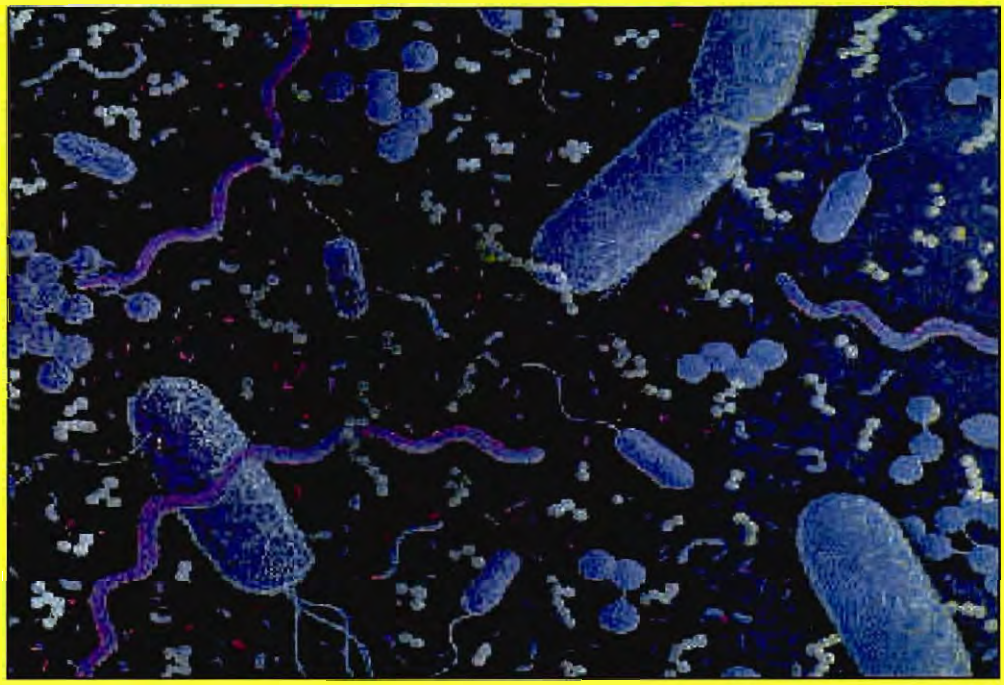
BAD BUG BOOK:

Foodborne Pathogenic Microorganisms & Natural Toxins Handbook



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Bad Bug Book

U.S. Food & Drug Administration
Center for Food Safety & Applied Nutrition
Foodborne Pathogenic Microorganisms and Natural Toxins Handbook

The *"Bad Bug Book"*

This handbook provides basic facts regarding foodborne pathogenic microorganisms and natural toxins. It brings together in one place information from the Food & Drug Administration, the Centers for Disease Control & Prevention, the USDA Food Safety Inspection Service, and the National Institutes of Health.

Some technical terms have been linked to the National Library of Medicine's Entrez glossary. Recent articles from Morbidity and Mortality Weekly Reports have been added to selected chapters to update the handbook with information on later outbreaks or incidents of foodborne disease. At the end of selected chapters on pathogenic microorganisms, hypertext links are included to relevant Entrez abstracts and GenBank genetic loci. A more complete description of the handbook may be found in the Preface.

PATHOGENIC BACTERIA

- *Salmonella* spp.
- *Clostridium botulinum*
- *Staphylococcus aureus*
- *Campylobacter jejuni*
- *Yersinia enterocolitica* and *Yersinia pseudotuberculosis*
- *Listeria monocytogenes*
- *Vibrio cholerae* O1
- *Vibrio cholerae* non-O1
- *Vibrio parahaemolyticus* and other *vibrios*
- *Vibrio vulnificus*
- *Clostridium perfringens*
- *Bacillus cereus*
- *Aeromonas hydrophila* and other spp.
- *Plesiomonas shigelloides*
- *Shigella* spp.
- Miscellaneous enterics
- *Streptococcus*

Enterovirulent Escherichia coli Group (EEC Group)

- *Escherichia coli* - enterotoxigenic (ETEC)
- *Escherichia coli* - enteropathogenic (EPEC)
- *Escherichia coli* O157:H7 enterohemorrhagic (EHEC)
- *Escherichia coli* - enteroinvasive (EIEC)

PARASITIC PROTOZOA and WORMS

- *Giardia lamblia*
- *Entamoeba histolytica*
- *Cryptosporidium parvum*
- *Cyclospora cayetanensis* Updated!
- *Anisakis* sp. and related worms
- *Diphyllobothrium* spp.
- *Nanophyetus* spp.
- *Eustrongylides* sp.
- *Acanthamoeba* and other free-living amoebae
- *Ascaris lumbricoides* and *Trichuris trichiura*

