated shell eggs. An estimated 1,939 persons were infected in 10 states beginning in 2010. An outbreak of Salmonella serotype Poona infections attributed to cucumbers in 2015 had the second highest number of illnesses (907 illnesses in 40 states). This outbreak also had the most outbreak-associated hospitalizations (204 [22% of cases]). An outbreak of Salmonella serotype Heidelberg infections attributed to chicken during 2013-2014 had the second most hospitalizations (200 [32% of cases]) and involved persons from 29 states and Puerto Rico. An outbreak of Listeria monocytogenes infections attributed to cantaloupes in 28 states in 2011 had the most deaths (33 [22% of cases]), followed in 2014 by an outbreak in 12 states of Listeria monocytogenes infections attributed to

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Abbreviations: DC = District of Columbia; PR = Puerto Rico.

* Total number of reported outbreaks in each area (N = 5,760), includes 177 multistate outbreaks (i.e., outbreaks in which exposure occurred in more than one state) assigned as an outbreak to each state involved. Multistate outbreaks involved a median of seven states (range: 2–45).

⁺ Per ¹ million population using U.S. Census Bureau estimates of the mid-year populations for 2009–2015. Source: US Census Bureau. Population and housing unit estimates. Washington, DC: US Department of Commerce, US Census Bureau; 2016. https://www.census.gov/programssurveys/popest.html. Cut points for outbreak rate categories determined by using quartiles.

caramel apples, another novel food vehicle (9), in which seven persons (20% of cases) died.

Discussion

Despite considerable advances in food safety in the United States during recent decades, foodborne disease outbreaks remain a serious public health problem. The majority of the outbreaks reported had relatively small case counts, and affected persons often were exposed in a single state. However, outbreaks with the largest case counts and most severe outcomes (e.g. highest proportion of ill persons hospitalized and most deaths) typically involved exposures in multiple states, reflecting factors such as the geographical distribution of the implicated food and the characteristics of the pathogens involved. Foods produced in other countries sometimes were implicated, highlighting the interconnectedness of the U.S. food supply with that of other nations, and the continued need to ensure that all foods are safe to eat (*10*).

As reported in previous summaries (11), norovirus remains the leading cause of foodborne disease outbreaks and outbreak-associated illnesses in the United States. Most foodborne norovirus outbreaks are associated with ready-toeat foods contaminated during preparation by infected food workers in restaurants and other food service settings (12). As such, continued efforts are needed to strengthen and ensure compliance with requirements in the FDA Model Food Code (13), specifically those that exclude symptomatic and post-symptomatic workers, prohibit bare-hand contact with ready-to-eat foods, and ensure appropriate hand washing. Contaminated raw food products, specifically leafy vegetables, fruits, and mollusks, also have been implicated in norovirus outbreaks (12); thus, upstream contamination during production also should be considered in foodborne norovirus outbreak investigations.

Fish was the most frequently implicated food, but the number of illnesses associated with these outbreaks tended to be small compared with other food vehicles, largely because of the pathogens involved. Differences in outbreak size are in part attributable to how pathogens contaminate foods: toxins are produced in individual fish, whereas *Salmonella* and other bacterial pathogens, such as STEC, can contaminate large amounts of product across vast distribution chains (14). This helps explain why bacterial pathogens are the most common causes of multistate outbreaks and why many persons can become ill during a single bacterial disease outbreak.

Identification of novel food sources provides insight into evolving food preferences in the United States and the types of foods that pathogens can contaminate. It also raises important scientific questions regarding how these pathogens remain viable in these foods long enough to cause infection. During the study period, a few novel food vehicles were identified as the sources of multistate outbreaks of *Listeria, Salmonella*, and STEC infections. Some of these (chia seed powder, raw wheat flour, and moringa leaf powder) are dried, shelf-stable foods not usually considered as possible sources of illness. These outbreak reports provide additional evidence that *Salmonella* and STEC can survive extensive processing steps as well as months in a desiccated state. This ability of pathogens to remain viable combined with the long shelf life of these products emphasizes the need for clear, well-publicized product recall notices.

Salmonella and STEC were two of the most common causes of large outbreaks. Regulatory-focused public health interventions, such as the 2009 Egg Safety Rule, the 2011 Food Safety Modernization Act, and the 2013 Salmonella Action Plan, were designed and implemented in part to help ensure the safety of foods that can be contaminated by these pathogens (15-17). Some members of the food industry also are promoting a culture of food safety by requiring growers, producers, and distributors to adhere to strict safety guidelines designed to prevent contamination. Additional efforts will likely be needed by both government and industry to help control these pathogens.

Limitations

The findings of this report are subject to at least four limitations. First, because CDC's foodborne outbreak surveillance is dynamic and agencies can submit, update, or delete reports at any time, the results of this analysis might differ slightly from previous or future reports. Second, not all outbreaks are identified and the majority of foodborne illnesses occur outside the context of a recognized outbreak. The degree to which the food vehicles, etiologies, and locations implicated in outbreaks represent the vehicles, etiologies, and locations of sporadic foodborne illness is unknown. Third, some outbreaks have an unknown food vehicle, an unknown etiology, or both, and analyses and conclusions drawn from outbreaks with an identified food vehicle and confirmed etiology might not be representative of all outbreaks. Finally, pathogens that are not known to cause illness sometimes are reported as a confirmed or suspected etiology.

Conclusion

Foodborne disease outbreaks remain an important public health issue. Data collected during outbreak investigations provide insight into the foods and pathogens that cause illnesses and the settings and conditions in which they occur. Continued efforts must be made to track and to analyze the causes of foodborne illness to inform targeted prevention efforts. In particular, strengthening the capacity of state and local health departments to investigate and to report outbreaks will improve foodborne disease outbreak surveillance and could help decrease the burden of foodborne illness through identification of foods, etiologies, outbreak settings, and specific points of contamination, which can inform intervention efforts.

Conflict of Interest

No conflicts of interest were reported.

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		Outb	reaks			Illne	esses		I	lospita	alizations			De	aths	
Etiology	CE*	SE	Total	%	CE	SE	Total	%	CE	SE	Total	%	CE	SE	Total	%
Bacterial																
Salmonella [†]	896	53	949	23	23,662	510	24,172	30	3,168	39	3,207	60	29	0	29	20
<i>Escherichia coli</i> , Shiga toxin- producing (STEC) [§]	191	12	203	5	2,378	87	2,465	3	672	21	693	13	12	1	13	9
Campylobacter [¶]	155	46	201	5	2,095	214	2,309	3	134	17	151	3	1	0	1	1
Clostridium perfringens	108	90	198	5	5,132	2,702	7,834	10	16	2	18	0	4	0	4	3
Staphylococcus aureus	35	40	75	2	1,255	426	1,681	2	69	17	86	2	0	0	0	0
Bacillus cereus	23	42	65	2	551	288	839	1	2	4	6	0	0	0	0	0
Vibrio parahaemolyticus	35	14	49	1	227	53	280	0	18	2	20	0	0	0	0	0
Shigella**	32	7	39	1	1,193	33	1,226	1	108	2	110	2	1	0	1	1
Listeria monocytogenes	35	1	36	1	380	8	388	0	334	7	341	6	74	1	75	52
Clostridium botulinum	19	2	21	1	85	6	91	0	72	6	78	1	4	0	4	3
Escherichia coli, Enterotoxigenic	6	1	7	0	437	19	456	1	1	0	1	0	0	0	0	0
Staphylococcus spp.	2	4	6	0	38	15	53	0	0	0	0	0	0	0	0	0
Yersinia enterocolitica	3	1	4	0	20	4	24	0	7	0	7	0	1	0	1	1
Vibrio cholerae	1	2	3	0	3	14	17	0	3	1	4	0	1	0	1	1
Streptococcus, Group A	2	1	3	0	72	40	112	0	0	0	0	0	0	0	0	0
Escherichia coli, Enteroaggregative	3	0	3	0	50	0	50	0	0	0	0	0	0	0	0	0
Vibrio other	2	0	2	0	7	0	7	0	3	0	3	0	0	0	0	0
Vibrio vulnificus	0	1	1	0	0	2	2	0	0	1	1	0	0	1	1	1
Aeromonas hydrophila	0	1	1	0	0	4	4	0	0	0	0	0	0	0	0	0
Coxiella burnetti	0	1	1	0	0	5	5	0	0	1	1	0	0	0	0	0
Francisella novicida	1	0	1	0	3	0	3	0	3	0	3	0	1	0	1	1
Brucella spp.	1	0	1	0	4	0	4	0	1	0	1	0	0	0	0	0
Clostridium other	1	0	1	0	12	0	12	0	0	0	0	0	0	0	0	0
Escherichia coli, Enteropathogenic	1	0	1	0	30	0	30	0	0	0	0	0	0	0	0	0
Enterococcus faecalis	1	0	1	0	13	0	13	0	0	0	0	0	0	0	0	0
Other	0	34	34	1	0	469	469	1	0	0	0	0	0	0	0	0
Subtotal	1,553	353	1,906	47	37,647	4,899	42,546	52	4,611	120	4,731	88	128	3	131	92
Chemical and toxin																
Scombroid toxin/histamine	95	6	101	2	280	19	299	0	1	1	2	0	0	0	0	0
Ciguatoxin	80	13	93	2	294	43	337	0	32	7	39	1	0 0	0	0	0 0
Mycotoxins	13	1	14	0	36	6	42	0	22	0	22	0	4	0	4	3
Puffer fish tetrodotoxin	3	0	3	0	9	0	9	0	4	Ő	4	Ő	0	0	0	0
Paralytic shellfish poison	3	0	3	0	12	0	12	0	6	0	6	0	0	0	0	0
Pesticides	2	0	2	0	42	0	42	0	2	0	2	0	0	0	0	0
Amnesic shellfish poison	1	0	1	0		0		0	2	0	2	0	0	0	0	0
Other	20	20	40	1	106	175	281	0	20	6	26	0	1	0	1	1
Subtotal	20	40	257	6	781	243	1,024	1	89	14	103	2	5	0	5	3
	21/	40	257	0	701	243	1,024	'	09	14	103	2	5	v	5	5

TABLE 1. Number and percentage of foodborne disease outbreaks, outbreak-associated illnesses, and hospitalizations, by etiology (confirmed or suspected) — Foodborne Disease Outbreak Surveillance System, United States and Puerto Rico, 2009–2015

See table footnotes on the next page.

		Outb	reaks			Illne	sses		I	Hospita	alizations			De	aths	
Etiology	CE*	SE	Total	%	CE	SE	Total	%	CE	SE	Total	%	CE	SE	Total	%
Parasitic																
Cryptosporidium	10	2	12	0	160	22	182	0	6	2	8	0	0	0	0	0
Trichinella	8	1	9	0	30	3	33	0	7	1	8	0	0	0	0	0
Cyclospora	9	0	9	0	432	0	432	1	17	0	17	0	0	0	0	0
Giardia	3	0	3	0	12	0	12	0	1	0	1	0	0	0	0	0
Subtotal	30	3	33	1	634	25	659	1	31	3	34	1	0	0	0	0
Viral																
Norovirus	1,130	740	1,870	46	27,623	9,413	37,036	45	275	99	374	7	7	0	7	5
Hepatitis A	15	0	15	0	260	0	260	0	107	0	107	2	0	0	0	0
Sapovirus	7	1	8	0	127	3	130	0	1	0	1	0	0	0	0	0
Rotavirus	1	1	2	0	58	28	86	0	0	1	1	0	0	0	0	0
Astrovirus	0	1	1	0	0	22	22	0	0	0	0	0	0	0	0	0
Other	0	2	2	0	0	25	25	0	0	0	0	0	0	0	0	0
Subtotal	1,153	745	1,898	46	28,068	9,491	37,559	46	383	100	483	9	7	0	7	5
Single etiology ^{††}	2,953	1,141	4,094	71	67,130	14,658	81,788	81	5,114	237	5,351	94	140	3	143	99
Multiple etiologies ^{§§}	33	50	83	1	925	1,070	1,995	2	56	21	77	1	0	0	0	0
Unknown etiology ^{¶¶}	0	0	1,583	27	0	0	17,156	17	0	0	271	5	0	0	2	1
Total	2,986	1,191	5,760	100	68,055	15,728	100,939	100	5,170	258	5,699	100	140	3	145	100

TABLE 1. (*Continued*) Number and percentage of foodborne disease outbreaks, outbreak-associated illnesses, and hospitalizations, by etiology (confirmed or suspected) — Foodborne Disease Outbreak Surveillance System, United States and Puerto Rico, 2009–2015

Abbreviations: CE = confirmed etiology; SE = suspected etiology.

* Guidelines for reporting agencies are to consider an etiology confirmed if it meets confirmation criteria (https://www.cdc.gov/foodsafety/outbreaks/investigatingoutbreaks/confirming_diagnosis.html); otherwise, it is considered suspected. Agents that are not listed in confirmation criteria or that are not known to cause illness are sometimes reported as confirmed or suspected etiologies.

⁺ Salmonella serotypes causing more than five outbreaks were Enteritidis (264 outbreaks), Typhimurium (102), Newport (73), Heidelberg (49), I 4,[5],12:i- (41), Javiana (37), Braenderup (29), Infantis (24), Montevideo (20), Muenchen (18), Thompson (17), Saintpaul (16), Oranienburg (15), Paratyphi B (10), Uganda (9), Agona (8), Typhimurium var Cope (8), Hadar (7), Mbandaka (7), Miami (6), and Virchow (6).

§ STEC serogroups O157 (156 outbreaks), O26 (14), O111 (7), O121 (6), O145 (5), multiple serogroups (4), O45 (4), O103 (3), unknown serogroup (3), and O186 (1).

[¶] Campylobacter jejuni (140 outbreaks), Campylobacter unknown species (49), Campylobacter multiple species (6), Campylobacter coli (5), and Campylobacter other (1).

** Shigella sonnei (33 outbreaks), Shigella flexneri (4), and Shigella unknown species (2).

⁺⁺ The denominator for the etiology percentages is the single etiology total. The denominator for the single etiology, multiple etiologies, and unknown etiology is the total. Because of rounding, numbers might not add up to the single etiology total or the total.

§§ If at least two etiologies are confirmed in an outbreak, it is considered a confirmed multiple etiology outbreak; otherwise it is considered a suspected multiple etiology outbreak.

^{¶¶} An etiologic agent was not confirmed or suspected based on clinical, laboratory, or epidemiologic information.

	Outbi	reaks	Illne	sses	— Mean illnesses	
Location	No.	%	No.	%	per outbreak	
Restaurant	2,880	61	33,465	43	12	
Sit-down dining	2,239	48	25,150	33	11	
Fast-food	369	8	4,414	6	12	
Buffet	9	0	97	0	11	
Other or unknown type	229	5	3,231	4	14	
Multiple types	34	1	573	1	17	
Catering or banquet facility	636	14	18,141	24	29	
Private home	561	12	8,080	10	14	
Institutional location	211	4	9,806	13	46	
School	69	1	2,164	3	31	
Prison or jail	67	1	5,077	7	76	
Camp	29	1	904	1	31	
Day care	7	0	193	0	28	
Office or indoor workplace	26	1	937	1	36	
Other	13	0	531	1	41	
Other location	26	1	482	1	19	
Other commercial location	258	5	4,284	6	17	
Grocery store	104	2	1,611	2	15	
Fair, festival, or temporary mobile service	37	1	620	1	17	
Farm or dairy	79	2	1,178	2	15	
Other	38	1	875	1	23	
Hospital or nursing home	68	1	1,527	2	22	
Nursing home	55	1	1,349	2	25	
Hospital	13	0	178	0	14	
Other private location	44	1	1,203	2	27	
Place of worship	32	1	1,014	1	32	
Picnic	5	0	37	0	7	
Other	7	0	152	0	22	
Hotel or motel	8	0	151	0	19	
Ship or boat	4	0	31	0	8	
Single location*	4,696	82	77,170	76	16	
Multiple locations	326	6	10,920	11	33	
Unknown location	738	13	12,849	13	17	
Total	5,760	100	100,939	100	18	

TABLE 2. Number and percentage of foodborne disease outbreaks and outbreak-associated illnesses, by location of food preparation — Foodborne Disease Outbreak Surveillance System, United States and Puerto Rico, 2009–2015

* The denominator for the location percentages is the single location total. The denominator for the single location, multiple locations, and unknown location is the total. Numbers might not add up to the single location total or the total due to rounding.

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TABLE 3. Number and percentage of foodborne disease outbreaks and outbreak-associated illnesses, by food category — Foodborne Disease Outbreak Surveillance System, United States and Puerto Rico, 2009–2015

	Outb	reaks	Illnes	ses
Food category*	No.	%	No.	%
Aquatic animal				
Crustaceans	12	1	74	0
Mollusks [†]	105	8	846	3
Fish	222	17	1,353	5
Other aquatic animals	5	0	15	0
Subtotal	344	27	2,288	9
Land animal				
Dairy [§]	136	11	1,639	6
Eggs	36	3	2,470	9
Beef	106	8	1,934	7
Pork	89	7	2,670	10
Other meat (e.g., sheep or goat)	6	0	50	0
Chicken	123	10	3,114	12
Turkey	50	4	1,675	6
Other poultry	6	0	71	0
Game	13	1	86	0
Subtotal	565	44	13,709	52
Plant				
Oils and sugars	4	0	18	0
Fungi	16	1	56	0
Sprouts	21	2	766	3
Root and other underground vegetables [¶]	20	2	383	1
Seeded vegetables**	44	3	2,572	10
Herbs	7	1	476	2
Vegetable row crops ^{††}	81	6	1,972	7
Fruits ^{§§}	78	6	2,420	9
Grains and beans ^{¶¶}	52	4	838	3
Nuts and seeds***	11	1	245	1
Subtotal	334	26	9,746	37
Other	38	3	807	3
Food reported, attributed to a single food category ^{†††}	1,281	22	26,550	26
Food reported, not attributed to a single food category	1,161	20	24,791	25
No food reported	3,318	58	49,598	49
Total ^{†††}	5,760	100	100,939	100

* Source: Interagency Food Safety Analytics Collaboration (IFSAC) food categorization scheme (https://www.cdc.gov/foodsafety/ifsac/projects/ food-categorization-scheme.html).

[†] Bivalve mollusks (102 outbreaks) and nonbivalve mollusks (3).

[§] Unpasteurized dairy products (109 outbreaks), pasteurized dairy products (20), and pasteurization unknown (7).

[¶] Tubers (12 outbreaks), roots (5), and bulbs (3).

** Solanaceous seeded vegetables (23 outbreaks), vine-grown seeded vegetables (11), legumes (7), other seeded vegetables (2), and seeded vegetables not further classified (1).

⁺⁺ Leafy vegetables (77 outbreaks) and stem vegetables (4).

^{§§} Fruits not further classified (24 outbreaks), pome fruits (15), melons (14), small fruits (11), sub-tropical fruits (7), tropical fruits (5), and stone fruits (2).

¹¹ Grains (32 outbreaks), beans (15), and grains and beans not further classified (5).

*** Nuts (8 outbreaks) and seeds (3).

**** The denominator for the food category percentages is the "food reported, attributed to a single food category" total. The total comprises "food reported attributed to a single food category," "food reported, not attributed to a single food category," and "no food reported." Numbers might not add up exactly due to rounding.

Characteristic	Food category*	No. outbreaks	No. illnesses	No. hospitalizations	No. deaths
Top 5 pathogen-food category pair	rs resulting in outbreaks				
Etiology	-				
Scombroid toxin/histamine	Fish	85	250	1	0
Ciguatoxin	Fish	72	269	31	0
Campylobacter	Dairy	60	917	51	1
Salmonella	Chicken	49	1,941	372	0
Salmonella	Pork	43	1,539	206	3
Top 5 pathogen-food category pair	rs resulting in outbreak-associate	ed illnesses			
Etiology	5				
Salmonella	Eggs	31	2,422	41	1
Salmonella	Seeded vegetables	25	2,203	419	7
Salmonella	Chicken	49	1,941	372	0
Salmonella	Pork	43	1,539	206	3
Campylobacter	Dairy	60	917	51	1
Top 5 pathogen-food category pair	rs resulting in outbreak-associate	ed hospitalizations			
Etiology	2	·			
Salmonella	Seeded vegetables	25	2,203	419	7
Salmonella	Chicken	49	1,941	372	0
Salmonella	Fruits	24	838	227	6
Salmonella	Pork	43	1,539	206	3
Listeria monocytogenes	Fruits	3	184	179	41
Top 5 pathogen-food category pair	rs resulting in outbreak-associate	ed deaths			
Etiology	-				
Listeria monocytogenes	Fruits	3	184	179	41
Listeria monocytogenes	Dairy	14	106	70	14
Salmonella	Seeded vegetables	25	2,203	419	7
Salmonella	Fruits	24	838	227	6
Listeria monocytogenes	Vegetable row crops	2	29	29	6

TABLE 4. Most common confirmed pathogen-food category pairs resulting in outbreaks, outbreak-associated illnesses, hospitalizations, and deaths — Foodborne Disease Outbreak Surveillance System, United States and Puerto Rico, 2009–2015

* Source: Interagency Food Safety Analytics Collaboration (IFSAC) food categorization scheme: https://www.cdc.gov/foodsafety/ifsac/projects/food-categorizationscheme.html.

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Table 2. Estimated annual number of episodes of illnesses caused by 31 pathogens transmitted commonly by food, United States*

		Multi	pliers		Travel-			
Pathogen	Laboratory- confirmed	Under- reporting†	Under- diagnosis‡	Total, mean (90% Crl)	related, percentage	Domestically acquired, mean (90% Crl)	Foodborne, percentage§	Domestically acquired foodborne, mean (90% Crl)
Bacteria								
Bacillus cereus, foodborne¶	85"	25.5	29.3	63,623 (15,770–147,827)	<1	63,411 (15,721–147,380)	100	63,400 (15,719–147,354)
Brucella spp.	120**	1.1	15.2	2,003 (1,302–2,964)	16	1,679 (1,089–2,484)	50	839 (533–1,262)
Campylobacter spp.	43,696**	1.0	30.3	1,322,137 (530,126-2,521,026)	20	1,058,387 (423,255–2,019,498)	80	845,024 (337,031–1,611,083)
Clostridium botulinum, foodborne¶	25**	1.1	2.0	56 (34–92)	<1	55 (34–91) 1		55 (34–91)
Clostridium perfringens, foodborne¶	1,295"	25.5	29.3	969,342 (192,977-2,492,003)	<1	966,120 (192,331–2,483,682)	100	965,958 (192,316–2,483,309)
STEC 0157	3,704**	1.0	26.1	96,534 (26,982–227,891)	4	93,094 (26,046–219,676)	68	63,153 (17,587–149,631)
STEC non-O157	1,579 ⁺⁺	1.0	106.8	168,698 (17,163-428,522)	18	138,063 (14,080–350,891)	82	112,752 (11,467–287,321)
ETEC, foodborne¶	53"	25.5	29.3	39,781 (53–102,250)	55	17,897 (24–46,215)	100	17,894 (24–46,212)
<i>Diarrheagenic E. coli</i> other than STEC and ETEC	53	25.5	29.3	39,871 (53–102,378)	<1	39,739 (52–102,028)	30	11,982 (16–30,913)
Listeria monocytogenes	808++	1.0	2.1	1,662 (582–3,302)	3	1,607 (563–3,193)	99	1,591 (557–3,161)
Mycobacterium bovis	195**	1.0	1.1	208 (177–241)	70	63 (49–78)	95	60 (46–74)
Salmonella spp., nontyphoidal‡‡	41,930++	1.0	29.3	1,229,007 (772,129–2,008,076)	11	1,095,079 (687,126–1,790,225)	94	1,027,561 (644,786–1,679,667)
S. enterica serotype Typhi	433**	1.0	13.3	5,752 (299–17,357)	67	1,897 (91–5,756)	96	1,821 (87–5,522)
Shigella spp.	14,864++	1.0	33.3	494,908 (93,877–1,420,877)	15	421,048 (79,844–1,208,445)	31	131,254 (24,511–374,789)
Staphylococcus aureus, foodborne¶	323**	25.5	29.3	241,994 (72,584–531,398)	<1	241,188 (72,352–529,509)	100	241,148 (72,341–529,417)
Streptococcus spp. group A, foodborne¶	15'	25.5	29.3	11,257 (15–78,104)	<1	11,219 (15–77,875)	100	11,217 (15–77,875)
Vibrio cholerae, toxigenic	8**	1.1	33.1	277 (94–630)	70	84 (19–212)	100	84 (19–213)
V. vulnificus	111**	1.1	1.7	207 (138–287)	2	203 (136–281)	47	96 (60–139)
V. parahaemolyticus	287**	1.1	142.4	44,950 (23,706–74,984)	10	40,309 (21,277–67,282)	86	34,664 (18,260–58,027)
Vibrio spp., other	220**	1.1	142.7	34,585 (21,756–51,535)	11	30,727 (19,278–45,886)	57	17,564 (10,848–26,475)
Yersinia enterocolitica	950**	1.0	122.8	116,716 (36,363–204,898)	7	108,490 (33,797–190,605)	90	97,656 (30,388–172,734)
Subtotal				4,883,568 (3,160,412-7,148,360)		4,330,358 (2,771,307–6,438,919)		3,645,773 (2,321,468-5,581,290)
Parasites								
Cryptosporidium spp.	7,594††	1.0	98.6	748,123 (162,961–2,135,110)	9	678,828 (147,796–1,940,626)	8	57,616 (12,060 166,771)
Cyclospora cayetanensis	239††	1.0	83.1	19,808 (239–65,135)	42	11,522 (139–38,031)	99	11,407 (137–37,673)
Giardia intestinalis	20,305**	1.3	46.3	1,221,564 (892,393–1,633,965)	8	1,121,864 (818,627–1,501,290)	7	76,840 (51,148–109,739)
Toxoplasma gondii		1.0	0	173,995 134,593-218,866)	<1	173,415 (134,172–218,169)	50	86,686 (64,861-111,912)
Trichinella spp.	13**	1.3	9.8	162 (44–355)	4	156 (42–341)	100	156 (42–341)
Subtotal				2,163,652 (1,401,591-3,596,566)		1,985,785 (1,292,817–3,290,175)		232,705 (161,923–369,893)
Viruses								
Astrovirus	NA	NA	NA	3,090,384 (2,350,589–3,833,232)	0	3,089,868 (2,350,263-3,832,706)	<1	15,433 (5,569–26,643)
Hepatitis A virus	3,576**	1.1	9.1	35,769 (21,505–60,715)	41	21,041 (12,455–35,918)	7	1,566 (702–3,024)
Norovirus	NA	NA	NA	20,865,958 (12,842,072-30,743,963)	<1	20,796,079 (12,798,628–30,638,633)	26	5,461,731 (3,227,078-8,309,480)
Rotavirus	NA	NA	NA	3,090,384 (2,350,589–3,833,232)	0	3,089,868 (2,350,263-3,832,706)	<1	15,433 (5,569–26,643)
Sapovirus	NA	NA	NA	3,090,384 (2,350,589–3,833,232)	0	3,089,868 (2,350,263-3,832,706)	<1	15,433 (5,569–26,643)
Subtotal				30,172,879 (21,795,012-40,272,501)		30,086,723 (21,733,225-40,154,878)		5,509,597 (3,273,623-8,355,568)
TOTAL				37,220,098 (28,434,745-47,630,066)		36,402,867 (27,698,948–46,716,681)		9,388,075 (6,641,440–12,745,709)

*All estimates were based on US population in 2006. Modal or mean value shown unless otherwise stated; see online Technical Appendix 3 (www.cdc.gov/EID/content/17/1/7-Techapp3.pdf) for the parameters of these distributions. The credible interval (CrI) lower bound for total illnesses was replaced with the number of laboratory–confirmed illnesses when that lower bound was zero. The observed lower bound was then carried forward using the travel–related and foodborne percentages. STEC, Shiga toxin–producing *Escherichia coli*; ETEC, enterotoxigenic *E. coli*; NA, not applicable.

Adjustment for underreporting because of surveillance method; underreporting multiplier for passive surveillance systems (Cholera and Other *Vibrio* Illness Surveillance [COVIS] or the National Notifiable Disease Surveillance System [NNDSS]) derived by comparing the incidence of laboratory–confirmed illnesses for *Listeria*, non–typhoidal *Salmonella* spp., *Shigella*, and STEC 0157 (for bacteria) and *Cryptosporidium* spp. and *Cyclospora cayetanensis* (for parasites) ascertained in the Foodborne Diseases Active Surveillance Network (FoodNet) to the incidence of laboratory–confirmed illnesses for the same pathogens reportable to NNDSS; underreporting multiplier for outbreakassociated illness reported through the Foodborne Disease Outbreak Surveillance System (FDOSS) derived by comparing the incidence of laboratory–confirmed illnesses caused by *Campylobacter* spp., *Cryptosporidium* spp., *Cyclospora cayetanensis*, STEC, *Listeria monocytogenes*, *Salmonella* spp., *Shigella* spp., *vibrio* spp., and *Yersinia* enterocolitica ascertained in FoodNet to the incidence of laboratory–confirmed illnesses of these bacterial infections reported to FDOSS. The modal value is presented here; online Technical Appendix 3 has the low and high values of these PERT distributions. More detail on the data used to estimate underreporting multipliers is given in online Technical Appendix 4 (www.cdc.gov/EID/content/17/1/7-Techapp4.pdf).

*Adjustment for underdiagnosis because of variations in medical care seeking, specimen submission, laboratory testing, and test sensitivity. The modal value is presented here; online Technical Appendix 3 describes the low and high values of these PERT distributions.

§Percent foodborne among domestically acquired illnesses.

¶Estimates based on the number of foodborne illnesses ascertained in surveillance and therefore assumed to reflect only foodborne transmission.

#Passive surveillance data on outbreak-associated illnesses from FDOSS.

**Passive surveillance data from COVIS or NNDSS.

++Active surveillance data from FoodNet, adjusted for geographical coverage; data from the NTSS for M. bovis.

##For all analyses in this article, S. enterica serotype Paratyphi is grouped with nontyphoidal Salmonella spp.

National Center for Emerging and Zoonotic Infectious Diseases

Division of Foodborne, Waterborne, and Environmental Disease



Surveillance for Waterborne Disease Outbreaks Associated with Drinking Water — United States, 2013–2014

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Provision of safe water in the United States is vital to protecting public health (1). Public health agencies in the U.S. states and territories* report information on waterborne disease outbreaks to CDC through the National Outbreak Reporting System (NORS) (https://www.cdc.gov/healthywater/surveillance/index.html). During 2013-2014, 42 drinking waterassociated[†] outbreaks were reported, accounting for at least 1,006 cases of illness, 124 hospitalizations, and 13 deaths. Legionella was associated with 57% of these outbreaks and all of the deaths. Sixty-nine percent of the reported illnesses occurred in four outbreaks in which the etiology was determined to be either a chemical or toxin or the parasite Cryptosporidium. Drinking water contamination events can cause disruptions in water service, large impacts on public health, and persistent community concern about drinking water quality. Effective water treatment and regulations can protect public drinking water supplies in the United States, and rapid detection, identification of the cause, and response to illness reports can reduce the transmission of infectious pathogens and harmful chemicals and toxins.

To provide information about drinking water–associated waterborne disease outbreaks in the United States in which the first illness occurred in 2013 or 2014 (https://www.cdc. gov/healthywater/surveillance/drinking-surveillance-reports. html), CDC analyzed outbreaks reported to the CDC Waterborne Disease and Outbreak Surveillance System through NORS (https://www.cdc.gov/nors/about.html) as of December 31, 2015. For an event to be defined as a waterborne disease outbreak, two or more cases must be linked epidemiologically by time, location of water exposure, and illness characteristics; and the epidemiologic evidence must implicate water exposure as the probable source of illness. Data requested for each outbreak include 1) the number of cases, hospitalizations, and deaths; 2) the etiologic agent (confirmed or suspected); 3) the implicated water system;

4) the setting of exposure; and 5) relevant epidemiologic and environmental data needed to understand the outbreak occurrences and for determining the deficiency classification.[§] One previously unreported outbreak with onset date of first illness in 2012 is presented but is not included in the analysis of outbreaks that occurred during 2013–2014.

Public health officials from 19 states reported 42 outbreaks associated with drinking water during the surveillance period (Table 1) (https://www.cdc.gov/healthywater/surveillance/ drinking-water-tables-figures.html). These outbreaks resulted in at least 1,006 cases of illness, 124 hospitalizations (12% of cases), and 13 deaths. At least one etiologic agent was identified in 41 (98%) outbreaks. Counts of etiologic agents in this report include both confirmed and suspected etiologies, which differs from previous surveillance reports. Legionella was implicated in 24 (57%) outbreaks, 130 (13%) cases, 109 (88%) hospitalizations, and all 13 deaths (Table 1). Eight outbreaks caused by two parasites resulted in 289 (29%) cases, among which 279 (97%) were caused by Cryptosporidium, and 10 (3%) were caused by Giardia duodenalis. Chemicals or toxins were implicated in four outbreaks involving 499 cases, with 13 hospitalizations, including the first reported outbreaks (two outbreaks) associated with algal toxins in drinking water.

The most commonly reported outbreak etiology was *Legionella* (57%), making acute respiratory illness the most common predominant illness type reported in outbreaks (Table 2). Thirty-five (83%) outbreaks were associated with public (i.e., regulated), community or noncommunity water systems,[¶] and three (7%) were associated with unregulated,

^{*} Outbreak reports can be submitted by public health agencies in the U.S states, District of Columbia, Guam, Puerto Rico, Marshall Islands, Federated States of Micronesia, Northern Mariana Islands, Palau, and U.S. Virgin Islands.

[†] Drinking water, also called potable water, is water for human consumption (e.g., drinking, bathing, showering, hand-washing, teeth brushing, food preparation, dishwashing, and maintaining oral hygiene) and includes water collected, treated, stored, or distributed in public and individual water systems, as well as bottled water.

[§] Waterborne disease outbreaks are assigned one or more deficiency classifications based on available data. The deficiencies provide information regarding how the water became contaminated, characteristics of the water system, and factors leading to waterborne disease outbreaks. Outbreaks are assigned one or more deficiency classifications based on available data. https://www.cdc.gov/ healthywater/surveillance/deficiency-classification.html.

⁹ Community and noncommunity water systems are public water systems that have ≥15 service connections or serve an average of ≥25 residents for ≥60 days per year. A community water system serves year-round residents of a community, subdivision, or mobile home park. A noncommunity water system serves an institution, industry, camp, park, hotel, or business and can be nontransient or transient. Nontransient systems serve ≥25 of the same persons for ≥6 months of the year but not year-round (e.g., factories and schools) whereas transient systems provide water to places in which persons do not remain for long periods of time (e.g., restaurants, highway rest stations, and parks). Individual water systems are small systems not owned or operated by a water utility that have <15 connections or serve <25 persons.

TABLE 1. Waterborne disease outbreaks associated with drinking water (N = 42), by state/jurisdiction and month of first case onset — Waterborne	e
Disease and Outbreak Surveillance System, United States, 2013–2014	

State/ Jurisdiction	Month	Year	Etiology*	Predominant illness [†]		No. of hospitalizations [§]	No. of deaths [¶]	Type of water system**	Water source	Setting
Alaska	Aug	2014	Giardia duodenalis ^{††}	AGI	5	0	0	Community	River/Stream	Community/Municipality
Arizona	Jan	2014	Norovirus (S)	AGI	4	0	0	Transient, noncommunity	Unknown	Camp/Cabin Setting
Florida	Sep	2013	L. pneumophila serogroup 1	ARI	4	4	0	Community	Well	Hospital/Health care
Florida	Nov	2013	L. pneumophila serogroup 1	ARI	4	4	0	Community	Other	Other ^{§§}
Florida	Apr	2014	L. pneumophila serogroup 1	ARI	2	2	0	Community	Well	Hotel/Motel/Lodge/Inn
Florida	Jun	2014	L. pneumophila serogroup 1	ARI	3	2	0	Community	Unknown	Long-term care facility
Florida	Aug	2014	L. pneumophila serogroup 1	ARI	6	4	0	Community	Unknown	Hotel/Motel/Lodge/Inn
Idaho	Sep	2014	Giardia duodenalis	AGI	2	0	0	Unknown	Unknown	Hotel/Motel/Lodge/Inn
Indiana	Jul	2013		AGI	7	0	0	Community	Unknown	Mobile home park
Indiana	Nov		Unknown	AGI	3	0	0	Community	Unknown	Apartment/Condo
Kansas	June		L. pneumophila serogroup 1	ARI	2	2	0	Community	Unknown	Hospital/Health care
Maryland	Nov	2012	L. pneumophila serogroup 1	ARI	2 ^{¶¶}	2 ^{¶¶}	0	Community	Well	Hotel/Motel/Lodge/Inn
Maryland	Feb	2013	Nitrite***	AGI, Neuro	14		0	Community	Lake/Reservoir/ Impoundment	Indoor workplace/Office
Maryland	Apr	2014	L. pneumophila serogroup 1	ARI	2	2	0	Community	Lake/Reservoir/ Impoundment	Apartment/Condo
Maryland	Jul	2014	L. pneumophila serogroup 1	ARI	2	1	0	Community	Well	Hotel/Motel/Lodge/Inn
Maryland	Aug	2014	L. pneumophila serogroup 1	ARI	2	2	0	Community	River/Stream	Prison/Jail (Juvenile/Adult
Michigan	Jun	2014	L. pneumophila serogroup 1	ARI	45	45	7	Community	River/Stream	Hospital/Health care, Community/ Municipality ^{†††}
Montana	Jul	2014	Norovirus GII.Pe-GII.4 Sydney	AGI	62	0	0	Transient, noncommunity	Well	Hotel/Motel/Lodge/Inn
New York	Jul	2013	L. pneumophila serogroup 1	ARI	2	2	0	Community	Lake/Reservoir/ Impoundment	Hospital/Health care
New York	Jun	2014	L. pneumophila serogroup 1	ARI	2	2	0	Community	Well	Hospital/Health care
North Carolina	Dec	2013	L. pneumophila serogroup 1	ARI	3	2	0	Community	Unknown	Long-term care facility
North Carolina	Dec	2013	L. pneumophila serogroup 1	ARI	7	3	0	Community	Unknown	Long-term care facility
North Carolina	May	2014	L. pneumophila serogroup 1	ARI	7	6	1	Community	Other	Long-term care facility
North Carolina	Jun	2014	L. pneumophila serogroup 1	ARI	3	3	0	Community	Unknown	Long-term care facility
North Carolina	Jul	2014	L. pneumophila serogroup 1	ARI	3	2	1	Community	Unreported	Long-term care facility
Ohio	Apr	2013	L. pneumophila	ARI	2	2	1	Unknown	Unknown	Long-term care facility
Ohio ^{§§§}	Sep		Cyanobacterial toxin ^{¶¶¶}	AGI	6	0	0	Community	Lake/Reservoir/ Impoundment	Community/Municipality
Ohio	Jul	2014	L. pneumophila serogroup 1	ARI	14	4	0	Community	River/Stream	Long-term care facility
Ohio	Aug	2014	Cyanobacterial toxin ^{¶¶¶}	AGI	110			Community	Lake/Reservoir/ Impoundment	Community/Municipality
Ohio	Oct	2014	Cryptosporidium sp. (S)****	AGI	100	0	0	Individual	River/Stream	Farm/Agricultural setting
Ohio	Dec	2014	Viral, unknown (S)	AGI	2	0	0	Commercially bottled	Unknown	Private residence
Oregon	Jun	2013	Cryptosporidium parvum IIaA15G2R1	AGI	119	2	0	Community	Lake/Reservoir/ Impoundment	Community/Municipality
Oregon	Sep	2014	<i>L. pneumophila</i> serogroup 1	ARI	4	4	1	Community	Well	Apartment/Condo
Pennsylvania	Dec	2013	L. pneumophila serogroup 1	ARI	2	2	0	Unknown	Unknown	Hospital/Health care
Pennsylvania	Feb	2014	L. pneumophila serogroup 1	ARI	5	5	0	Community	River/Stream	Long-term care facility
Pennsylvania	Oct	2014	L. pneumophila	ARI	2	2	1	Community	Unknown	Long-term care facility
Rhode Island	Apr		L. pneumophila serogroup 1	ARI	2	2	1	Community	Lake/Reservoir/ Impoundment	Hospital/Health care

See table footnotes on the next page.

State/ Jurisdiction	Month	Year	Etiology*	Predominant illness [†]		No. of hospitalizations [§]	No. of deaths [¶]	Type of water system**	Water source	Setting
Tennessee	Jul	2013	Cryptosporidium parvum	AGI	34	0	0	Transient, noncommunity ⁺⁺⁺⁺	Spring	Camp/Cabin setting
Tennessee	Jun	2014	Clostridium difficile (S); Escherichia coli, Enteropathogenic (S)	AGI	12	0	0	Nontransient, noncommunity	Well	Camp/Cabin setting; Community/Municipality
Virginia	Jun	2013	Cryptosporidium sp.	AGI	19	0	0	Individual	Well	Farm/Agricultural setting
West Virginia	Jan	2014	4-Methylcyclo hexanemethanol (MCHM) ^{§§§§}	AGI	369	13	0	Community	River/Stream	Community/Municipality
Wisconsin	Aug	2014	Giardia duodenalis	AGI	3	0	0	Nontransient, noncommunity	Other	National forest
Wisconsin	Sep	2014	Campylobacter jejuni	AGI	5	0	0	Individual	Well	Private residence

TABLE 1. (*Continued*) Waterborne disease outbreaks associated with drinking water (N = 42), by state/jurisdiction and month of first case onset — Waterborne Disease and Outbreak Surveillance System, United States, 2013–2014

Abbreviations: AGI = acute gastrointestinal illness; ARI = acute respiratory illness; *L. pneumophila* = *Legionella pneumophila*; Neuro = neurologic illnesses, conditions, or symptoms (e.g., meningitis); S = suspected.

* Etiologies listed are confirmed, unless indicated as suspected. For multiple-etiology outbreaks, etiologies are listed in alphabetical order.

⁺ The category of illness reported by ≥50% of ill respondents. All legionellosis outbreaks were categorized as ARI.

[§] Value was set to "missing" in reports where zero hospitalizations were reported and the number of persons for whom information was available was also zero or for instances where reports are missing hospitalization data.

¹ Value was set to "missing" in reports where zero deaths were reported and the number of persons for whom information was available was also zero or for instances where reports are missing data on associated deaths.

** Community and noncommunity water systems are public water systems that have ≥15 service connections or serve an average of ≥25 residents for ≥60 days per year. A community water system serves year-round residents of a community, subdivision, or mobile home park. A noncommunity water system serves an institution, industry, camp, park, hotel, or business and can be nontransient or transient. Nontransient systems serve ≥25 of the same persons for ≥6 months of the year but not year-round (e.g., factories and schools) whereas transient systems provide water to places in which persons do not remain for long periods of time (e.g., restaurants, highway rest stations, and parks). Individual water systems are small systems not owned or operated by a water utility that have <15 connections or serve <25 persons.

⁺⁺ Classification of all reported *Giardia* cases has changed from *Giardia intestinalis* to *Giardia duodenalis* to align with laboratory standards.

^{§§} Setting is listed as "other" because implicated facility houses both independent living and assisted living facilities.

^{¶¶} This count was not included in the analysis of the current report. This outbreak occurred in 2012 and was not reported in the previous drinking water outbreak report.
*** Patients' methemoglobin levels ranged from 1.6% to 32.3%. Water was determined to be the source rather than food because all cases had direct exposure to water. Of the 14 cases, five used the water to make oatmeal or cream of wheat.

⁺⁺⁺ This report includes both community and hospital-associated cases (27 of 45 patients reported health care/hospital exposure).

^{§§§} This is the first drinking water–associated outbreak of this etiology reported to the National Outbreak Reporting System.

^{¶¶¶} Microcystin was detected in finished water sampled from a community water system; levels exceeded state thresholds and resulted in a "Do not drink" advisory. **** *Cryptosporidium* was detected in water samples but not in any clinical specimens.

⁺⁺⁺⁺ This system was registered as a community system as a result of the outbreak investigation.

§§§§ Illnesses were associated with exposure to 4-methylcyclohexanemethanol following a documented industrial spill into water supplying a public water system. However, individual levels of exposure could not be quantified in clinical specimens. Propylene glycol phenyl ether was also present in the spill at low concentrations.

individual systems. Fourteen outbreaks occurred in drinking water systems with groundwater sources and an additional 14 occurred in drinking water systems with surface water sources. The most commonly cited deficiency, which led to 24** (57%) of the 42 drinking water–associated outbreaks, was the presence of *Legionella* in drinking water systems. In addition, 143 (14%) cases were associated with seven (17%) outbreak reports that had a deficiency classification indicating "unknown or insufficient information."

Among 1,006 cases attributed to drinking water–associated outbreaks, 50% of the reported cases were associated with chemical or toxin exposure, 29% were caused by parasitic infection (either *Cryptosporidium* or *Giardia*), and 13% were caused by *Legionella* infection (Table 2). Seventy-five percent of cases were linked to community water systems. Outbreaks in water systems supplied solely by surface water accounted for most cases (79%). Of the 1,006 cases, 86% originated from

outbreaks in which the predominant illness was acute gastrointestinal illness. Three (7%) outbreaks in which treatment was not expected to remove the contaminant were associated with a chemical or toxin and resulted in 48% of all outbreakassociated cases.

Discussion

Water treatment processes, regulations, and rapid response to illness outbreaks continue to reduce the transmission of pathogens, reduce exposure to chemicals and toxins, and protect the public drinking water supplies in the United States. Outbreaks reported during this surveillance period include the first reports of drinking water–associated outbreaks caused by harmful algal blooms as well as the continued challenges of preventing and controlling illnesses and outbreaks caused by *Legionella* and *Cryptosporidium*. Outbreaks in community water systems caused by chemical spills (West Virginia) (2), harmful algal blooms (Ohio), *Cryptosporidium* (Oregon) (3), and *Legionella* (Michigan) demonstrated that diverse contaminants can cause

^{**} One of the 24 outbreaks included both deficiencies 5a and 7 under the "multiple" classification.

	Outbreaks (N = 42)		Cases (N = 1,006)	
Characteristic/Rank	Category	No. (%)	Category	No. (%)
Etiology				
1	Bacteria, <i>Legionella</i>	24 (57.1)	Chemical/Toxin	499 (49.6)
2	Parasites	8 (19.1)	Parasites	289 (28.7)
3	Chemical/Toxin	4 (9.5)	Bacteria, Legionella	130 (12.9)
1	Viruses	3 (7.1)	Viruses	68 (6.8)
5	Bacteria, non-Legionella	1 (2.4)	Multiple bacteria	12 (1.2)
5	Multiple bacteria	1 (2.4)	Bacteria, non- <i>Legionella</i>	5 (0.5)
7	Unknown	1 (2.4)	Unknown	3 (0.3)
Vater system*				
1	Community	30 (71.4)	Community	759 (75.4)
2	Noncommunity	5 (11.9)	Individual	124 (12.3)
3	Individual	3 (7.1)	Noncommunity	115 (11.4)
4	Unknown	3 (7.1)	Unknown	6 (0.6)
5	Bottled	1 (2.4)	Bottled	2 (0.2)
Water source				
1	Ground water	14 (33.3)	Surface water	795 (79.0)
2	Surface water	14 (33.3)	Ground water	157 (15.6)
3	Unknown	12 (28.6)	Unknown	39 (3.9)
4	Mixed [†]	1 (2.4)	Mixed	12 (1.2)
5	Unreported	1 (2.4)	Unreported	3 (0.3)
Predominant illness [§]				
1	ARI	24 (57.1)	AGI	862 (85.7)
2	AGI	17 (40.5)	ARI	130 (12.9)
3	AGI; Neuro	1 (2.4)	AGI; Neuro	14 (1.4)
Deficiency¶				
1	Legionella spp. in drinking water system**	23 (54.8)	Treatment not expected to remove contaminant	485 (48.2)
2	Unknown/Insufficient information ^{††}	7 (16.7)	Unknown/Insufficient information	143 (14.2)
3	Multiple ^{§§}	3 (7.1)	<i>Legionella</i> spp. in drinking water system	126 (12.5)
4	Treatment not expected to remove contaminant ^{¶¶}	3 (7.1)	Treatment deficiency	119 (11.8)
5	Untreated ground water***	3 (7.1)	Untreated ground water	70 (7.0)
5	Distribution system ^{†††}	1 (2.4)	Multiple	42 (4.2)
7	Premises plumbing system ^{§§§}	1 (2.4)	Premise plumbing system	14 (1.4)
3	Treatment deficiency ^{¶¶¶}	1 (2.4)	Distribution system	7 (0.7)

TABLE 2. Rank order (most common to least common) of etiology, water system, water source, predominant illness, and deficiencies associated
with 42 drinking water outbreaks and 1,006 outbreak-related cases of illness — United States, 2013–2014

Abbreviations: AGI = acute gastrointestinal illness; ARI = acute respiratory illness; Neuro = neurologic illnesses, conditions, or symptoms (e.g., meningitis).

* Community and noncommunity water systems are public water systems that have ≥15 service connections or serve an average of ≥25 residents for ≥60 days per year. A community water system serves year-round residents of a community, subdivision, or mobile home park. A noncommunity water system serves an institution, industry, camp, park, hotel, or business and can be nontransient or transient. Nontransient systems serve ≥25 of the same persons for ≥6 months of the year but not year-round (e.g., factories and schools) whereas transient systems provide water to places in which persons do not remain for long periods of time (e.g., restaurants, highway rest stations, and parks). Individual water systems are small systems not owned or operated by a water utility that have <15 connections or serve <25 persons.

[†] Includes outbreaks with mixed water sources (i.e., ground water and surface water).

[§] The category of illness reported by ≥50% of ill respondents; all legionellosis outbreaks were categorized as ARI.

[¶] Outbreaks are assigned one or more deficiency classifications. https://www.cdc.gov/healthywater/surveillance/deficiency-classification.html.

** Deficiency 5A. Drinking water, contamination of water at points not under the jurisdiction of a water utility or at the point of use: *Legionella* spp. in water system, drinking water.

⁺⁺ Deficiency 99. Unknown/Insufficient information.

^{\$§} Multiple deficiency classifications were assigned to three outbreaks. One outbreak had deficiency 2, 3 one had 3, 4, and one had 5a, 7 (deficiency in building/ home-specific water treatment after the water meter or property line).

^{¶¶} Deficiency 13a. Current treatment processes not expected to remove a chemical contaminant: ground water.

*** Deficiency 2. Drinking water, contamination of water at/in the water source, treatment facility, or distribution system: untreated ground water.

⁺⁺⁺ Deficiency 4. Drinking water, contamination of water at/in the water source, treatment facility, or distribution system: Distribution system deficiency, including storage (e.g., cross-connection, backflow, and contamination of water mains during construction or repair).

§§§ Deficiency 6. Drinking water, contamination of water at points not under the jurisdiction of a water utility or at the point of use; plumbing system deficiency after the water meter or property line (e.g., cross-connection, backflow, or corrosion products).

¶¶ Deficiency 3. Treatment deficiency (e.g., temporary interruption of disinfection, chronically inadequate disinfection, or inadequate or no filtration).

interruptions in water service, illnesses, and persistent community concern about drinking water quality. Outbreaks in community water systems can trigger large and complex public health responses because of their potential for causing communitywide illness and decreasing the availability of safe water for community members, businesses, and critical services (e.g., hospitals). These outbreaks highlight the importance of public health and water utility preparedness for emergencies related to contamination from pathogens, chemicals, and toxins.

Legionella continues to be the most frequently reported etiology among drinking water-associated outbreaks (4). All of the outbreak-associated deaths reported during this surveillance period as well as all of the outbreaks reported in hospital/ health care settings or long-term care facilities, were caused by Legionella. A review of 27 Legionnaires' disease outbreak investigations in which CDC participated during 2000-2014 identified at least one water system maintenance deficiency in all 23 investigations for which this information was available, indicating that effective water management programs in buildings at increased risk for Legionella growth and transmission (e.g., those with more than 10 stories or that house susceptible populations) can reduce the risk for Legionnaires' disease (5,6). Although Legionella was detected in drinking water, multiple routes of transmission beyond ingestion of contaminated water more likely contributed to these outbreaks, such as aerosolization from domestic or environmental sources. Cryptosporidium was the second most common cause of both outbreaks and illnesses, demonstrating the continued threat from this chlorine-tolerant pathogen when drinking water supplies are contaminated. Existing drinking water regulations and filtration systems targeted to control Cryptosporidium help protect public health in community water systems that are primarily served by surface water sources or groundwater sources under the influence of surface water (7). Through the Epidemiology and Laboratory Capacity for Infectious Diseases (ELC) Cooperative Agreement, CDC has recently begun a laboratory-based cryptosporidiosis surveillance system in the United States, CryptoNet, to better track Cryptosporidium transmission and rapidly identify outbreak sources through molecular typing (8). The cyanobacterial toxin microcystin caused the largest reported toxin contamination of community drinking water in August 2013 and September 2014 and was responsible for extensive community and water disruptions. In June 2015, the Environmental Protection Agency released specific health advisory guidance for microcystin concentrations in drinking water (9). The contamination of a community drinking water supply with 4-metholcyclohexanementanol (MCHM) also illustrates the importance of source water protection from chemicals and toxins (2).

The findings in this report are subject to at least three limitations. First, 17% of drinking water-associated outbreak reports could not be assigned a specific deficiency classification other than "unknown or insufficient information," because of a lack of information. Furthermore, the deficiency classification most frequently reported ("presence of Legionella in drinking water systems") does not provide insight into the specific factors contributing to Legionella amplification and transmission. Second, the detection and investigation of outbreaks might be incomplete. Because of universal exposure to water, linking illness to drinking water is inherently difficult through traditional outbreak investigation methods (e.g., case-control and cohort studies) (10). Finally, reporting capabilities and requirements vary among states and localities. Therefore, outbreak surveillance data likely underestimate actual occurrence of outbreaks and should not be used to estimate the actual number of outbreaks or cases of waterborne disease.

Public health surveillance is necessary to detect waterborne disease and outbreaks, and to continue to monitor health trends associated with drinking water exposure. Despite resource constraints, 19 states reported drinking water-associated outbreaks for 2013-2014 compared with 14 for the previous reporting period (4). In this reporting cycle, more reported outbreaks and cases were caused by parasites and chemicals than by non-Legionella bacteria, and more cases were reported from community systems than from individual systems. Most of the outbreaks and illnesses reported in this period were in community systems, which serve larger numbers of persons; outbreaks in these systems can sicken entire communities. Although individual, private water systems likely serve fewer persons than community systems, they can still result in relatively large numbers of illnesses. One outbreak reported during 2013-2014 in an individual system led to 100 estimated illnesses associated with a wedding. The public health challenges highlighted here underscore the need for rapid detection, identification of the cause, and response when drinking water is contaminated by infectious pathogens, chemicals, or toxins to prevent and control waterborne illness and outbreaks.

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Conflict of Interest

No conflicts of interest were reported.

Summary

What is already known about this topic?

Waterborne disease and outbreaks associated with drinking water continue to occur in the United States. CDC collects data on waterborne disease outbreaks submitted from all states and territories through the National Outbreak Reporting System.

What is added by this report?

During 2013–2014, a total of 42 drinking water–associated outbreaks were reported to CDC, resulting in at least 1,006 cases of illness, 124 hospitalizations, and 13 deaths. *Legionella* was responsible for 57% of outbreaks and 13% of illnesses, and chemicals/toxins and parasites together accounted for 29% of outbreaks and 79% of illnesses. Eight outbreaks caused by parasites resulted in 289 (29%) cases, among which 279 (97%) were caused by *Cryptosporidium* and 10 (3%) were caused by *Giardia duodenalis*. Chemicals or toxins were implicated in four outbreaks involving 499 cases, with 13 hospitalizations, including the first outbreaks associated with algal toxins.

What are the implications for public health practice?

Continued public health surveillance is necessary to detect waterborne disease and monitor health trends associated with drinking water exposure. When drinking water is contaminated by infectious pathogens, chemicals, or toxins, public health agencies need to provide rapid detection, identification of the cause, and response to prevent and control waterborne illness and outbreaks. Effective water management programs in buildings at increased risk for *Legionella* growth and transmission can reduce the risk for disease from drinking water pathogens.

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Outbreaks Associated with Untreated Recreational Water — California, Maine, and Minnesota, 2018–2019

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Outbreaks associated with fresh or marine (i.e., untreated) recreational water can be caused by pathogens or chemicals, including toxins. Voluntary reporting of these outbreaks to CDC's National Outbreak Reporting System (NORS) began in 2009. NORS data for 2009-2017 are finalized, and data for 2018–2019 are provisional. During 2009–2019 (as of May 13, 2020), public health officials from 31 states voluntarily reported 119 untreated recreational water-associated outbreaks, resulting at least 5,240 cases; 103 of the outbreaks (87%) started during June-August. Among the 119 outbreaks, 88 (74%) had confirmed etiologies. The leading etiologies were enteric pathogens: norovirus (19 [22%] outbreaks; 1,858 cases); Shiga toxin-producing Escherichia coli (STEC) (19 [22%]; 240), Cryptosporidium (17 [19%]; 237), and Shigella (14 [16%]; 713). This report highlights three examples of outbreaks that occurred during 2018-2019, were caused by leading etiologies (Shigella, norovirus, or STEC), and demonstrate the wide geographic distribution of such outbreaks across the United States. Detection and investigation of untreated recreational water-associated outbreaks are challenging, and the sources of these outbreaks often are not identified. Tools for controlling and preventing transmission of enteric pathogens through untreated recreational water include epidemiologic investigations, regular monitoring of water quality (i.e., testing for fecal indicator bacteria), microbial source tracking, and health policy and communications (e.g., observing beach closure signs and not swimming while ill with diarrhea).

California

On July 22, 2019, the California Department of Public Health was notified of three cases of shigellosis in persons who reported playing in the Santa Ana River, a waterway spanning 100 miles through southern California. The department identified this exposure in other shigellosis cases and, in total, identified 24 cases with closely related isolates (within 0–2 alleles by core-genome multilocus sequence typing) of *Shigella sonnei*. Among 19 ill persons for whom epidemiologic data were available, 16 reported that during July 6–August 5 they played in a swim area in a shallow portion of the river where water quality was not regularly monitored. Two of the 16 ill persons also reported swallowing river water. No other

common risk factors were identified. The median age of these 16 ill persons was 7 years (range = 1-20 years); seven were female. Two of 15 ill persons for whom clinical data were available were hospitalized; none died. Date of symptom onset ranged from July 6 through August 7. In response to the outbreak, local public health officials closed public access to the swim area during August 8–15. Surface water samples were collected upstream, downstream, and at the swim area and tested for *E. coli*, a bacterial indicator of fecal contamination. The concentration of *E. coli* ranged from 350 through 1,600 most probable number/100 mL at these sites.* Investigation into possible sources of fecal contamination upstream and at the swim area did not definitively identify an outbreak source. No additional cases were identified after public access to the swim area was reopened on August 15.

Maine

On July 6, 2018, the Maine Center for Disease Control and Prevention received a report that multiple persons were ill with gastrointestinal symptoms after visiting Woods Pond Beach in Bridgton, Maine. Town officials in Bridgton closed the public beach during July 6-10. The agency used social media to identify persons who visited the pond during July 1–6, interviewed 34 heads of household, and completed surveys for 148 household members. A total of 139 persons reported visiting the pond during this period, 97 (70%) of whom reported illness. Among these 97 ill persons, 41 (42%) were male; among the 95 ill persons for whom age data were available, the median age was 12 years (range = 1-73 years). The median incubation period was 38 hours (range = 8-139 hours); the median symptom duration, reported for 91 cases, was 24 hours (range = 3-96 hours). Vomiting was reported by 78 (80%) of 97 ill persons. Visitors who reported swallowing pond water or going under water (a potential marker for swallowing water) were approximately three times more likely to be ill than were those who did not (relative risk = 3.19; 95% confidence interval [CI] = 1.69-6.05). Two of the stool specimens collected from

^{*} Most probable number is a method used to estimate the concentration of viable bacteria in water. All samples exceeded the Environmental Protection Agency (EPA)–recommended Beach Action Values of 190–235 colony forming units (CFU)/100mL for freshwater. Beach Action Values are EPA's suggested "do not exceed" value for beach advisory purposes.

four ill persons tested positive for norovirus genogroup I. Based on these test results and the reported symptomology, norovirus was thought to be the outbreak etiology. The source of water contamination was undetermined. No additional cases were reported after the beach reopened to swimmers on July 11.