VIRUSES

- Hepatitis A virus
- Hepatitis E virus
- Rotavirus
- Norwalk virus group
- Other viral agents

NATURAL TOXINS

- Ciguatera poisoning
- Shellfish toxins (PSP, DSP, NSP, ASP)
- Scombroid poisoning
- Tetrodotoxin (Pufferfish)
- Mushroom toxins
- Aflatoxins
- Pyrrolizidine alkaloids
- Phytohaemagglutinin (Red kidney bean poisoning)
- Grayanotoxin (Honey intoxication)

APPENDICES

- Infective dose
- Epidemiology summary table
- Factors affecting microbial growth in foods

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Hypertext last updated by mow/dms/xxz 1998-SEP-29

The *"Bad Bug Book"* Preface

The Center for Food Safety and Applied Nutrition (CFSAN) has prepared a handbook on foodborne pathogenic microorganisms (bacteria, viruses and parasites) and natural toxins. Each chapter focuses mainly on either one foodborne pathogenic microorganism or natural toxin. In some chapters, a closely related group of organisms or natural toxins is covered. Each chapter provides information that is general in nature and abbreviated for convenience. This book is not intended to serve as a comprehensive reference source. The intent in each chapter is to provide basic facts regarding these organisms and toxins, including their characteristics, habitat or source, associated foods, infective dose, characteristic disease symptoms, complications, recent and/or major outbreaks, and any susceptible populations. The chapters contain minimal information on the analytical methods used to detect, isolate, and/or identify the pathogens or natural toxins. For these methods, the reader should refer to the FDA's Bacteriological Analytical Manual (BAM) and the AOAC's Official Methods of Analysis (15th edition).

January 1992

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Salmonella spp.

Education | CDC/MMWR | NIH/PubMed

1. Name of the Organism:
Salmonella spp.

Salmonella is a rod-shaped, motile bacterium -- nonmotile exceptions *S. gallinarum* and *S. pullorum*--, nonsporeformin and Gram-negative. There is a widespread occurrence in animals, especially in poultry and swine. Environmental sources of the organism include water, soil, insects, factory surfaces, kitchen surfaces, animal feces, raw meats, raw poultry, and raw seafoods, to name only a few.

2. Nature of Acute Disease:

S. typhi and the paratyphoid bacteria are normally caused septicemic and produce typhoid or typhoid-like fever in humans. Other forms of salmonellosis generally produce mil symptoms.

3. Nature of Disease:

Acute symptoms -- Nausea, vomiting, abdominal cramps,mi diarrhea, fever, and headache. Chronic consequences -- arthr symptoms may follow 3-4 weeks after onset of acute symptoms.

Onset time -- 6-48 hours.

Infective dose -- As few as 15-20 cells; depends upon age an health of host, and strain differences among the members of genus.

Duration of symptoms -- Acute symptoms may last for 1 to 2 days or may be prolonged, again depending on host factors, ingested dose, and strain characteristics.

Cause of disease -- Penetration and passage of Salmonella organisms from gut lumen into epithelium of small intestine where inflammation occurs; there is evidence that an enterotoxin may be produced, perhaps within the enterocyte.

4. Diagnosis of Human Illness:

Serological identification of culture isolated from stool.

5. Associated Foods:

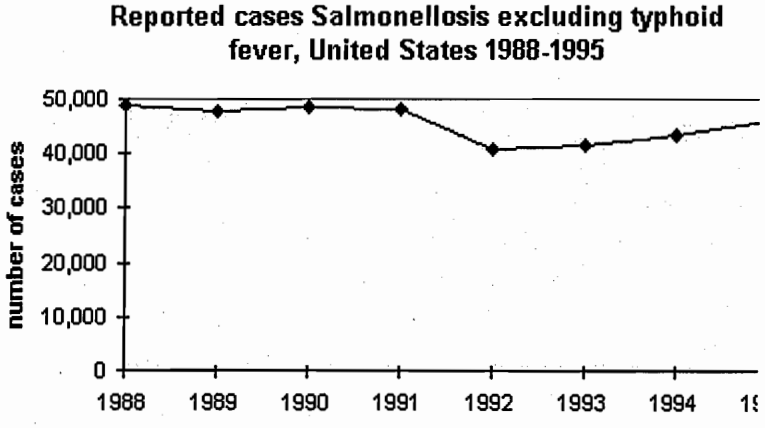
Raw meats, poultry, eggs, milk and dairy products, fish, shrimp, frog legs, yeast, coconut, sauces and salad dressing, cake mixes, cream-filled desserts and toppings, dried gelatin, peanut butter, cocoa, and chocolate.