Minnesota

On August 13, 2019, Minnesota Department of Health (MDH) epidemiologists identified three cases of STEC infection in persons who reported swimming at a public lake. Illness onset occurred during August 2-4. MDH notified park and recreation board officials of the cases on August 13 and advised them to close the lake to swimmers. MDH used social media to distribute a survey and identified 69 total cases, including four laboratory-confirmed STEC O145:H28 infections with closely related isolates (within 0-2 single nucleotide polymorphisms by whole genome sequencing). Dates of symptom onset ranged from July 18 through August 16. The median age of ill persons was 29 years (range = 1-65 years); 55 (80%) were female. Among the 24 (35%) ill persons who visited the beach only once, exposure dates ranged from July 16 through August 11. The two factors significantly associated with illness were swallowing lake water (odds ratio = 3.80; 95% CI = 1.17–12.38) and age ≤10 years (odds ratio = 2.90; 95% CI = 1.57–5.35). No hospitalizations or cases of hemolytic uremic syndrome were reported. The beach was monitored weekly for E. coli throughout the summer, but no test results exceeded Minnesota's recreational water criteria during April–October.[†] No evidence of a point source of fecal contamination was identified; however, 15 visitors and four lifeguards reported continuing to swim or work in the lake while ill. No additional cases were reported after the beach reopened to swimmers on September 5.

Discussion

Shigella, norovirus, STEC, and other enteric pathogens can be transmitted when persons ingest untreated recreational water contaminated with feces or vomit. Swimmers can contaminate water in untreated recreational water venues (e.g., lakes, oceans, and rivers) if they have a fecal or vomit incident in the water. Enteric pathogens can also be introduced into untreated recreational water venues by stormwater runoff and sewage system overflows and discharges. Other potential sources of fecal contamination and enteric pathogens include leaks from septic or municipal wastewater systems, dumped boating waste, and animal waste in or near swim areas.

Whereas the detection of Shigella and norovirus in untreated recreational water is indicative of human contamination, the detection of STEC does not necessarily indicate human contamination. Because E. coli and enterococci are part of the normal intestinal flora of humans and other animals, beach managers monitor levels of these bacteria as indicators of fecal contamination as recommended by the Environmental Protection Agency's 2012 recreational water quality criteria (1). Monitoring is conducted to detect changes in fecal contamination of water and not to indicate the presence of pathogens (2-4). For this reason, fecal indicator data alone cannot implicate the water as the route of outbreak exposure or identify the source of water contamination. This is particularly problematic for certain pathogenic strains of E. coli, such as E. coli O157:H7, which can persist in the sediment and be resuspended in the water but is not detected by most generic E. coli water tests.

In the outbreaks described in this report, the sources of contamination of the recreational waters were not definitively identified. Molecularly based microbial source tracking methods can be used to identify the host genus contributing to fecal contamination detected in water, which can inform more targeted environmental investigations and control measures (5). For example, identifying the host genus (e.g., horses) can help inform and optimize efforts to mitigate exposure (e.g., redesigning horse trails near untreated recreational water venues) to prevent outbreaks. Investigations into environmental influences include, but are not limited to, sanitary inspection of septic systems, identification of agricultural animal waste runoff or discharge, monitoring of wildlife activity in public areas, and identification of improper disposal of solid waste.

Multiple factors could hinder detection and investigation of outbreaks associated with untreated recreational water venues. First, persons often travel >100 miles to swim in lakes, oceans, and rivers (6). If swimmers become ill after returning to homes in multiple public health jurisdictions, identifying an outbreak can be difficult. Second, not all jurisdictions include questions about exposure to recreational water in their investigations of cases of illness caused by enteric pathogens. Third, issues with response activities (e.g., collection of water samples and decision-making about closures) might arise among agencies within the same jurisdiction (e.g., public health and natural resources agencies) or among jurisdictions if the outbreak source (i.e., untreated recreational water venue) is in multiple jurisdictions.

In addition to monitoring the level of fecal indicator bacteria at beaches, beach managers can promote healthy swimming by establishing policies that allow lifeguards to perform alternate duties that do not require them to enter the water if they are ill with diarrhea. This is equivalent to CDC recommendations for operators of public treated recreational water venues (e.g.,

[†] Minnesota recreational water criteria for freshwater call for a monthly geometric mean concentration of <126 CFU *E. coli*/100 mL water. For culturable *E. coli*, EPA criteria are a geometric mean concentration of 126 CFU/100mL and statistical threshold value of 410 CFU/100mL in freshwater.

Summary

What is already known about this topic?

Untreated recreational water–associated outbreaks can be caused by pathogens or chemicals, including toxins, in freshwater (e.g., lakes) or marine water (e.g., oceans).

What is added by this report?

This report highlights examples of untreated recreational water–associated outbreaks that occurred during 2018 or 2019, were caused by *Shigella* (California), norovirus (Maine), or Shiga toxin–producing *Escherichia coli* (Minnesota), the leading causes of such outbreaks, and demonstrate the wide geographic distribution of such outbreaks.

What are the implications for public health practice?

Swimmers should observe beach closure signs and water quality advisories, not swim in water made cloudier by heavy rain, not swim while ill with diarrhea, not swallow recreational water, and keep sand out of their mouths.

swimming pools) (7). Creating a workplace environment where employees feel comfortable disclosing that they are ill with diarrhea without fearing potential loss of wages or even work is important to the success of such policies. Because of the multiple potential sources of fecal contamination, beach managers and public health officials should educate swimmers and parents of young swimmers about steps they can take to minimize risk of infection from enteric pathogens (https:// www.cdc.gov/healthywater/swimming/oceans-lakes-rivers/ visiting-oceans-lakes-rivers.html). These healthy swimming steps include observing beach closure signs or water quality advisories because of elevated levels of fecal indicator bacteria, not swimming in water made cloudier by heavy rain, not swimming while ill with diarrhea, not swallowing the water;,and keeping sand out of mouths. In addition, for the 2020 summer swim season, CDC has released coronavirus disease 2019 (COVID-19) prevention considerations for beach managers (https://www.cdc.gov/coronavirus/2019-ncov/community/ parks-rec/public-beaches.html).

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[§]CDC's 2018 Model Aquatic Health Code (https://www.cdc.gov/mahc/ pdf/2018-MAHC-Code-Clean-508.pdf) element 6.3.4.7.1 states "Supervisors shall not permit employees who are ill with diarrhea to enter the water or perform in a qualified lifeguard role."

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Public Veterinary Medicine: Public Health

Compendium of Measures to Prevent Disease Associated with Animals in Public Settings, 2017

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The National Association of State Public Health Veterinarians Animal Contact Compendium Committee

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Preface

The Compendium of Measures to Prevent Disease Associated with Animals in Public Settings has been published by the NASPHV and the CDC since 2005.¹⁻³ This compendium provides standardized recommendations for public health officials, veterinarians, animal venue operators, animal exhibitors, visitors to animal venues and exhibits, teachers, camp operators, and others concerned with control of disease and with minimizing health risks associated with animal contact in public settings. The report has undergone several revisions, and this document updates information provided in the 2013 compendium.³

I. Introduction

Contact with animals in public settings (eg, fairs, educational farms, petting zoos, and schools) provides opportunities for entertainment and education. The NASPHV understands the positive benefits of human-animal contact. However, an inadequate un-

ABBREVIATIONS

NASPHV National Association of State Public Health Veterinarians STEC Shiga toxin-producing Escherichia coli derstanding among animal exhibitors and visitors in regard to disease transmission and animal behavior can increase the likelihood of infectious disease exposures, injuries, and other health problems among visitors in these settings. Zoonotic diseases (ie, zoonoses) are diseases shared between animals and humans; many of these diseases are potentially transmitted from animals to people in public animal contact venues (Appendix I). Of particular concern are instances in which zoonotic disease outbreaks result in numerous people becoming ill. During 1991 through 2005, the number of enteric disease outbreaks associated with animals in public settings increased.⁴ During 2010 through 2015, approximately 100 human infectious disease outbreaks involving animals in public settings were reported to the CDC (unpublished data, 2017). Such outbreaks have substantial medical, public health, legal, and economic effects.

Although completely eliminating risks from animal contact is not possible, this report provides recommendations for minimizing associated disease and injury. The NASPHV recommends that local and state public health, agricultural, animal health, wildlife, and environmental agencies use these recommendations to establish their own guidelines or regulations for reducing the risk for disease from human-animal contact in public settings. Public contact with animals is permitted in numerous types of venues (eg, animal displays, petting zoos, animal swap meets, pet stores, feed stores, zoological institutions, nature parks, circuses, carnivals, educational farms, livestock birthing exhibits, agricultural fairs, childcare facilities or schools, camps, agritourism venues, live animal markets, and wildlife photo opportunity settings). Managers of these venues should use the information in this report in consultation with veterinarians, public health officials, state and local agriculture officials, or other professionals to reduce risks for disease transmission.

Guidelines to reduce risks for disease from animals in health-care facilities, veterinary facilities, and various other occupational settings as well as from service animals (eg, guide dogs) have been developed.⁵⁻¹² Although not specifically addressed here, the general principles and recommendations in this report are applicable to these settings.

II. Methods

The NASPHV periodically updates the recommendations to prevent disease associated with animals in public settings. To revise the 2013 compendium,³ the NASPHV Animal Contact Compendium Committee members and external consultants met in Atlanta from October 4 through 6, 2016. The revision process included reviewing literature pertaining to outbreaks and diseases associated with animals in public settings since the previous compendium was published; examining reports of animal contact-associated enteric and nonenteric disease outbreaks from the CDC National Outbreak Reporting System as well as from CDC subject matter experts and state public health veterinarians; reviewing specific input solicited from NASPHV members and committee consultants; and evaluating publications and presentations from experts on specific topics of relevance to the compendium revision process. A committee consensus was required to add or modify existing language or recommendations. The 2017 recommendations reported here have been updated with new information and data on zoonotic disease outbreaks and prevention measures.

III. Background

A. Infectious diseases associated with animals in public settings

1. Diseases transmitted by direct or indirect animal contact

One of the most common routes of disease transmission from animals to people is direct physical contact with the animal, which includes touching, holding, kissing, being bitten, and being scratched. Disease transmission also occurs through indirect contact with an animal through contact with a surface contaminated by the animal's saliva, blood, urine, nasal secretions, feces, or other bodily fluids.

a. Enteric (intestinal) diseases

In 2012, a group of investigators estimated the burden of enteric illness attributable to animal contact in the United States.¹³ The pathogens included in that study were Campylobacter spp, Cryptosporidium spp, nontyphoidal Salmonella enterica, STEC O157:H7, non-O157 STEC strains, Listeria monocytogenes, and Yersinia enterocolitica. The investigators estimated that 445,213 illnesses, 4,933 hospitalizations, and 76 deaths caused by these pathogens occurred annually as a result of animal contact in all (ie, private and public) settings. Pathogens with the highest proportion of cases attributable to animal contact were Campylobacter spp (17%), Cryptosporidium spp (16%), nontyphoidal S enterica (11%), non-O157 STEC strains (8%), and STEC O157:H7 (6%).

Enteric bacteria and parasites pose the highest risk for human disease from animals in public settings.¹⁴ Enteric disease outbreaks among visitors to fairs, farms, petting zoos, and other public settings are well documented.^{15–40} Cattle, sheep, or goats^{15,17,20,21,26–28,30,31,34,36,38,40} have typically been identified as sources for infection; however, live poultry,^{16,41–48} rodents,^{49–53} reptiles,^{33,54–60} amphibians,⁶¹ and other domestic^{4,62,63} and wild⁴ animals also are established sources. Animals that appear healthy can carry pathogens that cause illness in people. A small number of pathogens is often enough to cause illness.^{64–68}

Outbreaks as well as sporadic infections with nontyphoidal S enterica have been associated with animal contact. Animals that present a high risk for human Salmonella spp infections and have been implicated as sources of outbreaks of human illness include poultry (eg, chicks, chickens, and ducklings)^{16,41-48,69-72}; reptiles (eg, turtles, snakes, or lizards)^{33,54-60,73-80}; and amphibians, especially frogs.^{61,81-83} From 1990 through 2014, 53 disease outbreaks linked to live poultry in the United States have been documented.^{16,43,69,84} Some of the ill persons in those outbreaks reported contact with live poultry at feed stores,^{16,43,69} schools or daycare facilities,^{16,41,69} fairs,⁶⁹ petting zoos,⁶⁹ and nursing homes (CDC, unpublished data, 2010). Since 2014, an additional 14 outbreaks and approximately 1,200 cases of illness associated with exposure to live poultry have been documented (CDC, unpublished data, 2017). Preventive measures at the hatchery level and in agricultural feed stores, along with proper handling of live poultry by poultry owners, can help prevent salmonellosis.42

Reptiles and amphibians can carry *Salmonella* spp and have been linked to numerous outbreaks of human illness. Despite laws banning their sale or distribution in the United States, small turtles (those with shells that measure < 4 inches long) continue to be distributed. From 2006 through 2014, 15 multistate outbreaks of salmonellosis, comprising 921 reported illnesses (including a fatal case in an infant), have been linked to contact with small turtles and their habitats.⁵⁶ Salmonella Typhimurium infections have been linked to contact with African dwarf frogs (an aquatic amphibian), their habitats, or water from their habitats. Ill people included those who reported acquiring frogs at carnivals, pet stores, and other retail stores.^{61,82} Activities associated with increased risk of zoonotic disease transmission from turtles, frogs, and other aquatic animals include direct and indirect contact with the animal, tank, water, filtration equipment, or other tank contents. These findings have implications for risk of infection from aquatic exhibits (eg, aquariums and aquatic touch tanks).

Other animals associated with outbreaks of Salmonella spp infections in people include hedgehogs^{63,85} and rodents such as hamsters, mice, and guinea pigs.⁴⁹⁻⁵³ In all animal species that might harbor Salmonella organisms, it is possible for animals that appear healthy and clean to carry and shed the bacteria in their excreta, which can contaminate their fur, hair, feathers, scales, or skin. Salmonella spp can also be present in environments where animals or animal excreta, fur, hair, feathers, scales, or skin are present (eg, barns, petting zoos, school classrooms, and pet stores). Pet food and treats, which may be present in public settings such as pet stores, fairs, and school classrooms, have been confirmed as sources of human salmonellosis in several instances.86-92

Case-control studies^{79,93-96} also have associated sporadic enteric infections (ie, those not linked to an outbreak) with animals including reptiles, amphibians, farm animals, and cats. For example, a study⁹⁵ of sporadic Escherichia coli O157:H7 infections in the United States determined that people who became ill were more likely to have visited a farm with cows than were people who did not become ill. Other investigations identified associations between E coli O157:H7 infection and visiting a farm⁹⁷ or living in a rural area.⁹⁸ Results of studies^{99,100} of cryptosporidiosis in people found that contact with cattle is a risk factor for infection. Another study¹⁰¹ identified consumption of raw milk and contact with farm animals among the factors associated with Campylobacter infection.

(1) Animals shedding enteric pathogens. Animals carrying human enteric pathogens frequently have no signs of illness but can still shed the organisms in feces.¹⁰² Removing ill animals, especially those with diarrhea, from public contact is necessary, but this step alone is not sufficient to protect the health of people and other animals. The fact that some pathogens can be shed intermittently and survive for months or years in the environment,¹⁰³⁻¹⁰⁷ as well as the limitations of laboratory testing, makes attempts to identify and remove infected animals unreliable as means of eliminating the risk for transmission. Antimicrobial treatment cannot reliably eliminate infection or prevent shedding, and it does not protect against reinfection. Antimicrobial use in animals can also prolong shedding and contribute to antimicrobial resistance.108-110

Disease transmission at animal exhibits can be influenced by multiple factors. Stress induced by transportation, confinement, physical crowding, and increased handling increases the likelihood of animals shedding pathogens.¹¹¹⁻¹¹⁷ Commingling increases the probability that the shed pathogens will infect other animals.¹¹⁸ Young animals, which are frequently included in settings such as petting zoos, farm visits, and educational programs for children, have a higher prevalence of shedding enteric pathogens such as E coli O157:H7 than do mature animals.¹¹⁹⁻¹²¹ Animal shedding of E coli O157:H7 and Salmonella organisms is highest in the summer and fall,^{116,121} when traveling animal exhibits, agricultural fairs, and farm or petting zoo visits are commonly scheduled.

(2) Transmission of enteric pathogens to people. Enteric pathogens are primarily transmitted by the fecal-oral route. Because animal fur, hair, feathers, scales, skin, and saliva harbor fecal organisms,¹²² transmission can occur when people pet, touch, feed, or are licked by animals. Exposure to contaminated materials such as animal bedding, environmental surfaces, clothing, and shoes has also been associated with transmission of pathogens.^{29,33,35,82,123,124} In addition, illness has resulted from fecal contamination of food,^{24,125} unpasteurized juice,¹²⁶ unpasteurized milk,^{19,127-130} and drinking water.¹³¹⁻¹³⁴

Young children (ie, < 5 years of age) are considered to be at greater risk for acquiring enteric pathogens from animals than most adults are. One study¹³⁵ found that certain risk behaviors for disease transmission such as physical contact with animals and hand-to-face contact were more common in children than in adults during petting zoo visits. In addition, young children, elderly adults, and people with weakened immune systems have an increased risk for developing severe illness, compared with healthy individuals outside these groups, when they do become infected.¹³⁶ Finally, attendees or visitors to animal venues are not the only persons potentially exposed to pathogens; livestock exhibitors have also become infected with *E coli* O157:H7 in outbreaks at fairs.³⁵

(3) Environmental exposures to enteric pathogens. Disease transmission can occur in the absence of direct animal contact if a pathogen is present in the environment. Outbreaks of enteric illness have been associated with exposure to environments after animals were removed,137 dust in the environment,¹²⁴ touching or stepping in manure,³² and falling down or sitting on the ground in a petting zoo.³² Ill people have also reported having contact with manure on a fence without having touched an animal.²² In an outbreak of E coli O157:H7 in 2004, the outbreak strain was isolated from shavings collected from a baby stroller and from the shoes of petting zoo visitors.³²

Enteric pathogens can persist in contaminated environments for long periods. For example, *E coli* O157:H7 can survive in soil for months.^{22,35,102,103,105,107,124,a} In a 2009 *E coli* O157:H7 outbreak associated with rodeo attendance, the outbreak strain was isolated from the rodeo grounds 90 days after the end of the event.²² Other outbreaks have also demonstrated long environmental persistence of pathogens, including *E coli* O157:H7 recovered from sawdust on the floor of an animal barn up to 42 weeks after a fair.¹²⁴

b. Internal parasites

Animal parasites can infect people who ingest materials contaminated with animal feces or who ingest or otherwise come into contact with contaminated soil. Exposure to parasites in public settings has led to outbreaks including toxoplasmosis at a riding stable^{138,139} and cutaneous larva migrans at a children's camp.¹⁴⁰ The presence of *Toxocara* eggs in public parks indicates a potential risk of toxocariasis to people in public settings.141-143 Exposure to Baylisascaris procyonis, raccoon roundworms, in public settings is also possible; a kinkajou purchased from a pet store was found to be infected with *B procyonis*,¹⁴⁴ and antibodies to *B procyonis* were detected in 7% of a sample of wildlife rehabilitators from the United States and Canada.145

c. Animal bites and scratches

(1) Rabies. People who have contact with rabid mammals can be exposed to rabies virus through a bite or when mucous membranes or open wounds become contaminated with infected saliva or nervous tissue. Although no human deaths due to rabies incurred through animal contact in public settings have been reported in the United States, multiple rabies exposures have occurred, requiring extensive public health investigations and medical follow-up. Thousands of people have received rabies postexposure prophylaxis after being exposed to rabid or potentially rabid animals or animal carcasses. Animals involved in reported exposures have included bats, raccoons, cats, goats, bears, sheep, horses, foxes, and dogs, at various venues: an urban public park,146 a pet store,147 a county fair,62,148 petting zoos,^{149,150} schools,⁶² rodeo events,⁶² a horse show,151 and summer camps.152 Important public health and medical care challenges associated with potential mass rabies exposures include difficulty in identifying and contacting individuals who are potentially at risk, correctly assessing exposure risks, and providing timely medical prophylaxis when indicated. Human infection with rabies virus is almost always fatal once clinical signs of rabies appear, and prompt assessment and appropriate treatment are critical.¹⁵³

(2) Other bite-related and scratch-related infections. Infections from animal bites and scratches are common; some may require extensive treatment or hospitalization. Bacterial pathogens associated with animal bites include Pasteurella spp, Francisella tularensis,^{154,155} Staphylococcus spp, Streptococcus spp, Capnocytophaga canimorsus, Bartonella henselae (the etiologic agent of cat scratch disease), and Streptobacillus moniliformis (the etiologic agent of rat bite fever).¹⁵⁶ Some monkey species (especially macaques) can be infected with B virus (formerly known as cercopithecine herpesvirus 1). Infected monkeys may have no clinical signs or have mild oral lesions; however, fatal meningoencephalitis has been reported in human patients infected through monkey bites or by exposure to bodily fluids.157,158

d. Skin infections

Skin contact with animals in public settings can also result in human infection. Cases of ringworm have been reported among animal exhibitors.¹⁵⁹ Infection with parapox virus (the causative agent of contagious ecthyma, also described as orf or sore mouth in sheep and goats) has developed in children after contact with sheep in a public setting.¹⁶⁰ Transmission of pox viruses to people in public settings also has been described, including cowpox virus in a circus animal keeper,¹⁶¹ cowpox virus in people who handled pet rats at a pet store,¹⁶² and monkeypox among people who contacted infected prairie dogs at a childcare center.^{163,164} Contact with aquatic animals and their environment has also been implicated in cutaneous infections,¹⁶⁵ such as *Mycobacterium marinum* infections in people who owned or had cleaned fish tanks.^{166,167}

e. External parasites

Ectoparasites and endoparasites can be spread to people who interact with exhibit animals. Sarcoptes scabiei is a skin mite with different host-specific variants that infest people and animals, including swine, dogs, cats, foxes, cattle, and coyotes.^{168,169} Although human infestation by animal variants is selflimiting, skin irritation and itching might occur for multiple days and can be difficult to diagnose.^{169,170} Bites from avian mites have also been reported in association with gerbils in schoolrooms.¹⁷¹ Ectoparasite control should be considered in animals in public settings to reduce the risk of human exposure to flea and tick-borne diseases.

2. Diseases transmitted through droplets or aerosols

Generation of infectious droplets or aerosols and subsequent contamination of the environment is an important risk for indirect transmission of disease in public settings. These droplets or aerosols can include infectious agents from animals' respiratory tracts, reproductive fluids, or other sources. Cleaning procedures (eg, pressure washing^{10,172}) or dust raised in animal environments, including dust generated from activities such as sweeping and leaf blowing, can lead to infectious aerosols in the immediate environment and surrounding areas.

a. Influenza

Transmission of influenza A viruses between people and animals has increasingly important implications for human-animal interactions in public settings. Influenza viruses that normally circulate in pigs are called variant viruses when they are found in people.¹⁷³ Although pigs with influenza can become ill, it has also been shown that apparently healthy pigs can carry influenza viruses.¹⁷⁴ Sporadic cases and small clusters of human infections with variant influenza viruses have been reported since the 1970s^{175,176}; most of these cases were associated with direct or indirect exposure to swine at agricultural fairs.¹⁷⁷⁻¹⁷⁹ From July 2011 through October 2012, > 300 confirmed infections with influenza A (H3N2) variant viruses were reported across 10 states.^{174,180-184} Most infections occurred in children who reported direct contact with swine at agricultural fairs. Although viruses that normally circulate in birds (avian influenza A viruses) usually do not infect humans, rare cases of human infection with these viruses have been reported.¹⁸⁵ Transmission of human influenza viruses from people to swine^{186,187} and other species also has been reported. For example, in 1998, a new strain of influenza A (H3N2) virus derived from human, avian, and classical swine influenza A viruses emerged and became established in swine.¹⁸⁸

b. Tuberculosis

Tuberculosis can be a concern in certain animal settings; however, the risk is primarily for close contacts, including handlers, of certain animal species,¹⁸⁹⁻¹⁹¹ particularly elephants.^{192,193} Guidelines have been developed regarding removal of tuberculosis-infected animals from public settings.¹⁹⁴

c. Q fever

Live-birthing exhibits, usually involving cattle, pigs, goats, or sheep, are popular at agricultural fairs and farm visits. Although members of the public do not typically have direct contact with animals during birthing, contact with newborn animals and their dams may occur afterward. Numerous cases of illness related to Q fever have been linked to viewing of animal births.^{195,b} Leptospirosis, listeriosis, brucellosis, and chlamydiosis are other serious zoonotic diseases that can be acquired through contact with aborted fetuses, newborn animals, reproductive tissues, or associated fluids.⁶⁷

The causative agent of Q fever is the Coxiella burnetii bacterium; goats, sheep, and cattle are the most frequently implicated animal sources of human infections in the United States.¹⁹⁶ Although C burnetii infection can cause abortion in animals, it is often subclinical. High numbers of organisms shed in reproductive tissues, and fluids can become aerosolized during birthing, and inhalation of aerosolized organisms can lead to infection in people. Most individuals exposed to C burnetii develop an asymptomatic infection, but clinically apparent illness can range from an acute influenza-like illness to life-threatening endocarditis, as well as premature birth, stillbirth, and miscarriage in pregnant women.¹⁹⁷ In 1999, an outbreak of Q fever involving 95 confirmed cases of the disease and 41 hospitalizations was linked to goats and sheep giving birth at petting zoos in indoor shopping malls in Canada.^b Another O fever outbreak, in which > 30 human cases were reported in the Netherlands, was associated with public lambviewing days at a sheep farm in 2009.195

d. Chlamydophila psittaci infections

Chlamydophila psittaci infections are usually acquired from psittacine birds and cause respiratory disease in people.¹⁹⁸ Cases of human psittacosis have occurred among staff members at a zoological garden,¹⁹⁹ among people exposed to an aviary in a church,²⁰⁰ and among pet store staff and visitors.³⁹ On rare occasions, chlamydial infections acquired from sheep and birds have resulted in human maternal and fetal illness and death.²⁰¹⁻²⁰⁴

3. Factors influencing the risk of zoonotic disease transmission

a. Handwashing

Handwashing following contact with animals has been associated with decreased rates of illness during disease outbreaks associated with animals in public settings. The CDC was prompted to establish recommendations for enteric disease prevention associated with farm animal contact after 2 outbreaks of *E coli* 0157:H7 infections in 2000 in Pennsylvania and Washington.²⁰⁵ Risk factors identified in the Pennsylvania outbreak were contact with cattle and inadequate handwashing. It was found that handwashing facilities were limited and not configured for children.³⁶

In 1996, an outbreak of salmonellosis at a Colorado zoo resulted in 65 cases of the disease (primarily among children) associated with touching a wooden barrier around a temporary Komodo dragon exhibit. Children who were not ill were significantly more likely to have washed their hands after visiting the exhibit than children who were ill.³³

In a 2005 Florida outbreak of *E coli* O157:H7 infections,²⁵ both direct animal contact and contact with sawdust or shavings were associated with illness. The likelihood of illness was higher for people who reported feeding animals and lower for those who reported washing their hands before eating or drinking, compared with those who did not. Creating a lather decreased the likelihood of illness for individuals who used soap and water for handwashing; however, drying hands on clothing increased the likelihood of illness.^c

In 2 outbreaks of infection with multiple enteric pathogens that took place in 2000 through 2001 at a Minnesota children's farm day camp, washing hands with soap after touching a calf and washing hands before going home were associated with decreased likelihood for illness.²⁷ Risk factors for children who became ill included caring for an ill calf and getting a visible amount of manure on their hands.

Interventions that have been shown to improve hand hygiene compliance include having venue staff provide verbal reminders about hand hygiene to guests before they leave the animal area, use of larger signs with more prominent messages combined with staff actively offering hand sanitizer to visitors,²⁰⁶ and having a staff member present within or at the exit to the animal contact area.²⁰⁷ Although the use of hand sanitizers (with an alcohol concentration of 60% to 95%) can be effective at killing pathogens, it should be noted that washing hands with soap and water is still preferred because hand sanitizers do not work equally well for all classes of pathogens and might not work well when hands are heavily soiled or greasy.²⁰⁸

b. Facility design

The layout and maintenance of facilities and animal exhibits can increase or decrease the risk for infections.²⁰⁹ Factors that increase this risk include inadequate handwashing facilities,⁶² inappropriate flow of visitors, and incomplete separation between animal exhibits and food preparation and consumption areas.^{29,38,210} Other factors include structural deficiencies associated with temporary food service facilities, contaminated or inadequately maintained drinking water systems, and poorly managed sewage or manure containment and disposal processes.^{33,124,132-134,211} In one of the largest waterborne disease outbreaks in the United States (1999),^{132,133} approximately 800 suspected cases of infection with E coli O157:H7, Campylobacter spp, or both were identified among attendees at a New York county fair. In that outbreak, unchlorinated water supplied by a shallow well was used by food vendors to make beverages and ice.133

Temporary and seasonal animal exhibits and activities are particularly vulnerable to design flaws.^{25,33} Animal displays or petting zoos added to attract visitors to zoos, festivals, roadside attractions, farm stands, farms where people can pick their own produce, feed stores, and Christmas tree lots are examples of these types of exhibits. In 2004 and 2005, separate outbreaks of E coli O157 occurred at seasonal state fairs in North Carolina and Florida. Both of these outbreaks involved exposure to vendor-run temporary petting zoos.25 Inadequate handwashing facilities were reported for a temporary exhibit in British Columbia, Canada, where childcare facility and school field trips to a pumpkin patch with a petting zoo resulted in E coli O157:H7 infections.38 Running water and signs recommending handwashing were not available, and alcohol-containing hand sanitizers were placed at a height that was unreachable for some children.

Venues not designed for or accustomed to public events, such as working farms, wildlife rehabilitation facilities, animal adoption events, and animal shelters, might be less likely to have facilities adequately designed to accommodate visitors and to reduce the risk of exposure to zoonotic disease agents. Limitations that might lead to increased infection risk include lack of or inadequate handwashing stations and dedicated food service areas and inappropriate traffic flow patterns. Public access to animal waste areas in these venues might also be problematic.¹³⁷

c. Food contamination

Contamination of food products or food preparation areas secondary to animal contact has previously resulted in outbreaks. Food products contaminated with zoonotic pathogens have included unpasteurized apple cider,126 produce,24 and raw milk.19,62 Contamination from inadequate sanitation (eg, of hands, utensils, or equipment) can occur during food preparation or consumption. Venues in which food contamination contributed to human illness include summer camps²⁴ and an apple orchard.^d Large, multistate foodborne outbreaks of salmonellosis have been attributed to food preparers having had contact with live poultry prior to handling food products and subsequently contaminating those products.16,212 Additionally, consumption of food in an animal environment has been associated with illnesses. In a 2015 outbreak of E coli O157:H7 infections at a dairy event in Washington, crude attack rates were higher for individuals who were involved in activities where food was served in an animal barn.¹³⁷ Purchase of food at a farm visit²⁰⁵ and the consumption of sticky foods¹²⁵ (eg, ice cream and cotton candy) have also been associated with E coli O157:H7-related illnesses.

d. Other factors influencing disease transmission

Events at which people have prolonged close contact with animals, such as day camps and livestock exhibitions, pose a unique challenge with regard to disease prevention. Examples of events where prolonged contact has led to illness include an outbreak of *E coli* O157:H7 infections that occurred at a day camp where prolonged contact with livestock was encouraged.²¹³

Failure to properly implement disease-prevention recommendations has also contributed to recurrent outbreaks. Following an outbreak of cryptosporidiosis with 31 ill students at an educational farm program in Minnesota, specific recommendations (including use of coveralls and rubber boots when handling calves, supervised handwashing, and provision of hand sanitizer) were provided to teachers but were inadequately implemented.³¹ A subsequent outbreak occurred several months later, with 37 additional illnesses.³¹ Handwashing facilities and procedures were still inadequate, and coveralls and boots that were used were found to be dirty, cleaned infrequently, and handled without subsequent handwashing.

Other disease outbreaks have resulted from contaminated animal products used during school activities. Salmonellosis outbreaks associated with dissection of owl pellets in classes have occurred²¹⁴; in 1 such outbreak, risk factors for infection included inadequate handwashing, use of food service areas for the activity, and improper cleaning of contact surfaces. Students in a middle school science class were among those infected in a multistate salmonellosis outbreak associated with frozen rodents sold as snake food.⁵¹

B. Physical injuries caused by animals in public settings

Although infectious diseases are the most commonly reported health problems associated with animals in public settings, injuries caused by animals are also commonly reported, and these can result in infection as well as trauma. For example, dog bites are an important community problem for which specific guidelines have been written.²¹⁵ Injuries associated with animals in public settings include bites, kicks, falls, scratches, stings, crushing of extremities, and being pinned between an animal and a fixed object. Serious and fatal injuries have been associated with various venues and species including commercial stables (interaction with horses),²¹⁶ animal sanctuaries (tigers),²¹⁷ petting zoos (llamas),²¹⁸ photo opportunities (tigers and bison),^{217,219} schools (snakes),²²⁰ animal safaris (camels),²²¹ and dog parks (dogs).²²²

IV. Recommendations for Disease Prevention

A. Overview

Information, publications, and reports from multiple organizations were used to create the recommendations in this document.²²³⁻²²⁵ Although no US federal laws address the risk for transmission of pathogens at venues where animals and the public come into contact, some states regulate actions such as the provision of handwashing stations in some or all such settings.^{226,227}

Certain federal agencies and associations in the United States have developed standards, recommendations, and guidelines for reducing health risks associated with animal contact by the public. The Association of Zoos and Aquariums has accreditation standards requiring training of staff on the risks of zoonotic diseases, including those associated with public contact.²²⁸ The USDA licenses and inspects

certain animal exhibits in accordance with the Animal Welfare Act²²⁹; although these inspections primarily address humane treatment of animals, they also impact animal health and public safety. In 2001, the CDC issued recommendations to reduce the risk of infection with enteric pathogens associated with farm visits.²⁰⁵ The CDC has also issued recommendations for preventing transmission of Salmonella spp from reptiles, amphibians, and live poultry to people^{69,71,74,76,82,230} and provides educational posters in English and other languages online.²³¹ The Association for Professionals in Infection Control and Epidemiology and the Animal-Assisted Interventions Working Group have developed guidelines to address risks associated with the use of animals in health-care settings.^{8,11} The NASPHV has developed guidance and compendia of measures to reduce risks for human exposure to C psittaci, rabies virus, C burnetii, novel influenza A viruses, and zoonotic pathogens that veterinary personnel might be exposed to in an occupational setting.10,198,232-234

Studies^{135,206,207,235} in multiple localities have suggested that the recommendations provided in the present compendium are not completely implemented by members of the public and managers or employees of animal contact venues. Stakeholders should strive to achieve comprehensive implementation of the recommendations in this compendium, to help ensure that visitors can stay healthy and reduce the risk of zoonotic disease transmission while enjoying animals.

B. Applicable venues

The recommendations in this report were developed for settings in which direct animal contact is possible. These settings include farm visits, agritourism venues, petting zoos, school field trips, camps, agricultural fairs, feed stores, wildlife sanctuaries, animal swap meets, childcare centers and schools, and other settings. Contact with animals in public settings should only occur where measures are in place to reduce the potential for disease transmission or injuries. Incidents or problems should be investigated, documented, and reported.

C. Recommendations for local, state, and federal agencies

Agencies should encourage or require oversight to ensure compliance with recommendations at animal contact venues. The recommendations should be tailored to specific settings and incorporated into best practices, protocols, and regulations developed at the state or local level. Additional research should be conducted regarding the risk factors and effective prevention and control methods for health issues associated with animal contact. Additionally, communication and cooperation to ensure public health and safety extends beyond human, animal, and environmental health agencies and should include additional stakeholders such as professional associations, schools, private companies, and industry groups.

1. Dissemination of recommendations

This compendium should be disseminated to cooperative extension personnel, venue operators, farms that host public events, veterinarians, schools and daycares, associations and industry groups, and others associated with managing animals in public settings. Development of a complete list of public animal contact venues within a jurisdiction is encouraged to facilitate dissemination of these recommendations. Agencies should disseminate educational and training materials to venue operators and other stakeholders. Sample materials are available in a variety of media in the NASPHV Animals in Public Settings Toolkit, which is available electronically (www.nasphy. org/documentsCompendiumAnimals.html and www.cdc.gov/healthypets/specific-groups/ contact-animals-public-settings.html).236

2. Investigating and reporting outbreaks

To evaluate and improve these recommendations, surveillance activities for human infections associated with animal contact should be enhanced. Agencies should take the following steps:

- Conduct thorough epidemiological investigations of outbreaks using a one-health approach across human, animal, and environmental health sectors.
- Follow appropriate protocols for collection and laboratory testing of samples from people, animals, and the environment, including molecular subtyping of pathogen isolates.
- Include questions on disease report forms and outbreak investigation questionnaires about exposure to animals and their environments, products, and feed.
- Report outbreaks to state public health departments.
- Local and state public health departments should also report all outbreaks of enteric infections resulting from animal contact to the CDC through the National Outbreak Reporting System (www.cdc.gov/nors/).

D. Recommendations for animal exhibitors and venue operators

Staff and visitor education, attention to hygiene, and appropriate facility design as well as proper care and monitoring of animals and their enclosures are essential components for reduction of risks associated with animal contact in public settings. It is important to be aware of and follow local, state, and federal regulations regarding animals in public settings.

1. Education

Awareness of zoonotic disease risk is protective against illness in outbreaks.³² Therefore, educating visitors to public animal contact venues about the risk for transmission of diseases from animals to humans is a potential disease-prevention measure. Education is important not only at traditional animal venues like petting zoos, but also at farms and other venues where live animals are sold or distributed to the public. Even in welldesigned venues with operators who are aware of the risks for disease, outbreaks and injuries can occur when visitors do not understand the risks and therefore are less likely to apply diseaseprevention measures. Mail-order hatcheries, agricultural feed stores, and other venues that sell or display live poultry should provide health-related information to owners and potential owners. This should include information about the risk of acquiring Salmonella infection from contact with live poultry and measures to prevent such infections. Other venues that sell live animals. such as pet stores, should also provide educational materials to customers about the risk of illness and prevention of zoonotic infections. This is especially important for animals considered to have a high risk of transmitting disease to humans (eg, reptiles, amphibians, and live poultry). Evidence-based prevention messages and free educational materials are available in multiple formats and in multiple languages on the CDC Healthy Pets, Healthy People website (www.cdc. gov/healthypets/).

a. Operators and staff

Operators and staff should be aware that certain populations are more likely than others to develop serious illness from pathogens transmitted in animal contact settings. The risk of infection leading to serious illness is particularly high in children < 5 years of age. Other groups that have an increased degree of risk include people with waning immunity (eg, individuals ≥ 65 years of age), pregnant women, or people who are immunocompromised (eg, those with HIV-AIDS, without a functioning spleen, or receiving immunosuppressive treatments). Individuals considered to be at high risk for serious illness should take heightened precautions or avoid animal exhibits. In addition to thorough and frequent handwashing, heightened precautions could include avoiding contact with animals and their environments.

Venue operators and staff (all individuals involved with animal contact activity in any public setting) should take the following steps for public health and safety:

- Become familiar with and implement the recommendations in this compendium.
- Consult with veterinarians, state and local agencies, and cooperative extension personnel on implementation of the recommendations.
- Become knowledgeable about the risks for disease and injury associated with animals and be able to explain risk-reduction measures to staff members and visitors.

• Be aware of populations at high risk for disease and injury interacting with animals and of the presence of animals that pose a high risk for causing disease and injury within the venue.

Each of the following aspects should be taken into consideration in facility design and operation, educational messaging, and animal care and management:

- Direct public contact with ill animals is inappropriate for any audience.
- Children < 5 years of age should not have direct contact with animals that are considered likely to carry zoonotic pathogens (eg, preweaned calves, reptiles, amphibians, or live poultry).
- Children < 5 years of age are also at high risk for disease and injury from contact with other animals and should be supervised at all times to discourage handto-mouth activities (eg, nail biting and thumb sucking), contact with manure, and contact with soiled bedding.
- Individuals ≥ 65 years of age and those with weakened immune systems (eg, people with HIV-AIDS, without a functioning spleen, or receiving immunosuppressive treatment) also have a high risk of developing serious illness from contact with animals carrying zoonotic diseases.
- Pregnant women are at risk of stillbirth, miscarriage, and preterm delivery from certain pathogens that might be present in animal contact settings.
- Direct contact with venomous or otherwise dangerous animals (eg, venomous reptiles, nonhuman primates, or certain carnivores and other rabies reservoir species) should be completely prohibited (*See* the Animal Care and Management section for more information on these species)
- Live animals, especially reptiles, amphibians, and poultry, should not be given as prizes at fairs, carnivals, or other events.
- Ensure that visitors receive educational messages before entering an exhibit, including information that animals can cause injuries or carry germs that can cause serious illness, along with recommended prevention measures (Figure 1; Appendix 2¹⁻³).
- Provide information in a simple and easy-tounderstand format that is age appropriate and language appropriate.
- Provide information in multiple formats (eg, signs, stickers, handouts, and verbal information) and languages.
- Provide information to people arranging school field trips or classroom exhibits so they can educate participants and parents before the visit.
- Encourage compliance by the public with riskreduction recommendations, especially compli-



Some people are more likely to get sick: babies, toddlers, pregnant women, older adults, and those with weakened immune systems.

Stay Healthy:

- No food, drinks, baby bottles, pacifiers, toys, or strollers in animal areas.
- Watch kids & those with intellectual disabilities around animals.
- Make sure kids don't put their fingers or other things in their mouths.
- Wash hands with soap & water right after visiting the animals.



Figure I—Suggested sign or handout for use in safety education of visitors entering animal areas of petting zoos or other exhibits (available at www.nasphv. org/documentsCompendiumAnimals.html [accessed Sep 14, 2017]).

ance with handwashing procedures as visitors exit animal areas (Figure 2; Appendix 3).^{1-3,237}

- Ensure compliance with licensing and registration requirements under the Animal Welfare Act per USDA guidelines for dealers, exhibitors, transporters, and researchers.²²⁹
- Comply with local and state requirements for reporting animal bites or other injuries.
- b. Visitors

Visitors to animal exhibits and those participating in interaction activities of any kind should be presented with effective educational messages aimed at ensuring compliance with the following recommendations:

- Be aware that the risks associated with animal contact are higher among people of certain age groups and health conditions, especially children < 5 years of age, pregnant women, anyone \geq 65 years of age, and individuals with weakened immune systems, than for others.
- Supervise children properly at all times while in the presence of animals and areas with animal waste; prevent inappropriate contact with animals and sitting or playing on the ground.
- Practice proper hand hygiene, including washing hands immediately upon exit of the animal area and before any hand-tomouth activity or eating is done.
- Practice proper hand hygiene after any contact with shoes, strollers, or clothing that might have come in contact with animals, their waste, or their bedding.
- Report any animal bites or injuries promptly to the venue operator and to authorities per local or state law.
- Understand that certain diseases shared between animals and people can also pass from people to animals.
- 2. Facility design and use

Venues should be divided into 3 types of areas: nonanimal areas (where animals are not permitted, with the exception of service animals), transition areas (located at entrances and exits to animal areas), and animal areas (where animal contact is possible or encouraged; **Figure 3**).

a. Layout and traffic patterns

(1) Animal area considerations. The design of facilities and animal pens

should minimize the risk associated with animal contact (Figure 3), including limiting direct contact with manure and encouraging handwashing (Appendix 3). The design of facilities or contact settings might include double barriers to prevent contact with animals or contaminated surfaces except in specified animal interaction areas. Contact with fecal material or soiled bedding in animal pens increases risk of exposure to pathogens, and facility designs and policies should limit or prevent this type of exposure, especially to individuals who might be at high risk for infection.

Investigations of previous outbreaks have revealed that temporary exhibits are

Wash Hands When Leaving Animal Exhibits

WHO

Everyone, especially young children, older individuals, and people with weakened immune systems

WHEN

Always Wash Hands:

- After touching animals or their living area
- After leaving the animal area
- After taking off dirty clothes or shoes
- After going to the bathroom
- 🥘 Before preparing foods, eating, or drinking

HOW

- Wet your hands with clean, running water
- 諽 Apply soap



- Rub hands together to make a lather and scrub well, including backs of hands, between fingers, and under fingernails
- Rub hands at least 20 seconds. Need a timer? Hum the "Happy Birthday" song from beginning to end twice
- Rinse hands

Dry hands using a clean paper towel or air dry them. Do not dry hands on clothing

For more information, visit CDC's Healthy Pets, Healthy People website (www.cdc.gov/healthypets) and CDC's Handwashing website (www.cdc.gov/handwashing)

Figure 2—Suggested sign to encourage compliance with handwashing procedures as a means of reducing the possible spread of infectious disease (available in several languages at www.cdc.gov/healthypets/publications/index.html#animal-exhibits-and-handwashing [accessed Jun 30, 2017]).

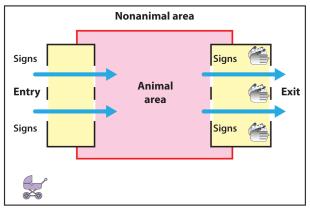
often not designed appropriately. Common problems include inadequate barriers, floors and other surfaces that are difficult to keep clean and disinfect, insufficient plumbing, lack of signs regarding potential health risks and risk prevention measures, and inadequate handwashing facilities.^{25,32,33,125} Specific recommendations might be necessary for certain settings, such as schools and childcare facilities **(Appendix 4**^{1–3}).

Recommendations for animal areas are as follows:

- Do not allow consumption of food or beverages in animal areas.
- Do not allow toys, pacifiers, spill-proof cups, baby bottles, strollers or similar items to enter animal areas.
- Prohibit smoking and other tobacco product use in animal areas.

- Children should not be allowed to sit or play on the ground in animal areas or on manure piles. If hands become soiled, supervise handwashing immediately.
- For areas where animal contact is encouraged, a 1-way flow of visitors is recommended, with separate entrance and exit points (Figure 3).
- Control visitor traffic to prevent overcrowding.
- Ensure that animal feed bowls or bins and water are not accessible to the public.
- Allow the public to feed animals only in circumstances where contact with animals is controlled (eg, with barriers).
- Do not provide animal feed in containers that can be eaten by people (eg, ice cream cones) to decrease the possibility of children eating food that has come into contact with animals.
- Promptly remove manure and soiled animal bedding from exhibit areas.
- Assign trained staff members to encourage appropriate human-animal interactions, to identify and reduce potential risks for patrons, and to process reports of injuries and exposures.
- Ensure that visitors do not have access to animals that are not part of the defined interaction area, especially in on-farm visit situations.
- Store animal waste and specific tools for waste removal (eg, shovels and pitchforks) in designated areas that are restricted from public access.
- Avoid transporting manure and soiled bedding through nonanimal areas or transition areas. If this is unavoidable, take precautions to prevent spillage.
- Where feasible, clean and disinfect the animal area (eg, flooring and railings) as necessary.
- Provide adequate ventilation for animals²³⁸ and people, but avoid creating air movement that distributes dust, which may contain contaminants.
- Minimize the use of animal areas for public activities (eg, weddings and dances). If areas previously used for animals must be used for public events, they should be cleaned and disinfected, particularly if food or beverages are served.
- For bird encounter exhibits, refer to the NASPHV's psittacosis compendium¹⁹⁸ for recommendations regarding disease prevention and control.
- Visitors to aquatic touch tank exhibits should

Design 1





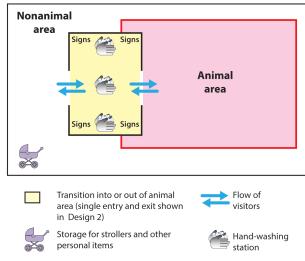


Figure 3—Examples of 2 designs for facilities with animal exhibit areas, including clearly designated animal areas, nonanimal areas, and transition areas with handwashing stations and signs.^{1–3} (Adapted from NASPHV Animal Contact Compendium Committee 2013. Compendium of measures to prevent disease associated with animals in public settings, 2013. *J Am Vet Med Assoc* 2013;243:1270–1288. Reprinted with permission.)

be advised not to participate if they have open wounds. Handwashing stations and signs should be provided as for other venues.

- When using animals or animal products (eg, pelts, fecal material, or owl pellets) for educational purposes, use them only in designated animal areas. Animals and animal products should not be brought into school cafeterias or other areas where food and beverages are stored, prepared, served, or consumed.
- When animals are in school classrooms, specific areas must be designated for animal contact (Appendix 4). These areas must be thoroughly cleaned after use. Parents should be informed of the pres-

ence of animals as well as the benefits and potential risks associated with animals in school classrooms.

• Immersion exhibits (where members of the public enter into the animal space) present additional opportunities for transmission of infectious agents. Entry into these spaces can lead to increased contamination of clothes, shoes, and other items, therefore increasing risk for disease. Lack of barriers between animals and people also increases the risk for injury. These exhibits heighten the need for supervision and awareness by venue operators and attendees.

(2) Transition area considerations. The following steps are recommended for management of transition areas between non-animal and animal areas. Establishing transition areas through which visitors pass when entering and exiting animal areas is critical. The transition areas should be designated as clearly as possible, even if they are conceptual rather than physical (Figure 3).

Entrance transition areas should be designed to facilitate education:

- Post signs or otherwise notify visitors that they are entering an animal area and that there are risks associated with animal contact (Figure 1).
- Instruct visitors not to eat, drink, smoke, place their hands in their mouth, or use bottles or pacifiers while in the animal area.
- Establish storage or holding areas for strollers and related items (eg, wagons and diaper bags).

Exit transition areas should be designed to facilitate handwashing (Appendix 3):

- Post signs or otherwise instruct visitors to wash their hands when leaving the animal area (Figure 2).
- Provide accessible handwashing stations for all visitors, including children and people with disabilities (Figure 3).
- Position venue staff members near exits to encourage compliance with proper handwashing.
- Post signs or otherwise instruct visitors to exercise proper handwashing when handling shoes, clothing, and strollers that might have come in contact with animal bedding or waste.

(3) Nonanimal area considerations. Recommendations for nonanimal areas are as follows:

- Do not permit animals, except for service animals, in nonanimal areas.
- Restrict storage, preparation, serving, and consumption of food and beverages to nonanimal areas.

- Provide handwashing facilities and display handwashing signs where food or beverages are served (Figure 2; Appendix 3).
- Separation of food from animal contact areas is of particular importance to farm visit venues; this includes food tasting, distribution of food samples, and consumption of beverages, snacks, or meals.

b. Cleaning and disinfection

Cleaning and disinfection practices should be tailored to the specific situation. For example, most parasitic pathogens, such as Cryptosporidium parvum, are resistant to most disinfectants. When a particular organism has been identified, additional guidance regarding specific disinfectants can be found in other resources.²³⁹ General recommendations are that all surfaces should be cleaned thoroughly to remove organic matter before disinfection. Prompt, safe removal of fecal matter reduces the risk of infection. Disinfectants, such as bleach and guaternary ammonium, should be used in accordance with the manufacturer label. Most compounds require > 10 minutes of contact time with a contaminated surface to achieve the desired result. Animals should be removed during the cleaning process and should not reenter the area until after disinfected surfaces have been thoroughly rinsed.

Venue operators should strive to develop an integrated pest management program to eliminate or reduce the risk of exposure to pathogens carried by pests. Carriers of concern include flies, mosquitos, ticks, and fleas as well as rodents.

c. Unpasteurized food and products

Unpasteurized or raw dairy products (eg, milk, cheese, and yogurt) and unpasteurized cider or juices are potential sources of foodborne pathogens. Consumption of such products should be prohibited.

d. Drinking water

Local public health authorities should inspect drinking water systems before use. Only potable water should be used for consumption by animals and people. Backflow prevention devices should be installed between outlets in livestock areas and water lines supplying other areas on the grounds. If the water supply is from a well, adequate distance should be maintained from possible sources of contamination (eg, animal holding areas and manure piles). Maps of the water distribution system should be available for use in identifying potential or actual problems. The use of outdoor hoses should be minimized, and hoses should not be left on the ground. Hoses that are accessible to the public should be labeled to indicate the water is not for human consumption. Operators and managers of facilities in settings where treated municipal water is not available should ensure that a safe water supply (eg, bottled water) is available.

3. Animal care and management

a. Selection of animals for use in public settings

The risk for disease or injury from animal contact can be reduced by carefully managing animal use. The following recommendations should be considered for management of animals in contact with the public:

- Direct contact with some animals is inappropriate in public settings, depending on expected audiences. Use of preweaned calves, reptiles, amphibians, and live poultry (including chicks) is not appropriate in nursing homes, schools, daycares, or other venues where groups at high risk for serious infection are expected to be present; contact with other young ruminants such as lambs or goat kids is also of increased concern in such settings. Animals showing signs of illness are not appropriate for use in public settings. In addition, direct contact with species known to serve as reservoirs for rabies virus (eg, bats, raccoons, skunks, foxes, and coyotes) should not be permitted. Certain nonhuman primates are of particular concern because of the types of pathogens they can transmit to people, such as B virus.²⁴⁰
- Because of their strength, unpredictability, or ability to produce venom, certain domestic, exotic, or wild animals should be prohibited from exhibition settings where a reasonable possibility of animal contact exists. Species of primary concern include certain nonhuman primates, certain carnivores (eg, lions, tigers, ocelots, wolves and wolf hybrids, and bears), and venomous species (eg, some reptiles and invertebrates).

b. Routine animal care

Venue operators and staff should monitor animals daily for signs of illness and ensure that animals receive appropriate veterinary care. Ill animals, animals known to be infected with a zoonotic pathogen, and animals from herds with a recent history of abortion, diarrhea, or respiratory disease should not be exhibited. To decrease shedding of pathogens, animals should be housed in a manner to minimize stress and overcrowding.

c. Veterinary care and animal health

Venue operators should retain and use the services of a licensed veterinarian. Regular herd or flock inspection while animals are present in the venue is a critical component of monitoring health. When necessary, Certificates of Veterinary Inspection from an accredited veterinarian should be up-to-date according to local or state requirements for animals in public settings. Preventive care, including vaccination and parasite control appropriate for the species, should be provided with appropriate input from the herd or flock veterinarian.

(1) Vaccination against rabies virus. All animals should be housed in a manner that reduces potential exposure to wild animals that may serve as rabies virus reservoirs. Mammals should also be up-to-date for rabies vaccinations according to current recommendations.²³² These steps are particularly critical in areas where rabies is endemic and in venues where human-animal contact is encouraged or possible. Because of the extended incubation period for rabies, unvaccinated mammals should be vaccinated ≥ 1 month before they have contact with the public. If no licensed rabies vaccine exists for a particular species (eg, goat, swine, llama, or camel) that is used in a setting where public contact occurs, consultation with a veterinarian regarding extralabel use of rabies vaccine is recommended. A vaccine administered in an extralabel manner does not provide the same degree of assurance as a vaccine labeled for use in a given species; however, extralabel use of rabies vaccine might provide protection for some animals and thus decrease the probability of rabies transmission.²³² Mammals that are too young to be vaccinated should be used in exhibit settings only if additional restrictive measures are available to reduce risks (eg, using only animals that were born to vaccinated mothers and housed in a manner to avoid rabies exposure). In animal contact settings, rabies testing should be considered for animals that die suddenly.

(2) Vaccination against enteric pathogens. While vaccines against certain enteric pathogens (eg, *Salmonella* spp and *E coli* O157:H7) are available for specific animal species, insufficient evidence currently exists to support the use of these products to reduce transmission of disease to people in public settings.²⁴¹ More research is necessary and encouraged before firm recommendations can be made.

(3) Other considerations for vaccination. Vaccination of slaughter-class animals before displaying them at fairs might not be feasible because of the slaughter withdrawal period that is needed when certain vaccines are used.

(4) **Testing for zoonotic pathogens.** Routine screening for zoonotic diseases is not recommended, except for *C psittaci* infection in bird encounter exhibits¹⁹⁸ and tuberculosis in elephants¹⁸⁹ and primates.²⁴² Screening tests are available for some en-

teric pathogens (eg, STEC and *Salmonella* spp); however, the interpretation of test results can be problematic. Shedding can be intermittent, and negative results do not indicate an animal was not shedding an organism at an earlier time or will not start shedding in the near future. There is no established guidance for management of animals with positive test results, and inappropriate interpretation might lead to unnecessary treatments, quarantine, or euthanasia.

4. Birthing exhibits

Animal birthing exhibits are increasingly popular. However, it is important for organizers and attendees to understand that animals such as goats, sheep, and cattle giving birth might be shedding pathogens, such as *C burnetii*, *Brucella* spp, *Leptospira* spp, and *L monocytogenes*. Organizers should be aware of the following steps to reduce the risk of disease transmission:

- Ensure that the public has no contact with newly born animals or birthing byproducts (eg, the placenta).
- Ensure that attendees and staff who are particularly vulnerable to zoonotic diseases (eg, pregnant women, people with cardiac valvular disease and other heart conditions, and people with weakened immune systems) and the parents of small children understand the risks of attending or working at these exhibits.
- Thoroughly clean and disinfect the birthing area after each birth, and use appropriate safety precautions and disposal methods for discarding waste products.
- If abortions or stillbirths occur, the exhibit should be closed; operators should work with their veterinarians to determine the cause of abortions or stillbirths.
- Birthing events should be held outdoors or in well-ventilated areas to reduce the risk for human exposure to aerosolized pathogens.

Additional information is available electronically in the CDC fact sheet on Q fever safety at livestock birthing exhibits.²⁴³

5. Considerations regarding variant influenza

In response to the influenza A (H3N2) variant virus outbreaks associated with swine at agricultural fairs in 2011 through 2012, the following prevention strategies have been recommended²⁴⁴:

- All people should take routine preventive actions (eg, practice appropriate hand hygiene) at fairs to reduce potential influenza virus transmission between pigs and people.
- People at high risk of serious influenza-related complications should avoid exposure to pigs at fairs.
- Measures should be taken to reduce the presence of pigs with clinical signs of disease at these events.

Potential strategies to mitigate the risk for intraspecies and interspecies transmission of influenza viruses at agricultural fairs include shortening the swine exhibition period, consulting with a veterinarian to determine whether vaccination of swine against influenza is appropriate, and allowing \geq 7 days' time between exhibitions before showing a pig or its penmates to reduce the risk of spreading influenza.²⁴⁴ More detailed and current recommendations for fairs can be found at the NASPHV website.²³⁴

V. Summary

Contact and interaction with animals in public settings can be a valuable means of education and entertainment. People who provide these opportunities to the public as well as those attending such venues should be aware of the potential health risks associated with such venues and understand that even apparently healthy animals can transmit pathogens. The recommendations included in this compendium will help venue operators, staff, and attendees reduce the risk for injury and zoonotic disease transmission in these settings.