Various Salmonella species have long been isolated from the outside of egg shells. The present situation with *S. enteritidis* complicated by the presence of the organism inside the egg, : the yolk. This and other information strongly suggest vertica transmission, i.e., deposition of the organism in the yolk by a infected layer hen prior to shell deposition. Foods other than eggs have also caused outbreaks of *S. enteritidis* disease.

6. Relative Frequency of Disease:

It is estimated that from 2 to 4 million cases of salmonellosis occur in the U.S. annually.

The incidence of salmonellosis appears to be rising both in tl U.S. and in other industrialized nations. *S. enteritidis* isolatio from humans have shown a dramatic rise in the past decade, particularly in the northeast United States (6-fold or more), a the increase in human infections is spreading south and west, with sporadic outbreaks in other regions.



Summary of Notifiable Diseases, United States MMWR 44(53): 1996 October

7. Complications:

S. typhi and *S. paratyphi* A, B, and C produce typhoid and typhoid-like fever in humans. Various organs may be infecte leading to lesions. The fatality rate of typhoid fever is 10% compared to less than 1% for most forms of salmonellosis. *S dublin* has a 15% mortality rate when septicemic in the elder and *S. enteritidis* is demonstrating approximately a 3.6% mortality rate in hospital/nursing home outbreaks, with the elderly being particularly affected.

Salmonella septicemia has been associated with subsequent infection of virtually every organ system.

Postenteritis reactive arthritis and Reiter's syndrome have als been reported to occur generally after 3 weeks. Reactive arthritis may occur with a frequency of about 2% of culture-

proven cases. Septic arthritis, subsequent or coincident with septicemia, also occurs and can be difficult to treat.

8. Target Populations:

All age groups are susceptible, but symptoms are most sever in the elderly, infants, and the infirm. AIDS patients suffer salmonellosis frequently (estimated 20-fold more than gener population) and suffer from recurrent episodes.

9. Foods Analysis:

Methods have been developed for many foods having prior history of Salmonella contamination. Although conventional culture methods require 5 days for presumptive results, sever rapid methods are available which require only 2 days.

10. Selected Outbreaks:

In 1985, a salmonellosis outbreak involving 16,000 confirme cases in 6 states was caused by low fat and whole milk from one Chicago dairy. This was the largest outbreak of foodborn salmonellosis in the U.S. FDA inspectors discovered that the pasteurization equipment had been modified to facilitate the running off of raw milk, resulting in the pasteurized milk bei contaminated with raw milk under certain conditions. The da has subsequently disconnected the cross-linking line. Person on antibiotic therapy were more apt to be affected in this outbreak.

In August and September, 1985, *S. enteritidis* was isolated fr employees and patrons of three restaurants of a chain in Maryland. The outbreak in one restaurant had at least 71 illnesses resulting in 17 hospitalizations. Scrambled eggs fro a breakfast bar were epidemiologically implicated in this outbreak and in possibly one other of the three restaurants. T plasmid profiles of isolates from patients all three restaurants matched.

The Centers for Disease Control (CDC) has recorded more th 120 outbreaks of *S. enteritidis* to date, many occurring in restaurants, and some in nursing homes, hospitals and prison

In 1984, 186 cases of salmonellosis (*S. enteritidis*) were reported on 29 flights to the United States on a single international airline. An estimated 2,747 passengers were affected overall. No specific food item was implicated, but fo ordered from the first class menu was strongly associated wi disease.

S. enteritidis outbreaks continue to occur in the U.S. (Table 1 The CDC estimates that 75% of those outbreaks are associat with the consumption of raw or inadequately cooked Grade A whole shell eggs. The U.S. Department of Agriculture published Regulations on February 16, 1990, in the Federal Register establishing a mandatory testing program for egg-producing breeder flocks and commercial flocks implicated i causing human illnesses. This testing should lead to a reducti in cases of gastroenteritis caused by the consumption of Gra A whole shell eggs.

Salmonellosis associated with a Thanksgiving Dinner in Nevada in 1995 is reported in MMWR 45(46):1996 Nov 22.

MMWR 45(34):1996 Aug 30 reports on several outbreaks of *Salmonella enteritidis* infection associated with the consumption of raw shell eggs in the United States from 199 to 1995.

A report of an outbreak of *Salmonella* Serotype Typhimuriur infection associated with the consumption of raw ground bee may be found in MMWR 44(49):1995 Dec 15.

MMWR 44(42):1995 Oct 27 reports on an outbreak of Salmonellosis associated with beef jerky in New Mexico in 1995.