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Appendix I

Selected Zoonotic Diseases of Importance in Public Settings in the United States, $2017.^{10}$

Disease	Agents	Most common species associated with transmission to people	Most common means of transmission to people	Most common clinical manifestations in people
Acariasis (mite infestation)	Sarcoptes scabiei (species-specific variants), Notoedres cari other species of mires	Dogs, cats, horses, goats, sheep, swine, birds	Direct or indirect contact	Itchy skin lesions
Bartonellosis	Bartonella henselae, other Bartonella spp	Cats	Scratches, bites	Fever, malaise, lymphadenopathy, skin lesions at inoculation site
Brucellosis	Brucella spp	Dogs, cervids, feral swine, bison, marine mammals	Ingestion, droplet or aerosol, contact	variable, nonspecific febrile illness
Campylobacteriosis	Campylobacter jejuni, other Campylobacter spp	Poultry, cattle, sheep, goats, swine, dogs,	Fecal-oral contact	Gastroenteritis, fever; usually self-limiting
Capnocytophaga spp	Capnocytophaga canimorsus, Capnocytophaga cynodeami	Dogs, cats	Scratches, bites	Fever, localized infections
Chlamydiosis (mammalian)	Chlamydophila abortus, Chlamydophila felis	Sheep, goats, llamas, cats, cattle	Aerosol, fecal-oral contact	Miscarriage, septicemia
Contagious pustular dermatitis (orf)	Parapoxvirus	Sheep, goats	Direct or indirect contact	Skin papules, lymphadenopathy, influenza-like illness
Cryptostory (01) Cryptosporidiosis Cutaneous larva migrans (zoonotic hockworm)	Cryptosporidium parvum Ancylostoma braziliense, Ancylostoma caninum	Cattle (typically calves), sheep, goats Dogs, cats	Fecal-oral contact Direct contact with contaminated soil	Gastroenteritis Skin lesions
Dermatophytosis	Microsporum spp, Trichophyton spp, Evidermochinton snc	Cats, dogs, cattle, goats, sheep, horses, rabbits,	Direct or indirect contact	Skin lesions
Giardiasis Herpes B virus infection	62	Dogs, cats, livestock Macaque monkeys	Fecal-oral contact Bites, scratches	Gastroenteritis Localized skin lesions, influenza-like
Influenza Leptospirosis	Influenza A virus Leptospira spp	Swine, poultry Swine, cattle, dogs, rodents	Droplet or aerosol Direct or indirect contact, droplet	symptoms, encepnaiomyelitis Fever, malaise, muscle and joint pain Fever, other nonspecific signs
Listeriosis	Listeria monocytogenes	Cattle, sheep, goats, pigs, dogs, cats	Fecal-oral contact, direct contact	Gastroenteritis, influenza-like symp-
Monkeypox	Orthopoxvirus	Nonhuman primates, rodents	Direct or indirect contact, bites, aerosol	toms, miscarriage Influenza-like symptoms followed by
Mycobacteriosis	Mycobacterium marinum	Aquarium fish	Direct contact with infected fish or	skin lesions Skin lesions
(nontuperculous) Pasteurellosis	Pasteurella multocida and other species	Dogs, cats, rabbits	contaminated water, aerosoi Bites, scratches, contact with mucous	Wound infections
Psittacosis Q fever	Chlamydophila psittaci Coxiella burnetii	Pet birds, poultry Goats, sheep, cattle	Aerosol	Influenza-like symptoms, cough Influenza-like symptoms, pneumonia
Rabies Rat bite fever Salmonellosis	Lyssavirus Sreptobacillus moniliformis, Spirillum minus Salmonella spp	Domestic and wild mammals Rats, mice.,gerbils Reptiles, amphibians, poultry, swine, cattle,	Bites Bites, scratches Fecal-oral contact	(rare), endocardito Acute, progressive neurologic disease Fever, severe muscle and joint pain Gastroenteritis
Staphylococcosis STEC infection	Staphylococcus spp STEC	goats, norses, rodents Swine, dogs, cats Cattle, goats, sheep, deer	Bites, scratches Fecal-oral contact	Localized skin and soft tissue infections Gastroenteritis, hemolytic-uremic
Streptococcosis Toxoplasmosis	Streptococcus spp Toxoplasma gondii	Swine, dogs, cats Cats	Bites, scratches Fecal-oral contact	Syndrome Localized skin and soft tissue infections Lymphadenopathy, mild influenza-like
Tuberculosis Visceral larva migrans	Mycobacterium tuberculosis complex Toxocara canis, Toxocara cati, Baylisascaris procyonis	Elephants, cattle, nonhuman primates Dogs, cats, raccoons	Aerosol Fecal-oral contact	Respiratory disease Various and nonspecific signs (eg. fever, lethargy, cough)
(Adapted from Willia Veterinarians: Veterinary	ms CJ, Scheftel JM, Elchos BL, et al. Compendiur r Infection Control Committee 2015. J Am Vet N	(Adapted from Williams CJ, Scheftel JM, Elchos BL, et al. Compendium of veterinary standard precautions for zoonotic disease prevention in veterinary personnel: National Association of State Public Health Veterinarians: Veterinary Infection Control Committee 2015; <i>J Am Vet Med</i> Assoc 2015; 247:1252–1277, Reprinted with permission.)	lisease prevention in veterinary personnel: N mission.)	lational Association of State Public Health

Animals in Public Settings: Recommendations for Venue Operators, Staff, and Volunteers¹⁻³

All individuals involved with animal contact activity in any public setting should be aware of the following risks for disease and injury associated with animals in public settings:

- Disease and injuries have occurred following contact with animals and their environment.
- Animals that appear healthy can carry harmful germs that can make visitors sick.
- Visitors can pick up harmful germs when they touch animals or animal droppings or enter animal environments (even without directly contacting the animals).
- Visitors can rid themselves of most harmful germs acquired if they wash their hands immediately after leaving an animal area. Visitors should wash their hands even if they did not directly contact the animals.
- The risk for developing serious or life-threatening zoonotic disease from contact with animals is higher for some visitors, especially children < 5 years of age, persons \geq 65 years of age, pregnant women, and people with weakened immune systems, than for others.
- Direct contact with some animals is inappropriate for some, or all, audiences in public settings.
- No visitors should have contact with ill animals.
 - Direct contact with preweaned calves, reptiles, amphibians, and live poultry is not appropriate for people at high risk for zoonotic disease transmission, and direct contact with young ruminants of other species (eg. goats and sheep) is of increased concern for these individuals.
 - Dangerous animals (eg, nonhuman primates, certain carnivores, other rabies reservoir species, and venomous reptiles) should be prohibited from having direct contact with the public.
- Live animals, especially reptiles, amphibians, and live poultry, should not be given as prizes at fairs, carnivals, or other events.

Operators and all individuals involved with the animal contact activity should educate visitors (with simple instructions in multiple age-appropriate and language-appropriate formats) about the following before they enter animal areas:

- Risks for disease and injury, including the information that children < 5 years of age, people ≥ 65 years of age, pregnant women, and those with weakened immune systems are at greater risk than others of developing serious zoonotic diseases.
- Handwashing and assisting children with handwashing immediately after visiting an animal area.
- Avoiding eating, drinking, or placing things in their mouths after animal contact or after visiting an animal area, until they have washed their hands.
- Closely supervising children.
- Awareness that objects such as clothing, shoes, and stroller wheels can become soiled and serve as a source of germs after leaving an animal area.

Operators and all individuals involved with the animal contact activity should take the following steps to maintain a safe environment when animals are present in public settings:

- Design the venue with safety in mind by having designated animal areas, nonanimal areas, and transition areas; temporary exhibits and animal interaction areas used in farm visits, agritourism venues, etc may need additional measures to minimize risks of injury or disease transmission.
- Do not permit animals other than service animals in nonanimal areas.
- Assign trained staff members to monitor animal contact areas to ensure visitor safety.
- Exclude food and beverages, toys, pacifiers, spill-proof cups, baby bottles, and smoking and related activities from animal contact areas.
- Keep the animal areas as clean and disinfected as possible, and limit visitor contact with manure and animal bedding.
- Allow feeding of animals only if contact with animals can be controlled (eg, over a barrier), and do not provide feed in containers that might be consumed by persons (eg, ice cream cones).
- Design transition areas for entering and exiting animal areas with appropriate signs or notifications regarding risks associated with animal contact and location of handwashing facilities.
- Maintain handwashing stations that are accessible to children and people with disabilities, and direct visitors to wash their hands immediately upon exiting animal areas.
- Position handwashing stations in places that encourage handwashing when exiting animal areas.
- Maintain handwashing facilities and stations to include routine cleaning and restocking to ensure an adequate supply of paper towels and soap.
- Ensure that animals receive appropriate preventive care, including vaccinations and parasite control appropriate for the species.
- Provide potable water for animals.
- Provide handwashing facilities where food and beverages are stored, prepared, served, or consumed.
- · Prohibit consumption of unpasteurized dairy products (eg, raw milk), ciders, and juices.
- Minimize use of animal areas at other times for public activities (eg, weddings, dances, and barbecues).

Handwashing is the most important prevention step for reducing disease transmission associated with animals in public settings.

(Adapted from NASPHV Animal Contact Compendium Committee 2013. Compendium of measures to prevent disease associated with animals in public settings, 2013. J Am Vet Med Assoc 2013;243:1270–1288. Reprinted with permission.)

Handwashing Recommendations to Reduce Disease Transmission From Animals in Public Settings¹⁻³

General Recommendations

Handwashing is the most important prevention step for reducing disease transmission associated with animals in public settings. Hands should always be washed immediately when exiting animal areas, even if direct animal contact was not made; handwashing is also important after removing soiled clothing or shoes and before eating, drinking, or handling food. Venue staff members should encourage visitors to wash hands immediately upon exiting animal areas.

Correct Handwashing Procedure

- Wet hands with clean, running water (warm or cold) and apply soap; rub hands together to make a lather and scrub them well (be sure to scrub the backs of hands, between fingers, and under nails); continue rubbing hands for at least 20 seconds; rinse hands well under running water.
- Dry hands with a clean disposable paper towel or air-dry them. Do not dry hands on clothing.
- Assist young children with washing and drying their hands.

Establishment and Maintenance of Handwashing Facilities or Stations

- The number of handwashing facilities or stations should be sufficient for the maximum anticipated attendance, and facilities should be accessible for children (ie, low enough for children to reach or equipped with a stool) and people with disabilities as well as the general population.
- Handwashing facilities and stations should be conveniently located in transition areas between animal and nonanimal areas and in nonanimal food concession areas.
- Maintenance of handwashing facilities and stations should include routine cleaning and restocking to ensure an adequate supply of paper towels and soap.
- Running water should be of sufficient volume and pressure to remove soil from hands. Volume and pressure might be substantially reduced if the water supply is furnished from a holding tank; therefore, a permanent, pressurized water supply is preferable.
- Handwashing stations should be designed so that both hands are free for handwashing by having operation with a foot pedal or water that stays on after hand faucets are turned on.
- Liquid soap dispensed by a hand pump or foot pump is recommended.
- To increase compliance, water temperature should be set at what is considered comfortable.²³⁷
- Communal basins, in which water is used by more than I person at a time, are not adequate handwashing facilities.

Recommendations Regarding Hand-Sanitizing Agents

- Washing hands with soap and water is the best way to reduce the number of germs on them. If soap and water are not available, use an alcohol-based hand sanitizer that contains at least 60% alcohol in the interim until hands can be properly washed.
- Visible contamination and dirt should be removed before using hand sanitizers. Hand sanitizers may not be as effective when hands are visibly dirty or greasy.
- Even when hand sanitizer is used, visitors should always wash hands with soap and water as soon as possible after exiting animal areas; alcohol-based hand sanitizers can quickly reduce the number of germs on hands in some situations, but these products are not effective against all germs.

Correct Use of Hand Sanitizers

- Apply the product to the palm of I hand.
- Rub your hands together.
- Rub the product over all surfaces of your hands and fingers until your hands are dry.

Handwashing Sign Recommendations

- At venues where human-animal contact occurs, signs regarding proper handwashing practices are critical to reduce disease transmission.
- Signs that remind visitors to wash hands should be posted at exits from animal areas (ie, exit transition areas) and in nonanimal areas where food is served and consumed.
- Signs should be posted that direct all visitors to handwashing stations when exiting animal areas.
- Signs with proper handwashing instructions should be posted at handwashing stations and in restrooms to encourage proper practices.
 Handwashing signs should be available in multiple age-appropriate and language-appropriate formats.

(Adapted from NASPHV Animal Contact Compendium Committee 2013. Compendium of measures to prevent disease associated with animals in public settings, 2013. J Am Vet Med Assoc 2013;243:1270–1288. Reprinted with permission.)

Guidelines for Exhibition of Animals in School and Childcare Settings¹⁻³

General Recommendations

- Animals are effective and valuable teaching aids, but safeguards are required to reduce the risk for infection and injury. Other groups have developed recommendations similar to those provided here.^{175,204,205}
- Ensure that teachers and staff know which animal species are inappropriate as residents or visitors to the facility and which animals should not be in direct contact with children (See animal-specific recommendations in this Appendix).
- Ensure that personnel providing animals for educational purposes are knowledgeable regarding animal handling and zoonotic disease issues. People or facilities that display animals to the public should be licensed by the USDA.
- Inform parents of the presence of animals as well as the benefits and potential risks associated with animals in school classrooms. Consult with parents to determine special considerations needed for children who are immunocompromised, have allergies, or have asthma.
- Educate children about harmful germs that can spread between animals and people and about proper handwashing technique.
- Wash hands right after contact with animals, animal products, or feed or after being around animal environments.
- Supervise human-animal contact, particularly involving children < 5 years of age.
- Display animals in enclosed cages or under appropriate restraints.
- Do not allow animals used in schools or daycares to roam, fly free, or have contact with wild animals.
- Designate specific areas for animal contact. Do not allow food or drink in animal contact areas; do not allow animals in areas where food and drink are stored, prepared, served, or consumed.
- Clean and disinfect all areas where animals and animal products have been present. Children should perform this task only under adult supervision.
- Do not clean animal cages or enclosures in sinks or other areas used to store, prepare, serve, or consume food and drinks.
- Obtain a certificate of veterinary inspection, proof of rabies vaccination, or both according to local or state requirements for the species being exhibited. Also, ensure veterinary care, including preventive health programs for endoparasites and ectoparasites as appropriate for the species.

Animal-Specific Recommendations

Refer to the general guidelines regarding species for which specific recommendations are not provided in this section (eg, nonpsittacine birds and domestic dogs, cats, rabbits, and rodents [including mice, rats, hamsters, gerbils, guinea pigs, and chinchillas]).

- Reptiles (eg, turtles, snakes, and lizards): Do not keep reptiles in facilities with children < 5 years of age, and do not allow children of this
 age group to have direct contact with these animals.
- Amphibians (eg, frogs, toads, salamanders, and newts): Do not keep amphibians in facilities with children < 5 years of age, and do not allow children of this age group to have direct contact with these animals.
- Live poultry (eg, chicks, ducklings, and goslings): Do not keep live poultry in facilities with children < 5 years of age, and do not allow children of this age group to have direct contact with these animals.
- Ferrets: Do not keep ferrets in facilities with children < 5 years of age, and do not allow children of this age group to have direct contact with these animals to avoid bites. Ferrets should be up-to-date for rabies vaccination.
- Farm animals: Certain animals (eg, calves, goats, and sheep) intermittently excrete substantial numbers of germs; therefore, these farm animals are not appropriate in facilities with children < 5 years of age and should not be displayed to older children in school settings unless meticulous attention to personal hygiene can be ensured.
- Guide, hearing assistance, or other service animals and trained animals used in law enforcement: These may be used in accordance with recommendations from the sponsoring organizations when they are under the control of a person familiar with the specific animal.
- Psittacine birds (eg, parrots, parakeets, and cockatiels): Consult the psittacosis compendium¹⁹⁸ and seek veterinary advice.
- Fish: Children < 5 years of age and people with weakened immune systems should not clean aquariums. Wash hands before and after cleaning aquariums, and wear gloves if hands have cuts or wounds or when working with rough rocks or spiny fish. Do not dispose of aquarium water in sinks used for food preparation or for obtaining drinking water.
- Animal products: Assume that products such as owl pellets and frozen rodents used to feed reptiles are contaminated with Salmonella organisms. Dissection of owl pellets should not be performed in areas where food is stored, prepared, served, or consumed. Children < 5 years of age should not be allowed to have direct contact with animal products unless the product has been treated to eliminate harmful germs.

Animals Not Recommended in School or Childcare Settings

- Inherently dangerous animals (eg, lions, tigers, cougars, and bears).
- Nonhuman primates (eg, monkeys and apes).
- Mammals that pose a high risk for transmitting rabies (eg, bats, raccoons, skunks, foxes, and coyotes).
- Aggressive or unpredictable wild or domestic animals.
- Stray animals with unknown health and vaccination history.
- Venomous or toxin-producing spiders, insects, reptiles, and amphibians.
- Animals that pose a high risk for zoonotic disease transmission (eg, preweaned calves, reptiles, amphibians, and live poultry) or bites (eg, ferrets).

Adapted from NASPHV Animal Contact Compendium Committee 2013. Compendium of measures to prevent disease associated with animals in public settings, 2013. J Am Vet Med Assoc 2013;243:1270–1288. Reprinted with permission.)



MEASLES AND ACUTE FLACCID MYELITIS: TAMING TWO OLD ADVERSARIES

16

Figure 1. Measles in Oregon, 1 Jan 1993 - 30 Jan 2019

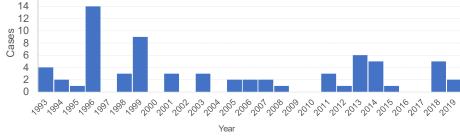
M easles has been much in the news, and understandably so. The number of cases^{*} just across the river in Clark County has soared past 30 and continues to rise, and Oregon has one linked, confirmed case.^{1,2} Though overall rates of measles, mumps, and rubella (MMR) vaccination among schoolaged youngsters in Oregon aren't bad (96%), some communities are substantially underimmunized, and their populations are at risk.

Acute flaccid myelitis (AFM) has also been getting a lot of press lately: no longer due to polio, but nonetheless causing suffering and disability among children.

In this *CD Summary*, we'll revisit these ancient scourges, review the epidemiology of recent outbreaks, and discuss strategies to stem the tide.

MEASLES

Measles is caused by a singlestranded RNA virus of the family Paramyxoviridae. Symptoms include fever and a characteristic morbilliform rash that starts on the face or at the hairline and spreads to the rest of the body, along with some combination of the "3 C's" of cough, coryza, and conjunctivitis. While most cases are self-limited, measles can be severe, causing pneumonia and encephalitis requiring hospitalization. Even in the U.S., CDC reports a measles mortality rate of 1-2 cases per 1000 cases.3 It spreads readily through the airborne route and is remarkably infectious: in a susceptible population, one measles case is likely to result in 12-18 additional infections.⁴ The measles outbreak in Clark County appears to have stemmed from a single imported case. Oregon has also had a few spikes like this over the years (Figure 1). This is typical of outbreaks in



the U.S. since 2000, when endemic measles was eliminated in this country through vaccination.

Common wisdom suggests that a community attaining a measles vaccination rate of about 95% will achieve herd immunity, that is, measles imported in a single case will not have enough susceptible folks around to be able to propagate, and it will die out. Alas, there is a clear risk of spread to underimmunized communities in Oregon. If measles got into these communities, it would go through them, to paraphrase an old "Down East"" story from Maine, like green corn goes through the new maid. Of the 30 cases of measles reported in Oregon since 2004, 21 (70%) were unvaccinated, including two too young to receive vaccine. Six were vaccinated, and vaccination status couldn't be documented for the other three. That at least 70% of cases have occurred among the ~4% of our population that is unvaccinated is testimony to the effectiveness of the vaccine.

Several strategies can be used to prevent outbreaks in undervaccinated communities. An easy one is making MMR vaccine available to unvaccinated patients in your practice. The number of measles cases in Oregon might be small at this point, but that could change quickly, and it's truly a kidney stone of a disease. CDC recommends an initial dose of MMR vaccine at 12–15 months, and a second on at 4–6 years of age. If families are anxious to protect their kids, the second dose may be given at any time at least 28 days after the first dose.⁵ Kids from kindergarten through college are considered fully immunized if they've had two doses. This also goes for adults who work in health care or who will be traveling to measles-endemic areas.[†]

Other adults are considered immune if they've had one documented MMR or were born before 1957. It there's any question, the vaccine is safe and about 97% effective with two doses. Err on the side of vaccinating.

Local public health officials in Washington and Oregon are investigating contacts of all known measles cases, identifying any who are unvaccinated, and checking in with them regularly to identify quickly any who become symptomatic. If this happens, they are asked to stay away from other possibly susceptible people at home, to call their clinician immediately and arrange for evaluation and testing in a way that doesn't expose other patients or clinic staff, and to isolate themselves at home until four days have passed since rash onset.

It's an effective strategy. To work well, it depends upon healthcare providers in several respects. First, public health can only find out about suspect measles cases early, and help arrange timely,

^{*} As of 9:50 AM January 30, 2019

[†] A designation we'd very much like to avoid

appropriate testing of high-risk cases, if clinicians call the case's local health department of residence to report the case. It's especially important to call about any unvaccinated patients with compatible illness and known, direct exposure to a measles case or a history of visiting one of the potential exposure venues at the time an infectious measles case was present. (For a list of venues and times of exposure, see Clark County's and the Public Health Division's measles websites, references 1 and 2, below.)

Early diagnosis has several benefits. In addition to being able to provide prompt supportive care to the patient, rapid identification of the measles patient's contacts might allow preventive therapy. If given within 72 hours of exposure, MMR may prevent or ameliorate disease. Immune globulin may have benefit in exposed infants, pregnant women, and immunocompromised folks up to six days after exposure. One study found that timely receipt of MMR or IG was 83% effective in preventing measles in exposed, susceptible people.6 Additionally, having a plan in your facility to meet a suspect measles case outside, give them a mask, and bring them in for evaluation or testing through a non-populated area of the clinic will keep immunocompromised or as-yet-unvaccinated patients in your practice safe, and help prevent further spread.

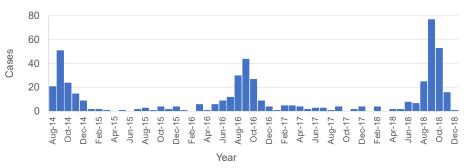
FOR MORE INFORMATION

 Oregon Health Authority: <u>www.ore-</u> <u>gon.gov/oha/ph/diseasesconditions/</u> <u>diseaseaz/Pages/measles.aspx</u>

Acute Flaccid Myelitis

Acute flaccid myelitis (AFM) is a rare condition that affects the gray matter of the spinal cord, resulting in limb weakness or paralysis.7 It's a syndrome rather than a diagnosis and can be caused by infection and possibly environmental exposures or genetic conditions. It's been around a long time. In the pre-vaccine era, millions of people worldwide were afflicted by the scourge of polio. Thanks to the work of Salk, Sabin and millions of dedicated health workers, endemic transmission of wild poliovirus persists today in only three countries: Afghanistan, Nigeria, and Pakistan.

In the U.S., AFM reared its ugly head again in 2014, with many of the cases tied to clusters of illness in Colorado and California.^{8,9} Another spike of cases was seen in 2016, many Figure 2. Confirmed AFM cases in U.S. reported to CDC, 2014–2018



of them in Arizona. As you've no doubt seen in the news, 2018 has also seen more than its quota of AFM cases, with 201 confirmed cases from 40 states reported to CDC.

The current definition for a confirmed case of AFM includes onset of acute flaccid limb weakness in the setting of spinal cord lesions largely restricted to gray matter and spanning one or more vertebral segments. A probable case is defined by the above symptoms, plus cerebrospinal fluid pleocytosis (white blood cell count >5 cells/mm³). We ask that you contact your local health department to report any illness involving acute-onset limb weakness or paralysis in anyone <21 years of age.

Here in Oregon from 2014 through 2017, six children with AFM were reported to the Public Health Division and subsequently confirmed to meet the above CDC case definition. We had no confirmed cases in 2018.

Nationally, the median age among confirmed cases is about four years. Almost 60% required ICU care. Testing at CDC has revealed evidence of a variety of enteroviruses in the CSF of four cases since 2014. The remaining 523 confirmed cases had none. Nonetheless, the fact that more than 90% of confirmed AFM patients had antecedent fever or mild respiratory illness prior to weakness onset suggests an infectious etiology for many of these illnesses.

MRI findings are consistent with lesions in lower motor neurons. This could be direct damage from infection or other insults, or it could reflect a maladaptive immune response.

To learn more about what is going on, we need to hear about cases of AFM, document the course of illness, and ensure systematic testing to determine their etiologies.

This is where you come in. For any patient <21 years of age who presents with acute-onset limb weakness, please contact your local health department and share with them:

- A completed AFM patient summary form available at: <u>www.cdc.gov/</u> <u>acute-flaccid-myelitis/hcp/data.html</u>,
- · Admission and discharge notes,
- Neurology and infectious disease consult notes,
- MRI reports and images, andLaboratory test results.

We also ask that you collect the following specimens from patients under investigation for AFM as early as possible in the course of illness.

- CSF,
- Serum,
- A nasopharyngeal or oropharyngeal swab, and
- Two stool samples, collected at least 24 hours apart early in the course of illness. This will help rule out poliovirus infection.

Public health can help coordinate shipment to CDC.

FOR MORE INFORMATION

CDC AFM Surveillance
 <u>www.cdc.gov/acute-flaccid-myelitis/</u>
 <u>afm-surveillance.html</u>

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March 2019 | Volume 68, Number 3

STOMACH FLU, WINTER VOMITING DISEASE OR NOROVIRUS: WHICH IS IT?

Winter and its frigid temperatures means more than the season for colds and influenza: it also means the season for norovirus illness, otherwise known as Winter Vomiting Disease. This issue of the *CD Summary* reviews noroviral illness, transmission, control measures and outbreaks.

Norovirus causes an estimated 19–21 million cases of acute gastroenteritis (AGE) in the United States each year, leading to 56,000– 71,000 hospitalizations and 570–800 deaths, mostly among young children and older adults.¹ Norovirus is the leading cause of severe AGE among medical care-seeking U.S. children <5 years of age, and a principal cause of AGE outbreaks on cruise ships and in preschools, hospitals, and long-term care facilities.²

NOROVIRUS BACKGROUND

Norovirus was first identified after an outbreak at an elementary school in Norwalk, Ohio in 1968. The investigators dubbed it "Norwalk virus," but the name was formally changed to "norovirus" in 2002. Norovirus belongs in the *Caliciviridae* family of small, non-enveloped viruses. There are six genogroups (G) of norovirus, of which GI, GII, and GIV afflict humans. Norovirus is highly contagious due to its low infectious dose, prolonged shedding following infection, lack of durable immunity in humans, and environmental stability.

NOROVIRUS SIGNS AND SYMPTOMS

Norovirus symptoms begin a median of 33 (range, 24–48) hours after exposure. Symptoms may include:

- vomiting,
- diarrhea, not bloody,

* Unfortunate cases find themselves spewing out of both ends.

- nausea,
- abdominal cramps,
- malaise,
- chills and
- low-grade fever.

In some cases, dehydration may occur. The illness is self-limiting, and most people start to feel better within a day or two.

NOROVIRUS TRANSMISSION

Human beings are the only known reservoir for norovirus. An infected person begins to shed norovirus a few hours before symptoms begin and can continue to shed for more than two weeks. Peak viral shedding occurs at 2 to 3 days after symptom onset, with a median of 95 billion noroviral genomic copies per gram of feces.³

Norovirus is commonly spread from person to person by the fecaloral route. This type of transmission is common in norovirus outbreaks in nursing homes, schools and day care centers. Point-source transmission may be seen when a person vomits in a crowded area.[†]

One common question is "how close do you have to be to the vomiter to get norovirus?" It so happens that there are researchers who are trying to figure out the answer to that very question, and they have done so by constructing vomiting machines (yes, more than one). "Vomiting Larry" demonstrated that the splash zone in an act of "projectile vomiting" was >3 m by >2.2 m.⁴ Another simulated vomiting machine built in 2015 showed that norovirus can be aerosolized during a vomiting event.⁵ Given the large splash zone and aerosolization of norovirus, it's not surprising that a vomiting incident in a public area can cause an outbreak.

† The common story is that someone vomited in a cafeteria and then there were 10 people sick the next day. When questioned, the 10 people that got sick later were sitting close to the vomiter.

Norovirus can also be spread via food or water (including ice) that is contaminated by a food handler who is shedding norovirus. Filter-feeding oysters can collect and concentrate norovirus from human feces, should it manage to reach the oyster bed. Regardless of how it gets into the food, foodborne norovirus outbreaks can rapidly sicken many partygoers or restaurant patrons. Onsets usually are clustered in time and typically associated with foods that had been handled by a food handler who worked while sick, and that were not cooked afterwards. Salads, sandwiches, fruits, and frostings are commonly implicated vehicles.

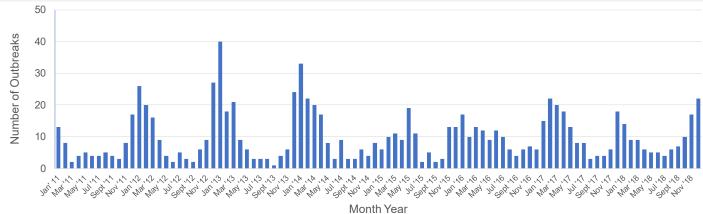
NOROVIRUS TESTING

Individual cases of noroviral AGE are clinically indistinguishable from those caused by other viral or bacterial agents. On the other hand, presumptive diagnosis of noroviral etiology may be made with a high degree of certainty given a *cluster* of cases in which more than half have vomiting, and more than half have diarrhea. Norovirus can be confirmed by testing a stool sample using real-time reverse-transcriptase polymerase chain reaction (RT-PCR). Some commercial lab scan test for norovirus; and increasingly, locally available multiplex gastroenteritis PCR panels can also detect it.

NOROVIRUS TREATMENT

There is no specific treatment for norovirus. Antibiotics will not help. Hydration is the key to managing it.

Nor is a commercial norovirus vaccine currently available. A promising bivalent vaccine, developed by Takeda, has reached the randomized trial stage, but no data are available on the duration of antibody persistence or clinical efficacy. One oral vaccine in tablet form, under development by Vaxart, recently completed phase 1 studies.