The report on the outbreak of Salmonella from commercially prepared ice cream is found in MMWR 43(40):1994 Oct 14.

An outbreak of *S. enteritidis* in homemade ice cream is repor in this MMWR 43(36):1994 Sep 16.

A series of *S. enteritidis* outbreaks in California are summarized in the following MMWR 42(41):1993 Oct 22.

For information on an outbreak of Salmonella Serotype Tennessee in Powdered Milk Products and Infant Formula -- see this MMWR 42(26):1993 Jul 09.

Summaries of Salmonella outbreaks associated with Grade A eggs are reported in MMWR 37(32):1988 Aug 19 and MMW 39(50):1990 Dec 21.

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

11. Education:

The CDC provides an informational brochure on preventing Salmonella enteritidis infection.

Food Safety Facts for Consumers (July 1999)

12. Other Resources:

A Loci index for genome *Salmonella enteritidis* is available from GenBank.

CDC/MMWR

The CDC/MMWR link will provide a list of Morbidity and Mortality Weekly Reports at CDC relating to this organism or toxin. The date shown is the date the item was posted on

the Web, not the date of the MMWR. The summary statement shown are the initial words of the overall document. The specific article of interest may be just one article or item within the overall report.

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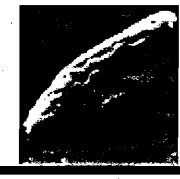


Clostridium botulinum

Education | CDC/MMWR | NIH/PubMed

1. Name of the organism:
Clostridium botulinum

Clostridium botulinum is an anaerobic, Gram-positive, spore-fo rod that produces a potent neurotoxin. The spores are heat-resis and can survive in foods that are incorrectly or minimally proce Seven types (A, B, C, D, E, F and G) of botulism are recognize based on the antigenic specificity of the toxin produced by each strain. Types A, B, E and F cause human botulism. Types C anc cause most cases of botulism in animals. Animals most commo affected are wild fowl and poultry, cattle, horses and some spec fish. Although type G has been isolated from soil in Argentina, outbreaks involving it have been recognized.

Foodborne botulism (as distinct from wound botulism and infar botulism) is a severe type of food poisoning caused by the inge of foods containing the potent neurotoxin formed during growt the organism. The toxin is heat labile and can be destroyed if he at 80oC for 10 minutes or longer. The incidence of the disease i but the disease is of considerable concern because of its high mortality rate if not treated immediately and properly. Most of t to 30 outbreaks that are reported annually in the United States a associated with inadequately processed, home-canned foods, bu occasionally commercially produced foods have been involved outbreaks. Sausages, meat products, canned vegetables and sea products have been the most frequent vehicles for human botuli

The organism and its spores are widely distributed in nature. Th occur in both cultivated and forest soils, bottom sediments of st lakes, and coastal waters, and in the intestinal tracts of fish and mammals, and in the gills and viscera of crabs and other shellfi

2. Name of the Disease:

Four types of botulism are recognized: foodborne, infant, woun a form of botulism whose classification is as yet undetermined. Certain foods have been reported as sources of spores in cases c infant botulism and the undetermined category; wound botulism not related to foods.

Foodborne botulism is the name of the disease (actually a foodb intoxication) caused by the consumption of foods containing th neurotoxin produced by *C. botulinum*.

Infant botulism, first recognized in 1976, affects infants under 1 months of age. This type of botulism is caused by the ingestion *botulinum* spores which colonize and produce toxin in the intes

tract of infants (intestinal toxemia botulism). Of the various pot environmental sources such as soil, cistern water, dust and food honey is the one dietary reservoir of *C. botulinum* spores thus fa definitively linked to infant botulism by both laboratory and epidemiologic studies. The number of confirmed infant botulisr cases has increased significantly as a result of greater awarenes: health officials since its recognition in 1976. It is now internatic recognized, with cases being reported in more countries.

Wound botulism is the rarest form of botulism. The illness resu when *C. botulinum* by itself or with other microorganisms infec wound and produces toxins which reach other parts of the body the blood stream. Foods are not involved in this type of botulisr

Undetermined category of botulism involves adult cases in whi specific food or wound source cannot be identified. It has been suggested that some cases of botulism assigned to this category result from intestinal colonization in adults, with in vivo produc of toxin. Reports in the medical literature suggest the existence form of botulism similar to infant botulism, but occurring in adı In these cases, the patients had surgical alterations of the gastrointestinal tract and/or antibiotic therapy. It is proposed tha these procedures may have altered the normal gut flora and allo *C. botulinum* to colonize the intestinal tract.

3. Nature of the Disease:

Infective dose -- a very small amount (a few nanograms) of toxi cause illness.

Onset of symptoms in foodborne botulism is usually 18 to 36 h after ingestion of the food containing the toxin, although cases l varied from 4 hours to 8 days. Early signs of intoxication consis marked lassitude, weakness and vertigo, usually followed by do vision and progressive difficulty in speaking and swallowing. Difficulty in breathing, weakness of other muscles, abdominal distention, and constipation may also be common symptoms.

Clinical symptoms of infant botulism consist of constipation tha occurs after a period of normal development. This is followed b poor feeding, lethargy, weakness, pooled oral secretions, and w: altered cry. Loss of head control is striking. Recommended trea is primarily supportive care. Antimicrobial therapy is not recommended. Infant botulism is diagnosed by demonstrating botulinal toxins and the organism in the infants' stools.

4. Diagnosis of Human Illness:

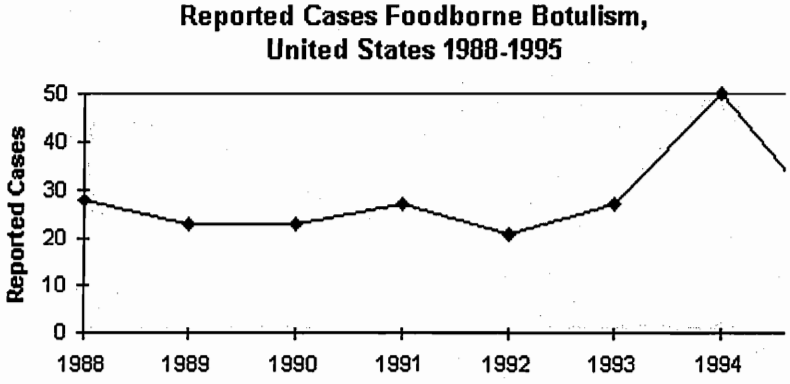
Although botulism can be diagnosed by clinical symptoms alon differentiation from other diseases may be difficult. The most d and effective way to confirm the clinical diagnosis of botulism : laboratory is to demonstrate the presence of toxin in the serum (feces of the patient or in the food which the patient consumed. Currently, the most sensitive and widely used method for detect toxin is the mouse neutralization test. This test takes 48 hours. Culturing of specimens takes 5-7 days.

5. Associated Foods:

The types of foods involved in botulism vary according to food preservation and eating habits in different regions. Any food tha conducive to outgrowth and toxin production, that when proces allows spore survival, and is not subsequently heated before consumption can be associated with botulism. Almost any type food that is not very acidic (pH above 4.6) can support growth a toxin production by *C. botulinum*. Botulinal toxin has been demonstrated in a considerable variety of foods, such as canned peppers, green beans, soups, beets, asparagus, mushrooms, ripe olives, spinach, tuna fish, chicken and chicken livers and liver p and luncheon meats, ham, sausage, stuffed eggplant, lobster, an smoked and salted fish.

6. Frequency:

The incidence of the disease is low, but the mortality rate is hig not treated immediately and properly. There are generally betw to 30 outbreaks a year in the United States. Some cases of botul may go undiagnosed because symptoms are transient or mild, o misdiagnosed as Guillain-Barre syndrome.



Summary of Notifiable Diseases, United States MMWR 44(53): 1996 Octot

7. The Usual Course of Disease and Complications:

Botulinum toxin causes flaccid paralysis by blocking motor ner terminals at the myoneural junction. The flaccid paralysis progr symmetrically downward, usually starting with the eyes and fac the throat, chest and extremities. When the diaphragm and ches muscles become fully involved, respiration is inhibited and deat from asphyxia results. Recommended treatment for foodborne botulism includes early administration of botulinal antitoxin (available from CDC) and intensive supportive care (including mechanical breathing assistance).

8. Target Populations:

All people are believed to be susceptible to the foodborne intoxication.

9. Food Analysis:

Since botulism is foodborne and results from ingestion of thet t of *C. botulinum*, determination of the source of an outbreak is b on detection and identification of toxin in the food involved. Th most widely accepted method is the injection of extracts of the into passively immunized mice (mouse neutralization test). The takes 48 hours. This analysis is followed by culturing all suspec in an enrichment medium for the detection and isolation of the causative organism. This test takes 7 days.

10. Selected Outbreaks:

Two separate outbreaks of botulism have occurred involving commercially canned salmon. Restaurant foods such as sauteed onions, chopped bottled garlic, potato salad made from baked potatoes and baked potatoes themselves have been responsible f number of outbreaks. Also, smoked fish, both hot and cold-smo (e.g., Kapchunka) have caused outbreaks of type E botulism.