NOROVIRUS OUTBREAKS IN OREGON

While individual cases of noro-like illness are not reportable, clusters of illness are reportable to public health authorities so we can implement control measures to hopefully stop transmission.

The Oregon Health Authority (OHA) receives hundreds of noroviral and noro-like outbreak reports each year. An outbreak is defined as cases with compatible symptoms occurring in at least two different households clustered in space and time or following a common exposure. We further define a "confirmed" norovirus outbreak as having norovirus detected in specimens from at least two patients. Noro-like outbreaks are those with a similar symptom profile but without the requisite noro-positive specimens.

From 2011 through 2018, 949 confirmed noroviral and noro-like outbreaks were reported in Oregon. Of these, 657 (69%) were confirmed as noroviral. The winter seasonality of the outbreaks can be appreciated in Figure 1.

Norovirus outbreaks were reported in a variety of settings including nursing facilities, fairs, restaurants, schools, private parties, hospitals, and camps. Six hundred forty-three (68%) of the confirmed and noro-like outbreaks were in nursing homes. The next most common setting was schools (n=71, 7%) and restaurants (n=65, 7%).

Of the 949 outbreaks, 500 (53%) had at least one specimen with norovirus genotyping. Most of these proved to be in norovirus genogroup 2 (n=424, 85%). Among these GII norovirus outbreaks, the most common genotype was GII.4 Sydney

(n=238, 56%), followed by GII.4 New Orleans (n=42, 10%) and GII.2 (n=34, 8%). Only 76 (18%) of the outbreaks with genotyping data were genogroup 1, of which the most common genotype was GI.3B (n=24, 32%), followed by GI.6A (n=15, 20%).

NOROVIRUS OUTBREAK CONTROL MEASURES

In congregate settings such as nursing homes, ill residents should be put on enteric contact precautions as soon as an outbreak begins. These precautions include gowns, gloves, masks, and washing hands with soap and water. Other control measures (e.g., stopping communal activities) should also be instituted promptly. Ill staff members should stay at home while ill and for 48 hours after symptoms resolve — and not work at any other facility during this time. Similar control measures and work restrictions should be employed in schools and day care facilities.

HAND HYGIENE

Alcohol-based hand sanitizers are ineffective against the non-enveloped norovirus. We will have to wash our hands the old-fashioned way.

NOROVIRUS CLEANING AND DISINFECTING

Proper cleaning and disinfection is important in stopping transmission of norovirus. First, wear the proper personal protective equipment (gowns, masks and gloves) before cleaning and disinfecting. "Cleaning" means removing foreign materials from surfaces or objects; it is done with water, a detergent and elbow grease. "Disinfection" is the killing of pathogens on surfaces or objects. The Environmental Protection Agency (EPA) maintains a list of registered disinfectants effective at killing norovirus. If you don't have an EPA-

registered disinfectant on hand, plain old bleach is also highly effective. We recommend using a 3500-ppm bleach solution — which can be prepared by mixing 1 cup of household bleach in a gallon of water. If you use bleach solution, prepare a fresh batch each day.

As far as potentially contaminated food goes: when in doubt, throw it out, and then wash your hands.

FOR MORE INFORMATION

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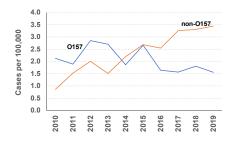
EBBS AND FLOWS OF COMMUNICABLE DISEASES: OREGON 2019

aboratories and health care professionals are required by Oregon law to report diseases of public health importance to public health authorities. Public health officials investigate these reports of communicable disease to characterize the illness, collect demographic information, and identify possible sources of infection. This allows public health to take steps to prevent further disease transmission and to monitor trends in communicable disease across the state. This CD Summary presents notable trends in the diseases reported during 2019.

SHIGA TOXIN STORM

Shiga toxin-producing Escherichia coli (STEC) infection causes gastroenteritis - it is often characterized by bloody diarrhea and in severe cases, illness can lead to post-diarrheal hemolytic uremic syndrome (HUS). E. coli O157:H7 is the most common strain of STEC, though there are many non-O157 strains as well. In Oregon in 2019, 354 cases were reported, a notable increase from the 315 and 217 cases in 2018 and 2017, respectively. Sixty-six (31%) of the STEC cases in 2019 were O157. The rate of O157 STEC infections in Oregon has been gradually declining over the past decade, while the rate of non-O157 STEC infections has continued to climb (Figure 1). In 2019, the rate

Figure 1. Incidence rate of shiga toxin-producing *E. coli* (STEC) by serotype, Oregon, 2010–2019



of non-O157 STEC cases reached a new high of 3.4 per 100,000 people, compared to 1.6 for O157 STEC cases. Incidence of infection is higher in children <5 years of age. Historically, the rate of STEC infections in Oregon has been higher than the rate in the rest of the U.S. and this remained true in 2019 — Oregon experienced nearly double the rate of STEC cases compared to other states.¹ There were four outbreaks of STEC investigated in Oregon in 2019: one outbreak associated with beef products sold at a grocery chain that resulted in 65 cases, two additional foodborne outbreaks, and one outbreak associated with animal contact. All four were outbreaks of the O157:H7 strain.

RESPIRATORY UNREST

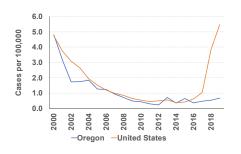
Legionellosis is an acute respiratory tract infection following exposure to Legionella spp. It varies in severity from a mild febrile illness to a serious and sometimes fatal form of pneumonia. Legionella bacteria are found naturally in the environment and are transmitted by inhalation of aerosolized water or soil infected with the bacteria. Person-to-person transmission does not occur. There was a dramatic spike in legionellosis cases in Oregon in 2019, with 73 cases, compared to only 40 cases in 2018. At the same time, national cases of legionellosis declined in 2019 after years of a steady rise in cases. The cause of the rise in Oregon in 2019 remains unknown; investigations did not identify any clusters. However, increases in older persons and those with underlying disease, aging plumbing infrastructure, and increased testing, detection and reporting may have played a role. Among the 73 cases in 2019, 97% were hospitalized and there were eight deaths.

Measles is an acute, highly communicable viral illness. The hallmark of the disease is a red, blotchy rash that starts on the face and then spreads widely over the body. The rash is preceded by a febrile prodrome that includes cough, coryza, and conjunctivitis. Efforts to increase vaccination among preschool children since 1989 has resulted in dramatic reduction in measles in the United States. In 2019, about 96% of K-12 kids in Oregon received two doses. Although the risk of exposure to measles in Oregon remains low, 2019 saw a large increase in measles cases; the highest count in 28 years. In fact, Oregon's incidence surpassed the rate in the rest of the U.S. for the first time since 2013 (0.7 cases per 100,000 in Oregon compared to 0.4 cases per 100,000 people in the rest of the U.S.).¹ The median age of cases has been 12.5 years (range, 6 months-49 years) since 2004. Four outbreaks of measles accounted for 27 of the 28 cases in 2019: two communitywide outbreaks, one at a missionary training school, and one associated with a flight. All cases in 2019 were importlinked cases (linked to an internationally imported case), and all were unvaccinated.

HEPATITIS A COUNTS CLIMB

<u>Hepatitis A</u> is a liver disease caused by the hepatitis A virus, which is transmitted via the fecal-oral route. Historically, Oregon had one of the higher state incidence rates of hepatitis A in the U.S.; however, the number of hepatitis A cases declined both nationally and in Oregon following the licensure of the hepatitis A vaccine in 1995–1996. Oregon's case count declined from 165 cases in 2000 to only 9 cases in 2012, but it has been gradually rising since that time, up to 28 cases in 2019. Despite this trend in recent years, the rate of hepatitis A cases in Oregon remains well below that of the rest of the U.S. (Figure 2).¹

Figure 2. Incidence rate of hepatitis A in Oregon and the rest of the United States, 2000–2019

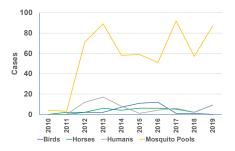


Since 2017, the U.S. has been experiencing widespread person-toperson outbreaks of hepatitis A across the county, resulting in a steep surge in national case rates.² The 28 cases Oregon saw in 2019 included a cluster of 5 five hepatitis A cases in a highrisk population reporting injection drug use and unstable housing in Central Oregon. Ten of the 28 cases in 2019 were acquired outside of Oregon or from household members who recently traveled outside of Oregon.

WHAT IS UP WITH WEST NILE?

Over the past 10 years, the incidence rate of WNV in Oregon has been highest in SE Oregon, especially Malheur County (9.8 cases per 100,000 people) and Harney Count (8.1 cases per 100,000 people). Animal surveillance for WNV found more animal cases of WNV compared to previous years, with 9 horses and 87 mosquito pools testing positive for WNV in 2019 (Figure 3). 2019 also saw an increase in human cases of WNV relative to the previous year, with a total of nine cases. Eight of them were locally acquired and one was imported. Only one in five humans infected with WNV will develop symptoms - typically flu-like symptoms such as fever, headache and muscle aches.

Figure 3. West Nile virus by species, Oregon, 2010–2019



EBBS IN ENTERIC DISEASES

Cryptosporidiosis results from infection with protozoal parasite of the genus Cryptosporidium and is characterized by watery diarrhea and abdominal cramps. The rate of cryptosporidiosis in Oregon has been generally inclining since 2000; however, there has been a gradual decline in cases since reaching a peak of 329 in 2016 - Oregon recorded 253 cases of cryptosporidiosis in 2019. The case rate in Oregon continues to remain above the rate in the rest of the U.S. (Figure 4).¹ Rapid cartridge tests and culture independent diagnostic testing for Cryptosporidium might be playing a role in the apparent increase in incidence for the past decade.

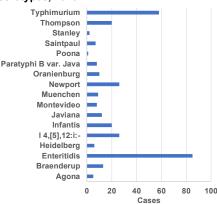
Figure 4. Incidence rate of cryptosporidiosis in Oregon and the rest of the United States, 2000–2019



Giardiasis is caused by infection with the flagellated protozoan Giardia intestinalis. While most infections occur without symptoms. Giardia cysts can be excreted in the stool intermittently for weeks or months, resulting in a prolonged period of communicability. Children in daycare and their close contacts, backpackers and campers, persons drinking from shallow wells. travelers to disease-endemic areas and men who have sex with men are at greatest risk. While giardiasis in Oregon (6.9 per 100,000 people in 2019) remains elevated above the rest of the U.S. (4.0 per 100,000 people in 2019)¹, cases have been gradually declining since 2010 and were down to 291 cases in 2019.

Salmonellosis is a bacterial illness characterized by acute abdominal pain, diarrhea and often fever. Symptoms typically begin one to five days after exposure, but excretion of *Salmonella* may persist for several days or even months beyond the acute illness. Since 2000, salmonellosis cases have been generally inclining in Oregon, reaching a peak of 582 cases in 2018. In 2019, however, this number decreased to 460 cases (10.9 cases per 100,000 people), mirroring a drop in case rates in the rest of the U.S.¹ and keeping Oregon below the national average. Despite this, there were nine outbreaks of salmonellosis investigated in 2019, which accounted for 64 of the 460 cases in Oregon. Of the 460 cases, 406 had lab-confirmed isolates, from which 64 different *Salmonella* serotypes were identified (Figure 5). **Figure 5.** *Salmonella* cases by selected*

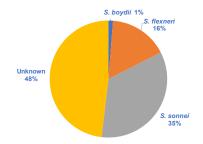
Figure 5. Salmonella cases by selected serotypes, 2019*



*Selected because at least one case was reported in 2019 and it is a more common serotype.

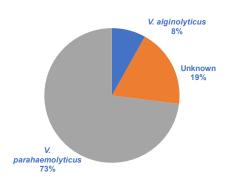
Shigellosis is an acute bacterial infection, and in Oregon, it is typically caused by S. sonnei or S. flexneri (Figure 6). The illness is characterized by diarrhea (sometimes bloody), vomiting, abdominal cramps and fever. After a large spike in cases led to a record case count of 289 in 2018. cases decreased again in 2019 with 160 cases of shigellosis recorded in Oregon. The high case count in 2018 was due, in part, to a large foodborne outbreak. outbreak. With the exception of 2018, the case rate of shigellosis cases in Oregon remains below that of the rest of the U.S.1

Figure 6. Shigellosis cases by species, Oregon, 2019



<u>Vibriosis</u> is caused by infection with bacteria from the *Vibrionaceae* family, which includes the species that causes cholera. Vibriosis is often acquired by eating raw or undercooked molluscan shellfish, although non-foodborne infection with *Vibrio* species can also occur through contact with sea or brackish water. In Oregon, *V. parahaemolyticus* is the most frequently reported species (Figure 7) and it is found naturally in the coastal waters and shellfish of the Pacific Northwest.

Figure 7. Vibriosis cases by species, Oregon, 2019



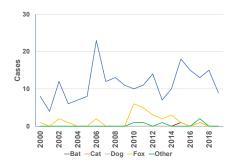
Oregon recorded a record high number of vibriosis cases in 2018 (67), but cases declined again in 2019 with 37 cases reported (0.9 cases per 100,000 people). The case rate in Oregon remains slightly elevated above the case rate in the rest of the U.S.¹Almost half of 2019 cases were initially detected from a polymerase chain reaction (PCR) test. In 2018, Oregon changed the case definition for Vibrio infections to exclude some of these PCR tests, in an attempt to mitigate the problem. Not all the increase in cases can be attributed to changes in culture independent diagnostic testing, however, 30 out of 37 reports in 2019 were culture confirmed.

DECLINES IN ZOONOTIC DISEASES

Lyme disease is a tick-borne zoonotic disease caused by the spirochete *Borrelia burgdorferi*. In most cases, the tick must be attached for 36-48 hours or more before the bacterium can be transmitted and the incubation period ranges from 3 to 30 days after exposure. Cases have been reported in 49 states and in Ontario and British Columbia, Canada. Following a record number of cases in 2017 (89), cases declined to 65 reported in 2019; however, there is an overall increasing trend relative to earlier in the decade. The median age of cases in 2019 was 40 years of age. Fifty-two (63%) cases were female. The rate of Lyme disease cases in Oregon (1.5 per 100,000 people in 2019) continues to remain well below the rate in the rest of the U.S. (10.4.per 100,000 people).¹

Rabies is an acute infection of the central nervous system caused by a neurotropic rhabdovirus of the genus Lyssavirus. All mammals are susceptible to rabies and in humans, it causes rapidly progressive and fatal encephalomyelitis. Bites from infected animals constitute the primary route of transmission. Oregon (and the rest of the Pacific Northwest) is considered to be free of terrestrial rabies — the main reservoir of rabies in Oregon is bats. Rabies in humans is rare and is 100% preventable through prompt appropriate medical care, but public health monitors rabies in animal populations as well. In 2019, despite testing similar numbers of animals that potentially exposed humans to rabies, the number of positive animal cases decreased to nine, all of which were in bats (Figure 8). The rate of animal rabies cases in Oregon continues to remain well below the national rate in the rest of the U.S.¹

Figure 8. Animal rabies cases by species, Oregon, 2000–2019



MORE HIGHLIGHTS FROM 2019

In 2019, Oregon recorded the lowest number of cases of meningococcal disease in this millennium – only 11 cases were reported. The case rate in Oregon has been steadily decreasing since 2000 and in 2019, neared the case rate in the rest of the U.S. While cases of invasive *Haemophilus influenzae* disease (IHiD) remained relatively stable in Oregon in 2019, there were no cases of *Haemophilus influenzae* serotype b (Hib) infection. Until the advent of an effective vaccine against Hib, *H*. *influenzae* was the leading cause of bacterial meningitis in children under 5 years of age in Oregon and elsewhere. Continued use of conjugate vaccine will help ensure Hib infection remains minimal well into the future. Cases of yersiniosis in Oregon have been elevated since 2013 and climbed further in 2019, reaching 53 cases. The increase in cases spanned all age, race, and sex categories.

CONCLUSION

Thank you for reporting to public health. Check out the complete 2019 report below.

FOR MORE INFORMATION:

- <u>2019 Communicable Disease An-</u> nual Report (Tableau)
- <u>Case counts by county of residence</u> (2019) (Tableau)
- <u>Select diseases by year (2000-2019)</u> (Tableau)

RESOURCES

- Centers for Disease Control and Prevention. National Notifiable Diseases Surveillance System, Weekly Tables of Infectious Disease Data. Atlanta, GA. CDC Division of Health Informatics and Surveillance. Available at: <u>www.cdc.gov/nndss/infectious-tables.html</u>.
- Centers for Disease Control and Prevention. Widespread outbreaks of Hepatitis A across the United States. Available at: <u>www.cdc.gov/hepatitis/</u> <u>outbreaks/2017March-HepatitisA.htm</u>



This activity has been planned and implemented in accordance with the accreditation requirements and policies of the Accreditation Council for Continuing Medical Education (ACCME) through the joint providership of Providence Oregon Region and the Oregon Pathologists Association. Providence Oregon Region is accredited by ACCME to provide continuing medical education for physicians.

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LOVE AT FIRST BITE: VECTOR-BORNE DISEASE IN OREGON

As Gregor Samsa awoke one morning from uneasy dreams, he found himself transformed in his bed into a gigantic insect.

- Franz Kafka, The Metamorphosis

amsa's experience would put a crimp in just about anyone's day, but there are other interactions with arthropods that can be just as unpleasant. In any case, the winter is past, the rain is over and gone; the flowers appear on the earth, the time of the singing of birds is come, the voice of the turtle is heard in our land ... and before too long, there will be so many mosquitoes, ticks, and other disease vectors out there it will make you long for winter again. Moreover, the geographic ranges of these arthropods are changing with the climate, bringing some diseases to parts of Oregon that haven't seen them before. In this CD Summary, we'll talk about where vectors are, what diseases they carry, and how to assess and protect your patients.

While we generally worry about only three species of ticks in Oregon, many species of mosquitoes from three genera thrive here. Several Culex species are found in eastern Oregon, and they regularly test positive for West Nile virus. Happily, none of the three Aedes species mosquitoes known to transmit dengue virus (A. albopictus, A. aegypti, A. japonicus) is native to Oregon, but they have been reported in neighboring California. Anopheles mosquitoes that could transmit malaria are found in Oregon, but imported cases are sufficiently uncommon and parasitemia is short-lived (thanks to treatment) that we don't fret too much about the possibility of autochthonous transmission.

Table. Diagnostic techniques for vector-borne disease

		Diagnosis by		
Disease	Vector	Clinical and exposure	Pathogen detection*	Antibody detection
Dengue	Mosquito		\checkmark	
Malaria	Mosquito	\checkmark		
West Nile	Mosquito			\checkmark
Zika	Mosquito		\checkmark	\checkmark
Anaplasmosis or Ehrlichiosis	Tick	\checkmark		
Babesiosis	Tick	\checkmark		
Colorado tick fever	Tick	✓		
Lyme	Tick			\checkmark
Plague	Flea	\checkmark		
RMSF	Tick			\checkmark
Tick-borne relapsing fever	Tick		~	✓
Tularemia	Tick	✓		

*culture or detection of pathogen-specific antigen or nucleic acid

WHERE ARE THE ARTHROPOD VECTORS?

Vector control folks from around the state tell us that ticks and mosquitoes were out earlier in the spring of 2018 than in years past. There are three main tick species in Oregon (see maps in references). Each has different ranges. The brown dog tick (Rhipicephalus sanguineus) is present throughout Oregon and can carry Rickettsia rickettsii, the pathogen that causes Rocky Mountain spotted fever (RMSF). The Rocky Mountain wood tick (Dermacentor andersoni) is found east of the Cascades, usually above 4,000 feet, though there are recent reports of exposures below that elevation. Its bite can result in RMSF, Colorado tick fever. and tularemia. West of the Cascades, you'll find the Western blacklegged tick (Ixodes

pacificus), which carries the agents of Lyme disease, anaplasmosis, and relapsing fever.

WHERE ARE THE DISEASES?

From 2014–2018, 1,348 cases of vector-borne disease were reported to the Oregon Health Authority: 255 confirmed, 355 presumptive, 738 suspect. Lyme disease accounted for more than half of these reports with 44 confirmed cases, 258 presumptive cases, and 530 suspect cases. Among confirmed cases of vector-borne disease were also 87 cases of malaria, 53 cases of Zika, 14 cases of tularemia, and 11 West Nile disease cases.

Following their vectors, three diseases have strong regional concentration in Oregon. Of the 288 confirmed and presumptive Lyme disease cases with location information, 248 resided west of the Cascades. All seven cases of Colorado tick fever all of which were confirmed — lived in central Oregon. Corresponding with mosquito testing, eight of the 11 confirmed and nine of the 10 presumptive cases of West Nile virus disease resided east of the Cascades.

Tularemia cases were split, with seven cases on each side of the Cascades. Remember that tick bites aren't the only way to get tularemia; 10 of 14 reported cases denied having seen a tick prior to their illness.

You might be surprised to see any cases of malaria and Zika in Oregon. Neither disease is endemic to Oregon, or the United States. Remember that we report not what was contracted here, but what was diagnosed among Oregon residents; all Oregonians with confirmed malaria, chikungunya, and dengue fever who could be interviewed had traveled to a country where the diseases are endemic. The same is true for all cases of Zika. except for one confirmed case that was transmitted by sexual contact and three presumptive cases of vertical transmission.

EMERGING TICK-BORNE DISEASES

Rickettsia sp. 364D causes Pacific Coast tick fever, which often presents with an eschar, fever, and headache; rash is relatively uncommon. The vector (Pacific Coast tick, *Dermacentor occidentalis*) is present in California and may have pushed into southwestern Oregon. Efforts to look for this potential emerging pathogen in Oregon are underway. Since it's relatively new, diagnostic capabilities for *R*. 364D infection are still expanding.

Borrelia miyamotoi is a species of spiral-shaped bacteria that also causes tick-borne relapsing fever (TBRF). As you can guess from the name, it is related to *B. buradorferi*. which causes Lyme disease. First identified in 1995 in ticks from Japan, B. miyamotoi has since been detected in one species of tick found in Oregon - viz., the western blacklegged tick (Ixodes pacificus). B. mivamotoi infection presents with nonspecific symptoms, including fever, headache, chills, myalgia, and arthralgia. Laboratory confirmation of B. miyamotoi is possible by PCR on blood from acutely symptomatic

patients; they may be seronegative at presentation.

Oregon Health Authority and Jackson County Vector Control officials collected 2,166 ticks in 459 vials between November 2017 and May 2018 at different locations countywide. *B. burgdorferi* was identified in 12/459 (2.6%, similar to that found in a study in 2000), and *B. miyamotoi* was identified in 37/459 vials (8.1%, no previous studies done in Oregon). We continue to collect *Ixodes pacificus* ticks, which live west of the Cascades, to learn more about the distribution of these pathogens.

HOW SHOULD I ASSESS MY PATIENT?

People do not always feel ticks and insect bites. For example, 22 of the 31 people with confirmed Lyme disease who were interviewed denied having seen a tick prior to their illness. Many early symptoms of vector-borne diseases are non-specific, including fatigue, chills, fever, and headaches. It is therefore important to ask all patients with such symptoms about travel history, time spent outdoors, and exposures to animals, including pets.

Some diseases can be identified by typical signs, such as erythema migrans ("bulls-eye" rash) with Lyme disease, and painful lymphadenitis with the bubonic form of plague. Many vector-borne diseases will cause typical hematologic changes; consult your favorite resource for details. The Centers for Disease Control and Prevention (CDC) has providerspecific pages for many diseases, and Heymann's Control of Communicable Diseases Manual is a handy resource.

As with all diseases, clinical presentation will guide you toward a diagnosis. Methods for definitively identifying one of these diseases fall into one of three categories: clinical presentation and exposure history, detecting the agent, or detecting the immune response. Consult the Table (*verso*) for details. Your clinical laboratory and the Oregon State Public Health Laboratory are excellent resources for determining which samples to collect.

PREVENTING VECTOR-BORNE DISEASE

The best way to prevent arthropodborne disease is to prevent being bitten. Insect repellent is very helpful

against both mosquitoes and ticks. Check the link below for the Environmental Protection Agency's (EPA's) insect repellent chooser to find those that will meet your patients' needs. Wearing long sleeves and pants can help prevent bites. It's also important to research areas before travel: check sites like CDC's Travelers' Health to see which diseases are present and which prevention methods are recommended at your destination. We also ask that you counsel patients who develop illnesses like dengue. Zika etc., to avoid mosquito bites for two weeks after symptom onset. (There is no evidence that mosquitoes native to Oregon can transmit dengue or Zika, but we don't want to find out the hard way.)

While mosquitoes generally stay outside, fleas are not above taking a free ride in with pets, and ticks may still be attached to patients and pets when they return indoors. Patients should check their body, clothing, gear, and pets for ticks. Potential problem hiding areas include the hair, ears, armpits, belly button and backs of the knees.

The proper way to remove a tick is to grasp the tick with forceps as close to your skin as possible and pull straight out (Figure). Do not twist. Repeat as necessary. Remedies from old wives' tales, such as burning a tick out or "painting" it with acetone are likely to increase the risk of disease transmission.

Your patients can help prevent disease in their pets and themselves by keeping their pets up to date on flea and tick repellent. Patients should work with their veterinarian to choose the appropriate product.

Figure. The proper way to remove a tick.

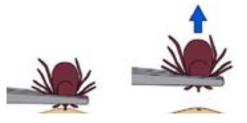


Image source: (accessed 3/17/2019) www.cdc.gov/lyme/removal/index.html



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MORE INFORMATION

- CDC's Division of Vector-Borne Diseases: www.cdc.gov/ncezid/dvbd/index.html
- ArboNET Disease Maps: <u>https://</u> wwwn.cdc.gov/arbonet/maps/ADB_ Diseases Map/index.html
- CDC maps: Geographic distribution of ticks that bite humans: <u>www.cdc.gov/ticks/geographic</u> <u>distribution.html</u>
- EPA insect repellent search tool: <u>www.epa.gov/insect-repellents/find-repellent-right-you</u>
- CDC Travelers' Health: <u>https://wwwnc.cdc.gov/travel</u>
- 2004 CD Summary on tick-borne disease:

www.oregon.gov/OHA/PH/ DISEASESCONDITIONS/ COMMUNICABLEDISEASE/ CDSUMMARYNEWSLETTER/ Documents/2004/ohd5309.pdf

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DISEASE OUTBREAKS IN OREGON, 2019

A n "outbreak" is the occurrence of a specific disease in time and space that is greater than what we would normally expect. In Oregon, any outbreak of illness is reportable to public health so that public health can investigate to determine its cause and to intervene to prevent further spread of illness. Interventions might include recalling contaminated food items, warning the public, providing targeted education, or immunizing susceptible contacts.

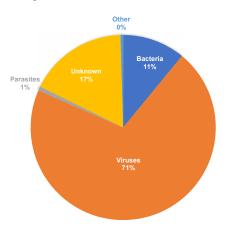
OUTBREAK OVERVIEW

Outbreaks are often first reported to public health when someone notices an increase in persons with a clinical syndrome such as gastroenteritis, respiratory or neurological illness, or rash. This initial clinical information guides the public health investigation. One of the basic tenets of outbreak investigation is to confirm the diagnosis. so a priority for outbreaks of infectious diseases is specimen collection and laboratory testing to identify a specific organism. The Oregon State Public Health Laboratory (OSPHL) routinely performs whole genome sequencing on enteric pathogens to identify cases with closely related genetic ancestrythe presumption being that genetically related isolates stemmed from a common source. We also find it helpful to classify outbreaks based on the primary mode of transmission, such as foodborne (spread through a contaminated food vehicle), person-to-person (including physical contact and droplet spread), water-borne, vector-borne or via contact with infected animals. The specific pathogen and its mode of transmission guide the health investigation and interventions.

2019 BY THE NUMBERS

Oregon state and local public health authorities logged 429 outbreaks of disease in 2019, an increase of 24% from the 346 investigated in 2018. The outbreaks investigated in 2019 affected at least 7,374 people. Viruses caused 303 (71%) of the 429 outbreaks (Figure 1). Next were bacteria, including bacterial toxins, which caused 47 (11%) of the outbreaks; and parasites, which caused 3 (0.7%). Two outbreaks in 2019 were caused by other agents: an outbreak of scabies (actually an infestation by the mite Sarcoptes sca-biei var. hominis rather than an "infec-tion"); and an outbreak of lung injury associated with vitamin E acetate in e-cigarette or vaping products.1

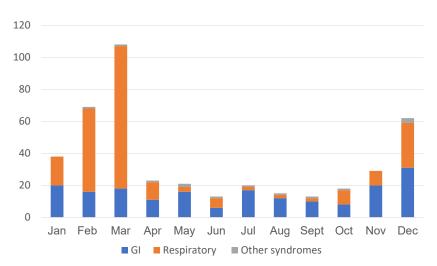
Figure 2 shows the number of outbreaks investigated in Oregon in 2019 by month and reported syndrome. While the number of gastrointestinal (GI) outbreaks remained somewhat stable throughout the year, most respiratory outbreaks in 2019 were investigated from January to March and in December, consistent with the Figure 1. Outbreaks by pathogen or agent, Oregon, 2019



influenza season. Of the 187 respiratory outbreaks investigated during those months in 2019, 162 (87%) of them were influenza.

By clinical syndrome, the most commonly reported outbreak was of respiratory illnesses: 231 (54%). Of these, the predominant etiology was influenza, causing 179 (77%) of these outbreaks and affecting at least 2,606 people. Influenza A accounted for 133

Figure 2. Number of outbreaks by month and clinical syndrome, Oregon, 2019

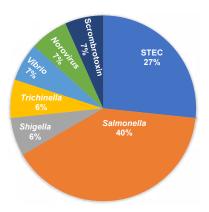


(74%) of these outbreaks and influenza B for 16 (9%). Consistent with most seasons, the 2018-19 influenza season in Oregon began with a wave of influenza A cases, which continued to predominate throughout the season. The 2019-2020 influenza season, however, reversed this trend and began with a large wave of influenza B followed by a smaller wave of influenza A.² Two outbreaks involved both influenza A and influenza B, and the type was unknown in 26 (15%) influenza outbreaks. The great majority of influenza outbreaks investigated in 2019 were in long-term care facilities (118, 66%) and schools (41, 23%).