In October and November, 1987, 8 cases of type E botulism occ 2 in New York City and 6 in Israel. All 8 patients had consume Kapchunka, an uneviscerated, dry-salted, air-dried, whole white The product was made in New York City and some of it was transported by individuals to Israel. All 8 patients with botulism developed symptoms within 36 hours of consuming the Kapchu One female died, 2 required breathing assistance, 3 were treated therapeutically with antitoxin, and 3 recovered spontaneously. T Kapchunka involved in this outbreak contained high levels of ty botulinal toxin despite salt levels that exceeded those sufficient inhibit *C. botulinum* type E outgrowth. One possible explanatio that the fish contained low salt levels when air-dried at room temperature, became toxic, and then were re-brined. Regulation were published to prohibit the processing, distribution and sale Kapchunka and Kapchunka-type products in the United States.

A bottled chopped garlic-in-oil mix was responsible for three ca botulism in Kingston, N.Y. Two men and a woman were hospit with botulism after consuming a chopped garlic-in-oil mix that been used in a spread for garlic bread. The bottled chopped garl relied solely on refrigeration to ensure safety and did not contai additional antibotulinal additives or barriers. The FDA has orde companies to stop making the product and to withdraw from the market any garlic-in-oil mix which does not include microbial inhibitors or acidifying agents and does not require refrigeratior safety.

Since botulism is a life-threatening disease, FDA always initiate Class I recall.

Janua

An incident of foodborne botulism in Oklahoma is reported in MMWR 44(11):1995 Mar 24.

A botulism type B outbreak in Italy associated with eggplant in reported in MMWR 44(2):1995 Jan 20.

The botulism outbreak associated with salted fish mentioned ab reported in greater detail in MMWR 36(49):1987 Dec 18.

For more information on recent outbreaks see the Morbidit Mortality Weekly Reports from CDC.

11. Education: The December 1995 issue of "FDA Consumer" has an article tit Botulism Toxin: a Poison That Can Heal which discusses Botul toxin with an emphasis on its medical uses.

12. Other Resources: FDA Warns Against Consuming Certain Italian Mascarpone Cr Cheese Because of Potential Serious Botulism Risk *(Sept. 9, 19*

A Loci index for genome *Clostridium botulinum* is available frc GenBank.

CDC/MMWR

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Staphylococcus aureus

CDC/MMWR NIH/PubMed

1. Name of the Organism:
Staphylococcus aureus

S. aureus is a spherical bacterium (coccus) which on microscopic examination appears in pairs, short chains, or bunched, grape-like clusters. These organisms are Gram-positive. Some strains are capable of producing a highly heat-stable protein toxin that causes illness in humans.

2. Name of Acute Disease:

Staphylococcal food poisoning (staphyloenterotoxicosis; staphyloenterotoxemia) is the name of the condition caused by the enterotoxins which some strains of *S. aureus* produce.

3. Nature of the Disease:

The onset of symptoms in staphylococcal food poisoning is usually rapid and in many cases acute, depending on individual susceptibility to the toxin, the amount of contaminated food eaten, the amount of toxin in the food ingested, and the general health of the victim. The most common symptoms are nausea, vomiting, retching, abdominal cramping, and prostration. Some individuals may not always demonstrate all the symptoms associated with the illness. In more severe cases, headache, muscle cramping, and transient changes in blood pressure and pulse rate may occur. Recovery generally takes two days, However, it us not unusual for complete recovery to take three days and sometimes longer in severe cases.

Infective dose--a toxin dose of less than 1.0 microgram in contaminated food will produce symptoms of staphylococcal intoxication. This toxin level is reached when *S. aureus* populations exceed 100000 per gram.

4. Diagnosis of Human Illness:

In the diagnosis of staphylococcal foodborne illness, proper interviews with the victims and gathering and analyzing epidemiologic data are essential. Incriminated foods should be collected and examined for staphylococci. The presence of relatively large numbers of enterotoxigenic staphylococci is good circumstantial evidence that the food contains toxin. The most conclusive test is the linking of an illness with a specific food or in cases where multiple vehicles exist, the detection of the toxin in the food sample(s). In cases where the food may have been treated to kill the staphylococci, as in pasteurization or heating, direct microscopic observation of the food may be an aid in the diagnosis. A number of serological methods for determining the enterotoxigenicity of *S. aureus* isolated from foods as well as methods for the separation and detection of toxins in foods have

been developed and used successfully to aid in the diagnosis of the illness. Phage typing may also be useful when viable staphylococci can be isolated from the incriminated food, from victims, and from suspected carrier such as food handlers.

5. Foods Incriminated: Foods that are frequently incriminated in staphylococcal food poisoning include meat and meat products; poultry and egg products; salads such as egg, tuna, chicken, potato, and macaroni; bakery products such as cream-filled pastries, cream pies, and chocolate eclairs; sandwich fillings; and milk and dairy products. Foods that require considerable handling during preparation and that are kept at slightly elevated temperatures after preparation are frequently involved in staphylococcal food poisoning.

Staphylococci exist in air, dust, sewage, water, milk, and food or on food equipment, environmental surfaces, humans, and animals. Humans and animals are the primary reservoirs. Staphylococci are present in the nasal passages and throats and on the hair and skin of 50 percent or more of healthy individuals. This incidence is even higher for those who associate with or who come in contact with sick individuals and hospital environments. Although food handlers are usually the main source of food contamination in food poisoning outbreaks, equipment and environmental surfaces can also be sources of contamination with *S. aureus*. Human intoxication is caused by ingesting enterotoxins produced in food by some strains of *S. aureus*, usually because the food has not been kept hot enough (60°C, 140°F, or above) or cold enough (7.2°C, 45°F, or below).

6. Frequency of Illness: The true incidence of staphylococcal food poisoning is unknown for a number of reasons, including poor responses from victims during interviews with health officials; misdiagnosis of the illness, which may be symptomatically similar to other types of food poisoning (such as vomiting caused by *Bacillus cereus* toxin); inadequate collection of samples for laboratory analyses; and improper laboratory examination. Of the bacterial pathogens causing foodborne illnesses in the U.S. (127 outbreaks, 7,082 cases recorded in 1983), 14 outbreaks involving 1,257 cases were caused by *S. aureus*. These outbreaks were followed by 11 outbreaks (1,153 cases) in 1984, 14 outbreaks (421 cases) in 1985, 7 outbreaks (250 cases) in 1986 and one reported outbreak (100 cases) in 1987.

7. Complications: Death from staphylococcal food poisoning is very rare, although such cases have occurred among the elderly, infants, and severely debilitated persons.

8. Target Population: All people are believed to be susceptible to this type of bacterial intoxication; however, intensity of symptoms may vary.

9. Analysis of Foods: For detecting trace amounts of staphylococcal enterotoxin in foods incriminated in food poisoning, the toxin must be separated from food constituents and concentrated before identification by specific precipitation with antiserum (antienterotoxin) as follows. Two principles are used for the purpose: (1) the selective adsorption of the enterotoxin from an extract of the food onto ion exchange resins and (2) the use of physical and chemical procedures for the selective removal of food constituents from the extract, leaving the enterotoxin(s) in solution. The use of these techniques and concentration of the resulting products (as much as possible) has made it possible to detect small amounts of enterotoxin in food.

There are developed rapid methods based on monoclonal antibodies (e.g., ELISA, Reverse Passive Latex Agglutination), which are being evaluated for their efficacy in the detection of enterotoxins in food. These rapid methods can detect approximately 1.0 nanogram of toxin/g of food.

10. Typical Outbreak: 1,364 children became ill out of a total of 5,824 who had eaten lunch served at 16 elementary schools in Texas. The lunches were prepared in a central kitchen and transported to the schools by truck. Epidemiological studies revealed that 95% of the children who became ill had eaten a chicken salad. The afternoon of the day preceding the lunch, frozen chickens were boiled for 3 hours. After cooking, the chickens were deboned, cooled to room temperature with a fan, ground into small pieces, placed into l2-inch-deep aluminum pans and stored overnight in a walk-in refrigerator at 42-45°F.

The following morning, the remaining ingredients of the salad were added and the mixture was blended with an electric mixer. The food was placed in thermal containers and transported to the various schools at 9:30 AM to 10:30 AM, where it was kept at room temperature until served between 11:30 AM and noon. Bacteriological examination of the chicken salad revealed the presence of large numbers of *S. aureus*.

Contamination of the chicken probably occurred when it was deboned. The chicken was not cooled rapidly enough because it was stored in l2-inch-deep layers. Growth of the staphylococcus probably occurred also during the period when the food was kept in the warm classrooms. Prevention of this incident would have entailed screening the individuals who deboned the chicken for carriers of the staphylococcus, more rapid cooling of the chicken, and adequate refrigeration of the salad from the time of preparation to its consumption.

11. Atypical Outbreaks:

In 1989, multiple staphylococcal foodborne diseases were associated with the consumption of canned mushrooms. (CDC Morbidity and Mortality Weekly Report, June 23, 1989, Vol. 38, #24.)