Other respiratory outbreaks in 2019 were caused by measles (4), mumps (1), pertussis (22) and respiratory syncytial virus (RSV) (12).

Forty-three percent (185) of the reported outbreaks presented as gastroenteritis. Consistent with outbreaks in previous years, norovirus was the etiologic agent in the majority of these, causing 96 (52%) of 185. Salmonella and Shiga-toxin producing Escherichia coli (STEC) caused 14 (8%) and 5 (3%), respectively. Among the strains typed in 19 norovirus outbreaks, genogroup II were the most common (74%), consistent with what was observed nationally.3 Among the fifteen foodborne GI outbreaks, Salmonella was confirmed as the etiologic agent in six and STEC in four (Figure 3).

Figure 3. Etiology of foodborne outbreaks, Oregon, 2019



NOTABLE OUTBREAKS

STEC in ground beef In November 2019, isolates from three cases of STEC O157:H7 in neighboring counties had similar molecular typing based on whole genome sequencing (they were identical). Over the following three weeks, four additional cases were identified among household contacts and through reports from astute clinicians. Each had purchased ground beef from the same grocery market chain, so a multi-agency outbreak investigation was initiated.⁴ Ground beef samples from multiple market locations were genetically similar to the clinical cases. Within three days of identifying the initial cluster, the grocery chain issued a recall of in-house ground beef from all 26 store locations; the recall was later expanded to all beef products. The Oregon Health Authority issued two press releases and used loyalty cards to identify other customers who had purchased beef. From an online customer survey, 56 more people with symptoms consistent with STEC infection were identified, for a total of 63 Oregon cases. None of these 56 cases had been tested for STEC. The cases ranged from 2 to 77 years of age (median, 44); 59% were female. Two cases were hospitalized for their illness, and one developed hemolytic uremic syndrome. None died. The investigation revealed that the market was unknowingly grinding beef that the processing facility intended only for intact use (to be sold e.g., as steaks or roasts) and was therefore not required to be tested for STEC. This misunderstanding highlights an opportunity for education and the need for improved communication between beef suppliers and their buvers.

A tale of two restaurants In August 2019, an Oregon local public health authority (LPHA) received reports of five cases of Salmonella typhimurium in a single day, prompting investigation. Another five cases had been reported in a neighboring county during the previous week. Over the following three weeks, ten more cases were reported to these LPHAs. Initial interviews identified no shared exposures, but genetic sequencing of case isolates found them to be closely related. In follow-up interviews, cases reported eating at one of two restaurants (one in each county). The two Mexican restaurants had a common food supplier from whom they ordered similar food items. Public health outreach to

other customers identified three more who experienced symptoms consistent with salmonellosis, bringing the count to 23 *Salmonella* serotype Typhimurium cases in this outbreak. The cases ranged from 5 to 86 (median, 41) years of age, and 11 (48%) were female. Two cases were hospitalized for their illness; none died. Due to substantial overlap in the two restaurants' orders from the supplier and in cases' food purchases, no single food item could be identified as the cause of this outbreak.

Bears—oh my!

In August 2019, a clinician contacted the LPHA after diagnosing trichinellosis based on a patient's clinical presentation and recent exposure to bear meat. Trichinellosis is contracted by eating raw or undercooked meat of animals infected with larvae of roundworms of the genus Trichinella. While the clinical manifestation varies, cases often experience fever and myalgia and have periorbital edema and eosinophilia. In the U.S. today, trichinellosis remains relatively uncommon. Review of the medical record revealed that the patient had become sick along with three others who attended a gathering where they consumed burgers of ground bear meat. The four all became ill within three days of consuming the bear meat, and three were hospitalized for their illnesses. Four samples of leftover bear meat were tested, and Trichinella murrelli larvae were identified in all four, ranging from 2-19 larvae per gram. Thanks to prompt reporting by an astute clinician, the leftover bear meat was discarded, and others were spared the illness.

Expounding the EVALI enigma Beginning in August 2019, multiple states across the U.S. began noticing cases of severe lung injury among young persons, often previously healthy, who reported using e-cigarettes or vaping devices; soon the condition was dubbed "e-cigarette or vaping product-associated lung injury" ("EVALI"). In total the outbreak affected 2,807 people in all 50 states, the District of Columbia and two U.S. territories. All cases were hospitalized, and there were 68 confirmed deaths. Oregon made EVALI reportable on October 9, and ultimately logged 23 cases and two

deaths.1 The median case age was 34 in Oregon and 24 in the U.S. CDC led a national investigation that included case interviews, medical record reviews, extensive clinical evaluations and testing of leftover vaping products.⁵ Laboratory data found vitamin E acetate in the products to be strongly associated with illness; however, a role for other chemicals in these products has not been ruled out. Vitamin E acetate was found in product samples tested by FDA and in 48 of 51 bronchoalveolar lavage fluid samples from EVALI patients but in 0 of 99 healthy controls.⁶ Thanks to increased awareness of the risk associated with ecigarette or vaping product use and the removal of vitamin E acetate from some products, cases of EVALI have since declined in the U.S. Due to this decline, mandatory reporting of EVALI to public health authorities in Oregon expired in February 2020.

Problems with pertussis Twenty-two outbreaks of pertussis were reported in Oregon in 2019nearly double the 12 reported in 2018. Outbreaks of pertussis in 2019 comprised 155 cases, ranging from 0 to 70 (median, 15) years of age. Females accounted for 54% of outbreak cases. The outbreaks did not result in any hospitalizations or deaths. The largest pertussis outbreak in 2019 was a community-wide outbreak of 42 cases. Investigations began in May 2019 when two cases of pertussis were reported in a single high school. Four additional cases were reported throughout May at different schools in the county, leading public health officials to classify this as a communitywide outbreak: and over the course of the summer, 34 additional cases were identified in multiple schools, camps and community sports clubs, among other locations. The cases ranged from 3 to 25 (median, 15) years of age, and 52% were female. Of the 40 cases less than 19 years old, 85% were up to date on pertussis vaccination. The outbreak was determined to be over in August 2019 when cases in the community had returned to baseline levels. Unfortunately, communitywide outbreaks of pertussis are not uncommon in Oregon: an even larger one was ignited in 2018 by cases in several high-school populations, resulting in a total of 227 cases. Pertussis outbreaks in previous years have

also consisted primarily of young adult cases.

Measles mayhem

After historically low case counts during the earlier years of the millennium, measles cases spiked in 2019 with 28 cases reported in Oregon-the most since 1991. This was consistent with outbreaks observed in the rest of the United States in 2019. Four Oregon measles outbreaks accounted for 27 of the 28 cases. In February 2019, public health officials investigated an outbreak of measles at a missionary training school after members were exposed to a case from out of state; ultimately, the outbreak comprised nine Oregon cases. In August 2019, a 10case outbreak began with a case acquired during international travel; nine secondary cases resulted from subsequent public exposures and close contact among household members, relatives, and others. A four-case outbreak was associated with exposure to an infectious case on an international flight. Finally, four Oregon cases of measles were associated with a large outbreak in Clark County, Washington in early 2019.7 The median age of all 28 measles cases in Oregon in 2019 was 10.5 years, and 79% were female. All cases were unvaccinated.

MORE HIGHLIGHTS FROM 2019

This CD Summary covers only a small portion of the disease outbreaks investigated in 2019; space precludes a discussion of the many other notable investigations. Oregonians were involved in six separate multi-state outbreaks of Salmonella infections associated with live poultry,8 each associated with a different Salmonella serotype, including 17 Oregon cases in all. CDC also investigated a multi-state outbreak of Vibrio parahaemolyticus infections that included 13 cases from Oregon, associated with consumption of raw oysters from various harvest locations. A five-case outbreak of hepatitis A occurred among persons reporting injection drug use and unstable housing. The index case in the outbreak had been exposed to hepatitis A in California. In response, multiple local vaccination clinics were organized to immunize this high-risk population.

CONCLUSION

These data would not exist without astute clinicians reporting, local and tribal public health jurisdictions investigating and reporting of outbreaks to the Oregon Health Authority's Public Health Division. If you suspect an outbreak of any illness, please alert your local public health authority (LPHA) promptly. Contact information for Oregon LPHAs can be found at <u>www.healthoregon.org/lhddirec-</u> tory.

FOR MORE INFORMATION

- List of 2019 Disease Outbreaks
 (Tableau)
- 2019 Communicable Disease Annual Report (Tableau)
- <u>ACDP's outbreak investigation</u>
 <u>webpage</u>

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A TALE OF TWO RESPIRATORY PATHOGENS: INFLUENZA AND SARS-COV-2

2016-2017, 2017-2018, 2018-2019, 2019-2020, 2020-2021

It was the best of times, it was the worst of times, it was the age of wisdom, it was the age of foolishness, it was the epoch of belief, it was the epoch of incredulity, it was the season of Light, it was the season of Darkness. – Charles Dickens

INFLUENZA DURING THE COVID-19 PANDEMIC

We entered the 2020-2021 respiratory viral pathogen season with a fear of the unknown: What would an influenza season superimposed on the COVID-19 pandemic look like? What would be the consequences of influenza and SARS-CoV-2 coinfection? Given that United States hospitals admit between 140,000 and 810,000 influenza hospitalizations annually¹ what would happen to hospital capacity? As we braced for a tumultuous season, CO-VID-19 mitigation strategies remained in place, with mask wearing and social distancing becoming a part of daily life. The results of these mitigation efforts were astounding-the southern hemisphere experienced record low influenza circulation,² and we subsequently experienced an absence of influenza throughout the season.

INFLUENZA CIRCULATION IN OREGON: PAST VS. PRESENT

The 2020–2021 influenza season saw historically low influenza circulation across Oregon. The Figure shows the percentage of hospital emergency department visits due to influenza-like illness (ILI) as captured by the Oregon Electronic Surveillance System for the Early Notification of Community-based Epidemics (ESSENCE). Throughout the 2020-2021 influenza season, the percentage of such visits attributable to ILI never exceeded 1.2%. Traditionally, a baseline of 2.6% is used to determine the beginning of influenza circulation.³

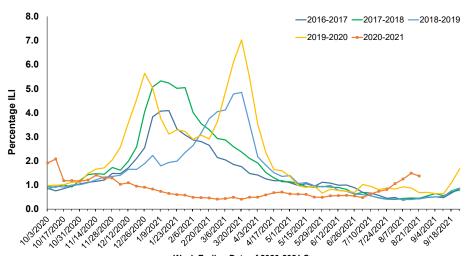


Figure. Percentage of ED Visits for ILI, Oregon ESSENCE Syndromic Surveillance,

Week Ending Date of 2020-2021 Season

Laboratory testing data from the National Respiratory and Enteric Virus Surveillance System (NREVSS) confirmed limited influenza circulation throughout 2020–2021 season. Only 0.1% of all influenza specimens tested at 22 Oregon laboratories reporting to NREVSS tested positive for influenza throughout the season. In comparison, the 2019–2020 season saw a 16.5% influenza test positivity. Influenza activity was low across the United States and globally despite adequate testing. Nationally, there was only one influenza-associated pediatric death, compared to 199 in the preceding season.4

Why did influenza disappear during the COVID-19 pandemic? While we can't say for sure, the answer most likely lies in the mitigation measures put in place to reduce the spread of COVID-19. Community-wide restrictions, basic public health interventions such as masking and hand washing, and behavioral changes such as avoiding social gatherings and staying home while sick all probably helped reduce influenza circulation. Record high influenza vaccine distribution, with 193.8 million doses distributed,⁴ also probably played an important role. These measures taken to lessen the spread of COVID-19 and protect the capacity of our healthcare systems proved effective across respiratory viral pathogens.

INFLUENZA AND COVID-19 HOSPITALIZATIONS

Oregon is one of 14 states that participates in the Centers for Disease Control and Prevention (CDC) Emerging Infections Program (EIP) hospitalization surveillance for influenza (FluSurv-NET) and SARS-CoV-2 (COVID-NET). The FluSurv-NET and COVID-NET surveillance networks identify residents of Clackamas, Multnomah, and Washington counties who are hospitalized within 14 days of a positive laboratory test for influenza or SARS-CoV-2, respectively. Detailed chart reviews are conducted to collect patient risk factor and outcome information.

Given what we've already shared above, this might not surprise you, but only three influenza hospitalizations were captured by FluSurv-NET during the 2020-2021 season. This compares with over 5,000 COVID-NET hospitalizations since the inception of COVID-NET in March 2020. COVID-19-related hospitalizations peaked at 209 during the week of November 11, 2020. Although COVID-19 hospitalizations initially waned as COVID-19 vaccines became broadly available, hospitalizations have increased dramatically in the wake of emerging variants such as B.1.617.2 (Delta) and modest vaccine uptake. The stark dichotomy between these influenza and COVID-19 hospitalization numbers suggests a couple of important take-home messages: 1) the protective measures put in place to mitigate the spread of COVID-19 are highly effective at reducing the transmission of many respiratory viral pathogens, 2) comparing seasonal influenza with pandemic SARS CoV-2, the virus that causes COVID-19, it is clear that a little residual immunity in a population goes a long way. What is less clear as we enter the 2021-2022 season is how influenza transmission will change with fewer COVID-19 mitigation measures in place.

THE 2021–2022 INFLUENZA SEASON

COVID-19 vaccines have been at the forefront of immunization planning, as nearly 170 million individuals have been fully vaccinated in the United States.⁵ Vaccination remains our strongest defense against COVID-19, but routine vaccination against other pathogens remains crucial. Vaccinations for both COVID-19 and influenza help protect both individuals and our health care system by preventing severe illness, hospitalization, and death. Influenza vaccinations for the upcoming season have been updated to match the viruses in circulation. This season's influenza vaccine varieties feature updated Flu A (H1N1) and A (H3N2) components (Table).⁶ Vaccination against influenza continues to be recommended for all individuals 6 months of age and older.

As in recent flu seasons, all regulardose vaccines will be quadrivalent. Live-attenuated influenza vaccine (LAIV) will also be available. LAIV is not recommended for immunocompromised individuals, close contacts of Table. 2020–2022 influenza vaccine components

Influenza vaccine strains northern hemisphere, 2020–2021 Season

Strain	Egg-based vaccines	Cell or recombinant- based vaccines
A/H1N1	A/Victoria/2570/2019 (H1N1)pdm09-like	A/Wisconsin/588/2019 (H1N1)pdm09-like
A/H3N2	A/Cambodia/ e0826360/2020 (H3N2)-like	A/Cambodia/ e0826360/2020 (H3N2)-like
B/Victoria	B/Washington/02/2019- like (B/Victoria lineage)	B/Washington/02/2019-like (B/Victoria lineage)
B/Yamagata	B/Phuket/3073/2013-like (B/ Yamagata lineage)	B/Phuket/3073/2013-like (B/Yamagata lineage)

severely immunosuppressed persons, pregnant women, children 2-4 years of age with asthma, children receiving salicylates (aspirin), or persons who have recently received influenza antiviral medication. Those who have received influenza antiviral medication should wait 48 hours after taking oseltamivir and zanamivir, 5 days after peramivir, and 17 days after baloxavir7 before taking LAIV. Licensure of the Flucelvax Quadrivalent vaccine has been updated this year and is now approved for people 2 years and older. Influenza vaccine manufacturers do not expect any delays in production or distribution of this year's vaccine supply.⁶ Individuals can receive COVID-19 vaccines and influenza vaccines at the same visit, and no longer need to wait 14 days between vaccines.8

FOR ADDITIONAL INFORMATION

- Oregon FluBites: http://bit.ly/flubites
- CDC FAQ for the 2021–2022 Influenza Season: <u>www.cdc.gov/flu/season/faq-</u> <u>flu-season-2021-2022.htm</u>
- CDC FluView: <u>https://gis.cdc.gov/</u> grasp/fluview/fluportaldashboard.htm/
- CDC COVID Data Tracker Weekly Review: <u>www.cdc.gov/coronavirus/2019-</u> ncov/covid-data/covidview/index.html

REFERENCES

- 1. CDC. Disease Burden of Influenza. www.cdc.gov/flu/about/burden/index. html?web=1&wdLOR=c25E02630-D5C4-455C-A625-68E3287AE8C8. Updated June 11, 2021.
- Olsen SJ, Azziz-Baumgartner E, Budd AP, et al. Decreased influenza activity during the COVID-19 pandemic — United States, Australia, Chile, and South Africa, 2020. MMWR 2020;69:1305–9.
- CDC. Weekly U.S. Influenza Surveillance Report. <u>www.cdc.gov/flu/weekly/index.</u> <u>htm?web=1&wdLOR=cE7064CF0-72BB-44C0-8CFE-3B49E1B9C4E5</u>. Accessed August 23, 2021.
- CDC. 2020–2021 Flu Season Summary. <u>www.cdc.gov/flu/season/faq-flu-sea-</u> <u>son-2020-2021.htm</u>. Updated July 22, 2021.
- CDC. COVID Data Tracker. <u>https://covid.cdc.</u> <u>gov/covid-data-tracker/#vaccinations_vacc-</u> <u>total-admin-rate-total</u>. Accessed August 23, 2021.

- CDC Frequently Asked Influenza (Flu) Questions: 2021–2022 Season. <u>www.cdc.gov/</u> <u>flu/season/faq-flu-season-2021-2022.htm</u>. Updated August 6, 2021.
- CDC. Live Attenuated Influenza Vaccine [LAIV] (The Nasal Spray Flu Vaccine). <u>www.</u> <u>cdc.gov/flu/prevent/nasalspray.htm</u>. Updated May 6, 2021.
- CDC. Getting Your COVID-19 Vaccine. <u>www.</u> <u>cdc.gov/coronavirus/2019-ncov/vaccines/</u> <u>expect.html</u>. Updated August 19, 2021.

OREGON WANTS YOU!

The Oregon Health Authority is asking Oregon licensed healthcare professionals to register with the State Emergency Registry of Volunteers in Oregon (SERV-OR). Volunteers receive notifications about deployment opportunities supporting urgent medical surge issues across the state. Visit SERV-OR to sign up for a local Medical Reserve Corps unit and the State Managed Volunteer Pool, today.

SERV-OR is a statewide registry for licensed health care professionals willing to volunteer for Federal, State, or local public health and medical emergencies

Hospitals and health facilities in Oregon need your expertise. You may be asked to:

- Staff facilities including clinical sitting, ICU, inpatient care, alternate care sites, med surge, outpatient care, etc.
- Fill administrative or clinical roles
- Provide COVID vaccination and testing

<u>Register today!</u> And thank you for doing your part to keep everyone in Oregon safe.

Visit the <u>COVID FAQ</u> and <u>Pro-</u> <u>gram FAQ</u> pages to learn more.



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You can get this document in other languages, large print, braille or a format you prefer. Contact the Public Health Division at 971-673-1222. We accept all relay calls or you can dial 711. for TTY.

03/03/2021

	Usual					Criteria for Confirmation in an Outbreak Setting
Agent	Incubation Period (Range)	Symptom Profile	Communicable Period	Mode of Transmission	Reservoirs	OSPHL Specimen Submission Requirements Use these instructions with caution. Current guidance is
_						available at www.healthoregon.org/labtests.

I. Ag	ents typified by	r fever ≥100°F wi	th cough and syste	mic symptoms	(chills, headad	che, myalgia, malaise, anorexia)
Influenza (A, B, C)	2 days (1–4 days)	Fever, cough, coryza, myalgia, prostration Vomiting, diarrhea in children	3–7 days	Mostly droplet; maybe via aerosol or contaminated surfaces	Humans, but transmission of novel viruses from birds & various mammals is possible	Positive rapid test (sensitivity 50-70%), RT-PCR or isolation of virus on culture (rarely performed at clinical labs).

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Agent	Usual Incubation Period (Range)	Symptom Profile	Communicable Period	Mode of Transmission	Reservoirs	Criteria for Confirmation in an Outbreak Setting OSPHL Specimen Submission Requirements Use these instructions with caution. Current guidance is available at www.healthoregon.org/labtests.
I. Agen	ts typified by	fever ≥100°F wi	th cough and syste	mic symptoms	(chills, headad	che, myalgia, malaise, anorexia) (continued)
Adenovirus (several serotypes. Adeno 7 and 14 circulate in Oregon and have caused several outbreaks. Adeno 7 is associated with severe infections.)	2–14 days	Depending on serotype, can present as sore throat, "croup" with runny nose in kids; serotype 14 commonly causes fever, cough, headache, muscle aches; occurs any time of year.	"Shortly" before onset & for duration of symptoms	Respiratory droplets, can be fecal/oral	Humans	 PCR testing for adenovirus and specifically for adenovirus serotype 14 are available. Preferred: using Dacron polyester or flocked swabs on a plastic shaft, collected 3-7 days after illness onset. Submit swabs in viral transport media. Store and transport specimens at refrigerated temperatures for receipt at the OSPHL within 3 days of specimen collection Acceptable: nasal swabs, throat swabs, combination nasopharyngeal swabs (2 swabs in one vial), nasal aspirates, nasal washes, bronchoalveolar lavages, bronchial washes, tracheal aspirates, sputum, lung tissue, or cell culture isolates
Haemophilus influenzae	Unknown (probably 2-4 days)	Abrupt onset fever, anorexia, vomiting, cough, lethargy. Headache, stupor suggest meningitis.	As long as organism is present in discharges from nose or throat. Exposure >7 days before symptom onset in case imparts low risk. Hib cases most infectious during the 3 days prior to sx onset.	Droplet	Humans	Culture of <i>H. flu</i> from a normally sterile site. Original testing laboratory will send specimen to the OSPHL. OSPHL accepts actively growing isolated organism in pure culture, on an agar slant, or plate media. Primary specimens from sterile sites are accepted if previously tested using culture- independent diagnostic tests and approved for forwarding to the OSPHL. Store and transport at ambient or refrigerated temperatures for receipt at the OSPHL within 24 hours of culture.

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I. Agen	I. Agents typified by fever ≥100°F with cough and systemic symptoms (chills, headache, myalgia, malaise, anorexia) (continued)								
Human meta- pneumovirus	2-8 days	Runs the gamut from mild URI to severe pneumonia, the latter more common in the elderly	Not well defined	Droplet, Contact	Humans	Viral culture or either RT-PCR or IFA of cell supernatant. ************************************			
Streptococcus pneumoniae (Pneumococcus)	Unknown (probably 1-4 days)	Productive cough, fever/chills, shortness of breath, chest pain. People look sick! Often follows viral infection.	As long as the organism appears in respiratory secretions.	Droplet	Humans (carriage is more common in children than in adults)	Isolation on culture from sputum, though this can be difficult. Characteristic gram-positive diplococci on gram stain of sputum is suggestive. ************************************			

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Agent I.	Usual Incubation Period (Range) Agents typified	Symptom Profile by fever ≥100°F	Communicable Period with cough and sy	Mode of Transmission stemic sympton	Reservoirs ns (chills, head	Criteria for Confirmation in an Outbreak Setting ************************************
<i>Legionella pneumophila</i> Legionellosis, Legionnaires' disease, Pontiac fever	Legionn- aires' disease: 5–6 days Pontiac fever: 24 - 48 hours	Both present with anorexia, malaise, myalgia, headache, and fever. Legionnaires' disease is characterized by pneumonia and a non- productive cough. Pontiac fever usually is accompanied by cough but does not progress to pneumonia or death.	Person-to-person transmission has not been documented	Airborne, aspiration of contaminated water droplets	Water systems (potable, air conditioning, spas, decorative fountains)	Positive urine antigen or isolation on culture of respiratory secretions (culture for legionella must be specifically requested). In some cases, direct fluorescent antibody staining or paired serologies may confirm diagnosis. Note: these tests aren't available at OSPHL. ************************************

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Agent	Usual Incubation Period (Range)	Symptom Profile	Communicable Period	Mode of Transmission	Reservoirs	Criteria for Confirmation in an Outbreak Setting ************************************
I. Ag	ents typified		with cough and sy	stemic sympton	ns (chills, head	dache, myalgia, malaise, anorexia) (continued)
Coronavirus Middle Eastern Respiratory Syndrome (MERS) (Call ACDP, pronto, if suspected)	4-5 days (2-14 days)	Can range from asymptomatic to fever, cough, and chills, to severe respiratory distress. See: www.cdc.gov/ coronavirus/m ers/interim- guidance.html for testing criteria	Unknown	Unknown. CDC recommends standard, contact, and airborne precautions	Camels; appears trans- missible, with low infectivity, from person to person	Positive PCR on lower respiratory specimen, serum, or nasopharyngeal or oropharyngeal swab. ************************************
Coronavirus Severe Acute Respiratory Syndrome (SARS)	2-10 days	Fever, cough, rapidly progressing shortness of breath. CXR consistent with pneumonia or acute respiratory distress syndrome. Can also present with milder disease.	Poorly defined; may be up to 21 days.	Droplet, contact	Humans, Civets (not many around!)	Viral culture or PCR. Visualization of characteristic virus on electron microscopy. Detection of viral antigens on immunohistochemistry. ************************************

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Agent	Usual Incubation Period (Range)	Symptom Profile	Communicable Period	Mode of Transmission	Reservoirs	Criteria for Confirmation in an Outbreak Setting ************************************
Coronavirus Disease-19 (COVID-19)	4–7 days (2–14 d)	Fever, cough, anosmia, ageusia, rapidly progressing shortness of breath. CXR consistent with pneumonia or acute respiratory distress syndrome. Can also present with milder disease.	2 days before to 10 days after illness onset.	Droplet, contact, aerosol in certain indoor settings, particularly with poor ventilation	Humans, probably originally from bats	Positive nucleic amplification test (NAAT) or antigen test from any respiratory tract specimen.
	-	<u> </u>	Agents assoc	iated with sever	e disease in ir	nfants & children
Bordetella pertussis whooping cough pertussis	7–10 days	Paroxysmal coughing w/ whoop & vomiting	Highly contagious during 1st week of symptoms; negligible after 5 days of treatment.	Respiratory droplets or direct contact w/ respiratory secretions	Humans	See Pertussis Investigative Guidelines (pdf). ************************************

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Respiratory syncytial virus (RSV)Often 2 days (1-8 d)In infants, croup w/ barking cough, wheezing, inspiratory stridor; Older kids/adults have URI, cough; "season" is OctMayI -5 days after onset; longer (weeks) in infants & the immune- compromisedHumans, rarely contactRT-PCR, rapid antigen test, viral isolation ************************************	Agent	Usual Incubation Period (Range)	Symptom Profile	Communicable Period	Mode of Transmission	Reservoirs	Criteria for Confirmation in an Outbreak Setting ************************************
Respiratory syncytial virus (RSV)Often 2 days (1-8 d)In infants, croup w/ barking cough, wheezing, inspiratory stridor; Older kids/adults have URI, cough; "season" is OctMay1-5 days after onset; longer (weeks) in infants & the immune- compromisedHumans, rarely chimp- 							at refrigerated temperatures for receipt at the OSPHL within 3 days of specimen collection.
	syncytial virus	days (1-8	croup w/ barking cough, wheezing, inspiratory stridor; Older kids/adults have URI, cough; "season" is	onset; longer (weeks) in infants & the immune-		rarely chimp-	 ************************************

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Agent	Usual Incubation Period (Range)	Symptom Profile	Communicable Period	Mode of Transmission	Reservoirs	Criteria for Confirmation in an Outbreak Setting ************************************
Bacillus anthracis inhalational anthrax (Call ACDP, pronto, if suspected)	1–7 days (1–60 days)	Fever, malaise, mild cough, shortness of breath, headache, chills; <u>then</u> abrupt onset of sweats, spiking fever, ARDS & shock; medastinal widening, pleural effusions on CXR	Not communicable	Inhaling aerosols from tissues, hair, wool, hides of ill herbivores	Herbivores (cattle, goats, sheep, bison, etc.) Potential bio- terrorism agent	 Culture and identification from clinical specimens by Laboratory Response Network (LRN)5,6; Demonstration of B. anthracis antigens in tissues by immunohistochemical staining using both B. anthracis cell wall and capsule monoclonal antibodies; Evidence of a four-fold rise in antibodies to protective antigen between acute and convalescent sera or a fourfold change in antibodies to protective antigen in paired convalescent sera using Centers for Disease Control and Prevention (CDC) quantitative anti-PA immunoglobulin G (IgG) ELISA testing in an unvaccinated person; ************************************

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Agent	Usual Incubation Period (Range)	Symptom Profile	Communicable Period	Mode of Transmission	Reservoirs	Criteria for Confirmation in an Outbreak Setting ************************************
<i>Brucella</i> species brucellosis	2-4 weeks	Fever, chills, sweats, headache, myalgia, arthralgia, anorexia, fatigue, weight loss	Not well known: sexual and neonatal transmission have both been documented.	 Primarily foodborne Respiratory transmission is possible, (e.g. aerosolizing medical procedures, slaughter- houses). Contact with mucous membranes (handling infected animal tissues, blood, urine, vaginal discharges, aborted fetuses & placentas). Needle jabs with <i>Brucella</i> vaccine (RB51) Organ transplants 	Cattle, goats, pigs (including wild pigs), dogs, sheep, bison, marine animals Potential bioterrorism agent	Culture confirmation, 1:160 BMAT result following symptom onset, PCR, any paired, 4-fold increase in Brucella antibodies by nonagglutination-based tests **********************************

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Agent	Usual Incubation Period (Range)	Symptom Profile	Communicable Period	Mode of Transmission	Reservoirs	Criteria for Confirmation in an Outbreak Setting ************************************
<i>Chlamydia</i> <i>psittaci</i> psittacosis	10 days (5–14 d)	Acute onset fever, chills, headache, kerato- conjunctivitis, myalgia, rash, pneumonia w/o cough. CXR with lobar consolidation, patchy infiltrates	Minimal risk. Theoretically possible during paroxysmal coughing	Inhalation of desiccated bird feces, feathers, dust	Psittacine birds (parrots, parakeets, love birds), pigeons and some poultry (primarily turkeys & ducks; not much in chickens)	4-fold rise in psittacosis complement-fixing antibody titer (to ≥ 1:32) in specimens obtained > 2 weeks apart. PCR can be used in the acute stage of the disease in sputum, pleural fluid and blood. ***********************************
III.	Agents assoc	ciated with expo	sure to animals or	animal settings	(kennels, avia	ries, abattoirs, laboratories, etc.) (continued)

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Agent	Usual Incubation Period (Range)	Symptom Profile	Communicable Period	Mode of Transmission	Reservoirs	Criteria for Confirmation in an Outbreak Setting OSPHL Specimen Submission Requirements Use these instructions with caution. Current guidance is available at www.healthoregon.org/labtests.		
Francisella tularensis tularemia (Call ACDP, pronto, if suspected)	3–5 days (1 – 21 d)	Rapid onset high fever, chills, fatigue, pleuretic chest pain, dyspnea, lymphadenopa thy, myalgia, headache, malaise, mild cough; <u>then</u> pneumonia, ARDS	Not communicable	Inhaling infectious aerosols and contaminated dust generated while handling hides, carcasses, contaminated grain; animal or insect bite.	Lagomorphs (rabbits, hares, etc.), rodents, hard ticks, biting flies Potential bio- terrorism agent	Confirmed: Isolation by culture of <i>F. tularensis</i> in a clinical specimen, or a fourfold or greater rise in serum antibody titer to <i>F. tularensis</i> antigen between acute and convalescent titers. Presumptive: elevated titers to <i>F. tularensis</i> without documented fourfold change, in the absence of prior tularemia vaccination, or detection of <i>F. tularensis</i> in a clinical specimen by fluorescent assay. ************************************		
III. Agents associated with exposure to animals or animal settings (kennels, aviaries, abattoirs, laboratories, etc.) (continued)								

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Agent	Usual Incubation Period (Range)	Symptom Profile	Communicable Period	Mode of Transmission	Reservoirs	Criteria for Confirmation in an Outbreak Setting 	
Hantavirus hantavirus pulmonary syndrome	2–4 weeks	Fever, myalgia, GI pain; then abrupt onset ARDS, sepsis thrombocytop enia leukocytes, hemo- concentration; interstitial lymphocyte infiltrates, alveolar pulmonary edema	Rare person-to- person transmission	Inhaling aerosolized rodent excreta	Rodents	Four-fold rise in hantavirus EIA (reactive or not) test IgM and IgG; draw one sample acutely and, if negative, a specimen no sooner than 15 days from symptom onset & convalescent 2 weeks after acute specimen ************************************	
<i>Leptospira</i> spp. Leptospirosis Weil's disease (icteric)	7 to 12 days, with a range of 2 to 29 days	Sudden onset fever, headache, chills, myalgia in legs & conjunctival suffusion; <u>then</u> pneumonia, hemoptysis, ARDS	Rare person-to- person transmission	Contact with mucous membranes or ingestion	Rodents, racoons, livestock, dogs, amphibians, reptiles, sealions; animal products of conception or urine; contaminate d water, soil, mud	Indirect hemo agglutination (titer) ************************************	

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Agent	Usual Incubation Period (Range)	Symptom Profile	Communicable Period	Mode of Transmission	Reservoirs	Criteria for Confirmation in an Outbreak Setting OSPHL Specimen Submission Requirements Use these instructions with caution. Current guidance is available at www.healthoregon.org/labtests.							
Yersinia pestis pneumonic plague (Call ACDP, pronto, if suspected)	2–4 days (1–7 d)	Acute-onset of fever, chills, headache, malaise & myalgias; <u>then</u> cough w/ bloody sputum, pneumonia, ARDS, circulatory collapse & death	From onset of symptoms, usually w/in 24– 48hrs of exposure, until done w/ 72hrs of appropriate antibiotics	Person-to- person transmission via respiratory droplets aerosol (bioterrorism)	Fleas, wild rodents (rats, squirrels, prairie dogs), pets with fleas Potential bioterrorism agent	Isolation by culture from a sputum specimen; four-fold rise in serum antibody titer to Y. pestis F1 antigen in acute & convalescent serum specimens; antibody titer of ≥1:128 to Y. pestis F1 antigen not explained by past infection or vaccination; detection of F1 antigen in a clinical specimen by fluorescent assay **********************************							
		-		IV. Other pa	athogens								
Mycobacterium tuberculosis	See TB Guidelines: health oregon.org/ iguides	Classically cough, blood in sputum, fever, night sweats.	See TB Guidelines: healthoregon.org/ iguides	Aerosol	Humans, other mammals (elephants, cattle, some primates)	See TB Guidelines: healthoregon.org/iguides							
		1	IV. Oth	ner pathogens (continued)	IV. Other pathogens (continued)							

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Acute and Communicable Disease Prevention (ACDP): 800 NE Oregon St, Suite 772, Portland, OR 97232/971.673.1111

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Agent	Usual Incubation Period (Range)	Symptom Profile	Communicable Period	Mode of Transmission	Reservoirs	Criteria for Confirmation in an Outbreak Setting ************************************	
Mycoplasma pneumoniae	1-4 weeks	URI possible with cough/congest ion. Sub-acute "Walking pneumonia" in ~10% w usually non- productive cough/fever.	Unclear. Perhaps ten or more days after onset.	Droplet	Humans	Isolation on culture is not that easy. PCR may be helpful, as is a fourfold rise in complement fixation antibody titers on samples collected 4 weeks apart. ************************************	
Parainfluenza virus types 1-4	Often ~2 days (0.5-7 d)	Types 1 and 2 commonly cause URIs, or croup in kids (barking cough or hoarseness); Type 3 can lead to bronchiolitis and pneumonia.	1 day before to 5 days after sx onset.	Droplet, Contact	Humans	Isolation on culture ************************************	

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Revision History

April 2018 – Pathogen descriptions, lab confirmation instructions, OSPHL address and formatting updated; added MERS and removed Q Fever (Allain, Ariail, Boyd, Crawford, DeBess, Humphrey, Leman, Liko, Poissant, Scott, Tran)

April 2012 – Contact information for OSPHL and availability of testing at OSPHL updated.

October 2010 – Original posted

Oregon State Public Health Lab (OSPHL): 7202 NE Evergreen Parkway, Suite 100, Hillsboro, OR 97214/ 503.693.4100 Acute and Communicable Disease Prevention (ACDP): 800 NE Oregon St, Suite 772, Portland, OR 97232/ 971.673.1111

Appendix: Oregon Public Health Division Links

Public Health Home:	healthoregon.org				
CDC Morbidity and Mortality Weekly Report:	<u>cdc.gov/mmwr</u>				
Communicable Disease Home:	healthoregon.org/acd				
CD Reporting Posters:	healthoregon.org/diseasereporting				
CD Summary Newsletter:	healthoregon.org/cdsummary				
Investigative Guidelines, Case Report Forms:	healthoregon.org/iguides				
Immunization Standing Orders:	https://public.health.oregon.gov/Prevention Wellness/VaccinesImmunization/Immunizatio nProviderResources/Pages/stdgordr.aspx				
Local Health Department Technical Assistance:	healthoregon.org/lhd				
Local Health Department Directory:	healthoregon.org/lhddirectory				
OSPHL Home:	healthoregon.org/phl				
OSPHL - Lab Test Menu:	healthoregon.org/labtests				
OSPHL - Order Forms & Kits:	http://public.health.oregon.gov/LaboratorySe rvices/CommunicableDiseaseTesting/forms/P ages/index.aspx				
OSPHL - Submitting Enteric Outbreak Specimens	https://public.health.oregon.gov/LaboratoryS ervices/Pages/SubmittingEntericOutbreakSpe cimens.aspx				
OSPHL - Packaging and Shipping of Specimens	http://public.health.oregon.gov/LaboratorySe rvices/SubmittingSamples/Pages/ShippingTra nsport.aspx				
Rabies and Animal Bites:	https://public.health.oregon.gov/DiseasesCon ditions/DiseasesAZ/rabies				
Animal to human algorithm:	http://public.health.oregon.gov/DiseasesCond itions/DiseasesAZ/rabies/Documents/alg- animal-people.pdf				
Animal to animal algorithm:	http://public.health.oregon.gov/DiseasesCond itions/DiseasesAZ/rabies/Documents/alg- animal-animal.pdf				

Α

AGENT: A factor, such as a microorganism, chemical substance, or form of radiation, whose presence, excessive presence, or (in deficiency diseases) relative absence is essential for the occurrence of a disease.

ANAEROBIC: An organism that grows best in the absence of oxygen. An obligate anaerobe can only grow in the absence of oxygen.

ANALYTIC EPIDEMIOLOGY: The aspect of epidemiology concerned with the search for health-related causes and effects. Uses comparison groups, which provide baseline data, to quantify the association between exposures and outcomes, and test hypotheses about causal relationships.

ANALYTIC STUDY: A comparative study intended to identify and quantify associations, test hypotheses, and identify causes. Two common types are cohort study and case-control study.

ANTITOXIN: A medication that contains antibodies against a specific toxin and neutralizes the effects of the toxin. Administration of an antitoxin does not always lead to full recovery of the patient because antitoxin (such as botulinum antitoxin) may only bind to circulating toxin and not toxin already bound to the tissue.

ASSOCIATION: Statistical relationship between two or more events, characteristics, or other variables.

ATTACK RATE: A variant of an incident rate, applied to a narrowly defined population observed for a limited period of time, such as during an epidemic.

В

BAR CHART: A visual display of the size of the different categories of a variable. Each category or value of the variable is represented by a bar.

BIAS: Deviation of results or inferences from the truth, or processes leading to such systematic deviation. Any trend in the collection, analysis, interpretation, publication, or review of data that can lead to conclusions that are systematically different from the truth.

BIOLOGICAL TRANSMISSION: The indirect vector-borne transmission of an infectious agent in which the agent undergoes biologic changes within the vector before being transmitted to a new host.

BOILING: Boiling occurs at 100 C (or 212 F).

С

CARRIER: A person or animal without apparent disease who harbors a specific infectious agent and is capable of transmitting the agent to others. The carrier state may occur in an

Glossary

individual with an infection that is inapparent throughout its course (known as asymptomatic carrier), or during the incubation period, convalescence, and postconvalescence of an individual with a clinically recognizable disease. The carrier state may be of short or long duration (transient carrier or chronic carrier).

CASE: In epidemiology, a countable instance in the population or study group of a particular disease, health disorder, or condition under investigation. Sometimes, an individual with a particular disease.

CASE-CONTROL STUDY: A type of observational analytic study. Enrollment into the study is based on presence ("case") or absence ("control") of disease. Characteristics such as previous exposure are then compared between cases and controls.

CASE DEFINITION: A set of standard criteria for deciding whether a person has a particular disease or health-related condition, by specifying clinical criteria and limitations on time, place, and person.

CASE-FATALITY RATIO: The proportion of persons with a particular condition (cases) who die from that condition. The denominator is the number of incident cases; the numerator is the number of cause-specific deaths among those cases.

CAUSE OF DISEASE: A factor (characteristic, behavior, event, etc.) that directly influences the occurrence of disease. A reduction of the factor in the population should lead to a reduction in the occurrence of disease.

CHI-SQUARE TEST: A test of statistical significance that is used to determine how likely it is that an observed association between an exposure and a disease could have occurred due to chance alone, if the exposure was not actually related to the disease. The Chi-Square test is the test of choice when the expected values for each cell in a two-by-two table are at least 5.

CLINICAL CHARACTERISTICS: Information about the illness including signs and symptoms, time of onset, and results of tests and examinations.

CLUSTER: An aggregation of cases of a disease or other health-related condition, particularly cancer and birth defects, which are closely grouped in time and place. The number of cases may or may not exceed the expected number; frequently the expected number is not known. Cohort: A well-defined group of people who have had a common experience or exposure, who are then followed up for the incidence of new diseases or events, as in a cohort or prospective study. A group of people born during a particular period or year is called a birth cohort.

COHORT STUDY: A type of observational analytic study. Enrollment into the study is based on exposure characteristics or membership in a group. Disease, death or other health-related outcomes are then ascertained and compared.

COMMON SOURCE OUTBREAK: An outbreak that results from a group of persons being exposed to a common noxious influence, such as an infectious agent or toxin. If the group is exposed over a relatively brief period of time, so that all cases occur within one incubation period, then the common source outbreak is further classified as a point source outbreak. In some common source outbreaks, persons may be exposed over a period of days, weeks, or longer, with the exposure being either intermittent or continuous. CONTACT: Exposure to a source of an infection, or a person so exposed. Continuous common source outbreak: An outbreak in which persons are exposed to the same source but exposure is prolonged over a period of days, weeks, or longer.

CONTROL: In a case-control study, comparison group of person without disease.

CRANIAL NERVE DYSFUNCTION: Abnormal function of one or more of the 12 nerve pairs that originate from the base of the brain and brain stem (as opposed to the spinal cord). The cranial nerves innervate primarily the head and neck region (including the eyes, ears, mouth, tongue, pharynx, and larynx).

CRITICAL CONTROL POINT: Steps in the preparation of a food item where action can be taken to prevent or eliminate a food safety problem or reduce it to an acceptable level. Control of the problem at the critical control point is necessary because it will not be addressed in subsequent steps in the preparation of the food.

D

DEMOGRAPHIC INFORMATION: The "person" characteristics – age, sex, race, and occupation – of descriptive epidemiology used to characterize the population at risk.

DENOMINATOR: The lower portion of a fraction used to calculate a rate or ratio. In a rate, the denominator is usually the population (or population experience, as in person-years, etc.) at risk.

DESCRIPTIVE EPIDEMIOLOGY: The aspect of epidemiology concerned with the organizing and summarizing health-related data according to time, place, and person.

DIPLOPIA: Double vision. A common symptom of botulism.

DIRECT TRANSMISSION: The immediate transfer of an agent from a reservoir to a susceptible host by direct contact or droplet spread.

DISTRIBUTION: In epidemiology, the frequency and pattern of health-related characteristics and events in a population. In statistics, the observed or theoretical frequency of values of a variable.

DROPLET NUCLELI: The residue of dried droplets that may remain suspended in the air for long periods, may be blown over great distances, and are easily inhaled into the lungs and exhaled.

DROPLET SPREAD: The direct transmission of an infectious agent from a reservoir to a susceptible host by spray with relatively large, short-ranged aerosols produced by sneezing, coughing, or talking.

DYSARTHRIA: Difficulties in speech. A common symptom of botulism.

DYSPHAGIA: Difficulties swallowing. A common symptom of botulism.

Ε

ENDEMIC DISEASE: The constant presence of a disease or infectious agent within a given geographic area or population group; may also refer to the usual prevalence of a given disease within such area or group.

ENVIRONMENTAL FACTOR: An extrinsic factor, (geology, climate, insects, sanitation, health services, etc.), which affects the agent and the opportunity for exposure.

ENVIRONMENTAL HEALTH ASSESSMENT: A focused investigation of a food or meal implicated in a foodborne disease outbreak. The assessment follows the implicated item from its raw ingredients to consumption by the consumer and considers how the causative agent, the host, and environmental conditions interacted to result in a foodborne disease.

EPI INFO: Epi Info is a series of program for use by public health professionals in conduction outbreak investigations, managing databases for public health surveillance, and general database and statistics applications. Epi Info can be used to develop a questionnaire, customize the data entry process, and enter and analyze data.

EPIDEMIC: The occurrence of more cases of disease than expected in a given area or among a specific group of people over a particular period of time.

EPIDEMIC CURVE: A histogram that shows the course of a disease outbreak or epidemic by plotting the number of cases by time of onset.

EPIDEMIOLOGY: The study of the distribution and determinants of health-related states or events in specified populations, and the application of this study to the control of health problems.

EPIDEMIOLOGIC TRIAD: The traditional model of infectious disease causation. Includes three components: an external agent, a susceptible host, an environment that brings the host and agent together, so that disease occurs.

EVALUATION: A process that attempts to determine as systematically and objectively as possible the relevance, effectiveness, and impact of activities in the light of their objectives.

EXPOSED (GROUP): A group whose members have been exposed to a supposed cause of disease or health state of interest, or posses a characteristic that is a determinant of the health outcome of interest.

F

FISHER EXACT TEST: A test of statistical significance that is used to determine how likely it is that an observed association between an exposure and a disease could have occurred due to

Glossary

chance alone, if the exposure was not actually related to the disease. The Fisher Exact Test is the test of choice when the expected values in a two-by-two table are less than 5.

FLOW DIAGRAM: A diagram of the steps used in the preparation of a food item. Each operation is represented by a rectangle and arrows indicate the flow of the process.

G

GRAM-POSITIVE: One of two large groups of bacteria based on their reaction to the Gram stain. Gram-positive bacteria appear purple as the result of staining; gram-negative bacteria appear pink. Gram staining is important in taxonomy and reflects differences in cell wall structure.

GRAPH: A way to sow quantitative data visually, using a system of coordinates.

Η

HEAT-LIABLE: Can be destroyed by heating.

HEAT-STABLE: Is not destroyed by heating.

HIGH-RISK GROUP: A group in the community with an elevated risk of disease.

HISTOGRAM: A graphic representation of the frequency distribution of a continuous variable. Rectangles are drawn in such a way that their bases lie on a linear scale representing different intervals, and their heights are proportional to the frequencies of the values within each of the intervals.

HOST: A person or other living organism that can be infected by an infectious agent under natural conditions.

HOST FACTOR: An intrinsic factor (age, race, sex, behaviors, etc.) which influences an individual's exposure, susceptibility, or response to a causative agent.

HYPOTHESIS: A supposition, arrived at from observation or reflection, that leads to refutable predictions. Any conjecture cast in a form that will allow it to be tested and refuted.

IMMUNITY, ACTIVE: Resistance developed in response to stimulus by an antigen (infecting agent or vaccine) and usually characterized by the presence of antibody produced by the host.

IMMUNITY, HERD: The resistance of a group to invasion and spread of an infectious agent, based on the resistance to infection of a high proportion of individual members of the group.

The resistance is a product of the number of susceptible and the probablility that those who are susceptible will come into contact with an infected person.

IMMUNITY, PASSIVE: Immunity conferred by an antibody produced in another host and acquired naturally by an infant from its mother or artificially by administration of an antibody-containing preparation (antiserum or immune globulin).

IMPACT MEASURES: One of the types of measures used to monitor the effectiveness of a program or intervention. Impact measures help describe the immediate results of a program such as changes in patient or physician knowledge or behavior (as opposed to changes in health status of the target population).

INCIDENCE RATE: A measure of the frequency with which an event, such as a new case of illness, occurs in a population over a period of time. The denominator is the population at risk; the numerator is the number of new cases occurring during a given period of time.

INCUBATION PERIOD: Time period between exposure to an infectious agent or toxin and the first appearance of symptoms of the infection or intoxication.

INDIRECT TRANSMISSION: The transmission of an agent carried from a reservoir to a susceptible host by suspended air particles or by animate (vector) or inanimate (vehicle) intermediaries.

L

LATENCY PERIOD: A period of subclinical or inapparent pathologic changes following exposure, ending with the onset of symptoms of chronic disease.

LINE LIST: A list of selected information about each case in an outbreak. Each column represents an important variable (e.g., patient identifier, age, sex) while each row represents a different case.

Μ

MATAMBRE: A traditional Argentinian dish prepared from meat, vegetables, spices and eggs.

MATE: Green tea.

MEAN, ARITHMETIC: The measure of central location commonly called the average. It is calculated by adding together all the individual values in a group of measurements and dividing by the number of values in the group.

MEASURE OF ASSOCIATION: A quantified relationship between exposure and disease; includes relative risk, rate ratio, odds ratio.

MEDIAN: The measure of central location which divides a set of data into two equal parts.

Glossary

MULTIPLE COMPARISONS: When a large number of associations are evaluated for a particular disease or outcome. Multiple comparisons are of concern because as the number of comparisons increases, the probability of finding a "statistically significant association" grows due to chance alone.

Ν

NUMERATOR: The upper portion of a fraction.

0

OBLIGATE ANAEROBE: An organism that can only grow in the absence of oxygen.

ODDS RATIO: A measure of association which quantifies the relationship between an exposure and health outcome from a comparative study; also known as the cross-product ratio. OUTBREAK: Synonymous with epidemic. Sometimes the preferred word, as it may escape sensationalism associated with the word epidemic. Alternatively, a localized as opposed to generalized epidemic.

OUTCOME MEASURES: One of the types of measures used to monitor the effectiveness of a program or intervention. Outcome measures examine changes in the health status of the target population as the program is implemented such as mortality, morbidity, disability, or the quality of life.

Ρ

PANDEMIC: An epidemic occurring over a very wide area (several countries or continents) and usually affecting a large proportion of the population.

PERCENTAGE The number of patients which a characteristic divided by the total number of patients with the characteristic.

pH: A measure of the acidity or alkalinity of a substance. A pH of 7 is considered neutral, a pH of less than 7 is acidic, and a pH of grater than 7 is alkaline.

POINT SOURCE OUTBREAK: An outbreak in which persons are exposed to the same source over a relatively brief period.

PREVALENCE: The number or proportion of cases or events or conditions in a given population.

PRION – A small proteinaceous particle that is believed to be responsible for the class of central nervous system diseases known as spongiform encephalopathies in animals and humans.

PROCESS MEASURES: One of the types of measures used to monitor the effectiveness of a program or intervention. Process measures help determine if a program has been implemented

as planned. Process measures tend to count services and activities that result from the program.

PROPAGATED OUTBREAK: An outbreak that does not have a common source, but instead spread from person to person.

PROPORTION: A type of ratio in which the numerator is included in the denominator. The ratio of a part to the whole, expressed as a "decimal fraction" (e.g., 0.2), as a fraction (1/5), or, loosely, as a percentage (20%).

PROTECTIVE FACTOR: An aspect of personal behavior or lifestyle, an environmental exposure, or an inborn or inherited characteristic that is associated with a decreased occurrence of disease or other health-related event or condition.

PTOSIS: Drooping eyelids. A common symptom of botulism.

PULSED FIELD GEL ELECTROPHORESIS: A method of subtyping microorganisms based on their DNA composition. In pulsed field gel electrophoresis, the bacterial DNA is cut into pieces. The pieces are separated by placing them in a jelly-like substance (i.e., the gel) to which a pulsing electric field is applied. The electric field separates the DNA pieces across the gel based on size. The resulting DNA bands are made to fluoresce and are read under ultraviolet illumination.

p-VALUE: The probability that an observed association between an exposure and a disease could have occurred due to chance alone, if the exposure was not actually related to the disease.

R

RATE: an expression of the frequency with which an event occurs in a defined population.

RATIO: The value obtained by dividing one quantity by another.

RELATIVE RISK: A comparison of the risk of some health-related event such as disease or death in two groups.

RETROSPECTIVE COHORT STUDY: A cohort study in which data collection occurs after exposure has occurred. Unlike other cohort studies, retrospective cohort studies rely on historical exposure information.

RISK: The probability that an event will occur, e.g. that an individual will become ill or die within a stated period of time or age.

RISK FACTOR: An aspect of personal behavior or lifestyle, an environmental exposure, or an inborn or inherited characteristic that is associated with an increased occurrence of disease or other health-related event or condition.

RISK RATIO: A comparison of the risk of some health-related event such as disease or death in tow groups.

S

SEQUELA: (plural is sequelae) A disability or complication following as a consequence of a disease.

SPORADIC: A disease that occurs infrequently and irregularly.

SPORE: Reproductive cells produced by some bacteria. Spores are highly resistant to heat and other conditions and, therefore, allow the organism to survive during poor growth conditions.

Т

TABLE: A set of data arranged in rows and columns.

TABLE SHELL: A table that is complete except for the data.

TREND: A long-term movement or change in frequency, usually upwards or downwards.

V

VARIABLE: Any characteristic or attribute that can be measures.

VITAL STATISTICS: Systematically tabulated information about births, marriages, divorces, and deaths, based on registration of these vital events.

W

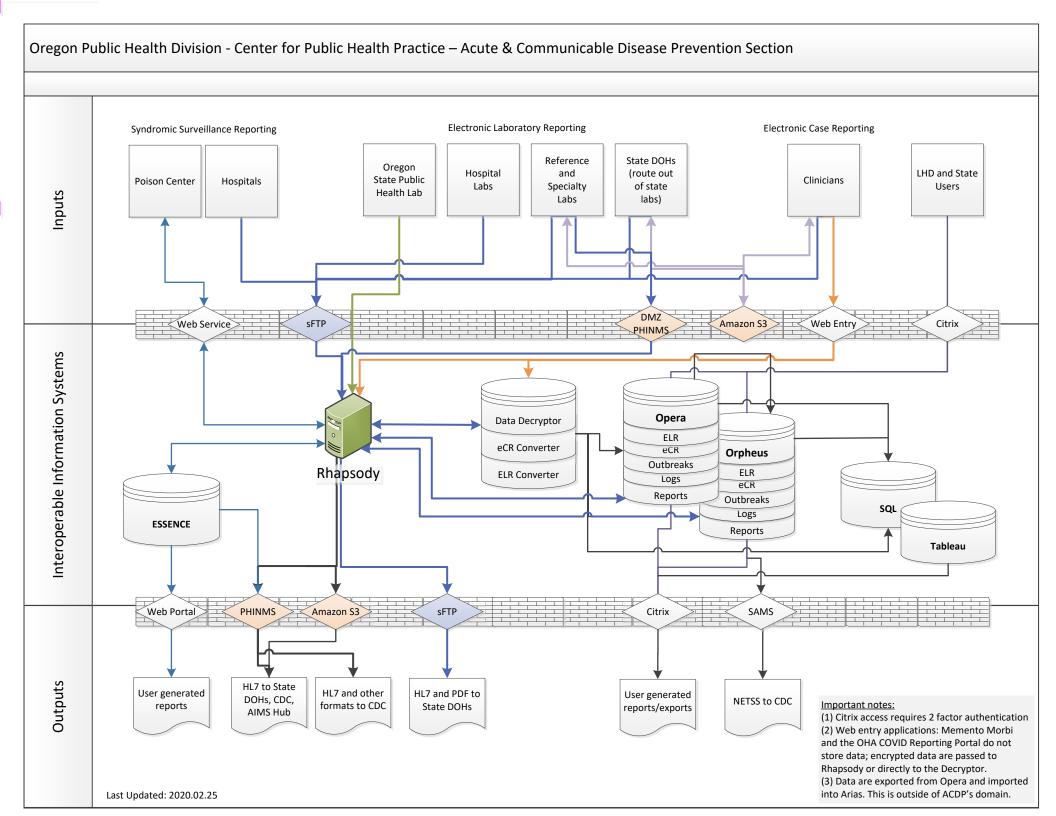
WATER CONTENT: The amount of moisture in a substance that is readily available for a microorganism to grow. Water content can be limited by dehydration but is usually controlled by the addition of NaCl (i.e. table salt).

Χ

X-AXIS: The horizontal axis in a rectangular coordinate graph.

Υ

Y-AXIS: The vertical axis in a rectangular coordinate graph.



Health Authority

Revised: June 21, 2021

Acute and Communicable Disease Prevention) ACDP							
		Cell #	Name		Cell #		
,		971-346-0720	Ladd-Wilson, Stephen 3-013		971-346-0892		
		971-757-4549	Laing, Rob		971-258-5113		
B ahrami, Sheida		503-341-6384	LaLonde, Laura	3-1120	971-286-0624		
Bancroft, June	3-1045	503-309-6822	Leahy, Jude	3-1130	503-348-8663		
Barber, Michelle	3-1030	971-271-2864	Leman, Richard	3-1089	503-572-5090		
Beauchamp, Brad	3-0288	971-271-2840	LeVasseur, Maddie	3-2383	971-346-0834		
B eldavs, Zints	3-0166	503-849-7802	Linder, Meghan	3-2384	971-645-9229		
B onner, Kim		503-484-0157	M ansour, Adel		971-378-7968		
B ruhn, Pam		971-337-4876	M artin, Tasha	3-1116	971-313-1594		
B yster, Leslie	3-1065	971-284-0923	M bund-Hubbard, Faraja		971-757-4551		
C arlson, Merry		971-313-4097	McArdle, Breanna		971-757-4548		
C ieslak, Paul	3-1082	503-741-6691	McNeese, Cody		971-420-5154		
C oke, Erin		971-757-4547	M illet, Meghan		971-930-5725		
C rawford, Heather		971-757-4840	Norris, Molly		971-501-0953		
D eBess, Emilio	3-1027	503-830-3131	Nugent, Michael		971-266-7897		
Eades, Anne		971-284-2360	O campo, Valerie	3-2793	971-273-3349		
Ehlers, Sara 3-3439		971-271-0016	O wusu-Dommey, Ama	3-1075	971-346-0857		
Escutia, Gabriela 3-0968		971-269-9399	Phan, Angela	3-1049	971-219-5123		
Falender, Becca		971-378-7979	Pierce, Rebecca	3-3498	971-666-9670		
Fitch, Tom 3-1005		971-286-0104	Powell, Melissa 3-113		503-849-0871		
Hacker, Sara		503-347-7318	Ryan-Mapolski, Mitch		503-490-3825		
Harger, Renee 3-1107		971-219-5384	Samper, Monika 3-0141		971-388-9811		
Hawkins, Sam		971-378-7978	Schindler, Monty		971-347-8476		
· · · ·		971-287-0160	Sutton, Melissa 3-2504		503-871-9772		
Herrera, Elizabeth (Zoe)		503-347-6689	Tammer, Roza 3-1074		971-346-0632		
Hertzel, Heather		971-346-0724	Thomas, Ann	3-1003	503-260-9672		
Hicks, Mike		971-380-1749	T ran, Dat	3-1095	971-409-8616		
Iguchi, Lisa	3-3437	971-346-0783	T revejo, Rosalie	3-1007	971-401-1048		
J amieson, Heather	3-1109	503-758-8295	West, Nicole	3-1009	971-346-0674		
J ohnson, Elizabeth		971-865-1779	Williams, Brittany		971-757-4550		
Joshi, Tejas		971-284-5522	Willett, Sierra		971-378-7977		
K idane, Helen		503-341-8052	Youngers, Emily		971-201-0702		

I