Starkville, Mississippi. On February 13, 22 people became ill with gastroenteritis several hours after eating at a university cafeteria. Symptoms included nausea, vomiting, diarrhea, and abdominal cramps. Nine people were hospitalized. Canned mushrooms served with omelets and hamburgers were associated with illness. No deficiencies in food handling were found. Staphylococcal enterotoxin type A was identified in a sample of implicated mushrooms from the omelet bar and in unopened cans from the same lot.

Queens, New York, On February 28, 48 people became ill a median of 3 hours after eating lunch in a hospital employee cafeteria. One person was hospitalized. Canned mushrooms served at the salad bar were epidemiologically implicated. Two unopened cans of mushrooms from the same lot as the implicated can contained staphylococcal enterotoxin A.

McKeesport, Pennsylvania. On April 17, 12 people became ill with gastroenteritis a median of 2 hours after eating lunch or dinner at a restaurant. Two people were hospitalized. Canned mushrooms, consumed on pizza or with a parmigiana sauce, were associated with illness. No deficiencies were found in food preparation or storage. Staphylococcal enterotoxin was found in samples of remaining mushrooms and in unopened cans from the same lot.

Philipsburg, Pennsylvania. On April 22, 20 people developed illness several hours after eating food from a take-out pizzeria. Four people were hospitalized. Only pizza served with canned mushrooms was associated with illness. Staphylococcal enterotoxin was found in a sample of mushrooms from the pizzeria and in unopened cans with the same lot number.

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

12. Other Resources:

A Loci index for genome *Staphylococcus aureus* is available from GenBank.

CDC/MMWR

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U.S. Food & Drug Administration
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Foodborne Pathogenic Microorganisms and Natural Toxins Handbook

Campylobacter jejuni

Education | CDC/MMWR | NIH/PubMed

1. Name of the Organism: *Campylobacter jejuni* (formerly known as *Campylobacter fetus* subsp. jejuni)

Campylobacter jejuni is a Gram-negative slender, curved, and motile rod. It is a microaerophilic organism, which means it has a requirement for reduced levels of oxygen. It is relatively fragile, and sensitive to environmental stresses (e.g., 21% oxygen, drying, heating, disinfectants, acidic conditions). Because of its microaerophilic characteristics the organism requires 3 to 5% oxygen and 2 to 10% carbon dioxide for optimal growth conditions. This bacterium is now recognized as an important enteric pathogen. Before 1972, when methods were developed for its isolation from feces, it was believed to be primarily an animal pathogen causing abortion and enteritis in sheep and cattle. Surveys have shown that *C. jejuni* is the leading cause of bacterial diarrheal illness in the United States. It causes more disease than *Shigella* spp. and *Salmonella* spp. combined.

Although *C. jejuni* is not carried by healthy individuals in the United States or Europe, it is often isolated from healthy cattle, chickens, birds and even flies. It is sometimes present in non-chlorinated water sources such as streams and ponds.

Because the pathogenic mechanisms of *C. jejuni* are still being studied, it is difficult to differentiate pathogenic from nonpathogenic strains. However, it appears that many of the chicken isolates are pathogens.

2. Name of Disease:

Campylobacteriosis is the name of the illness caused by *C. jejuni*. It is also often known as campylobacter enteritis or gastroenteritis.

3. Major Symptoms:

C. jejuni infection causes diarrhea, which may be watery or sticky and can contain blood (usually occult) and fecal leukocytes (white cells). Other symptoms often present are fever, abdominal pain, nausea, headache and muscle pain. The illness usually occurs 2-5 days after ingestion of the contaminated food or water. Illness generally lasts 7-10 days, but relapses are not uncommon (about 25% of cases). Most infections are self-limiting and are not treated with antibiotics. However, treatment with erythromycin does reduce the length of time that infected individuals shed the bacteria in their feces.

The infective dose of *C. jejuni* is considered to be small. Human feeding studies suggest that about 400-500 bacteria may cause illness in some individuals, while in others, greater numbers are required. A conducted volunteer human feeding study suggests that host susceptibility also dictates infectious dose to some degree. The pathogenic mechanisms of *C. jejuni* are still not completely understood, but it does produce a heat-labile toxin that

may cause diarrhea. *C. jejuni* may also be an invasive organism.

4. Isolation Procedures: *C. jejuni* is usually present in high numbers in the diarrheal stools of individuals, but isolation requires special antibiotic-containing media and a special microaerophilic atmosphere (5% oxygen). However, most clinical laboratories are equipped to isolate Campylobacter spp. if requested.

5. Associated Foods: *C. jejuni* frequently contaminates raw chicken. Surveys show that 20 to 100% of retail chickens are contaminated. This is not overly surprising since many healthy chickens carry these bacteria in their intestinal tracts. Raw milk is also a source of infections. The bacteria are often carried by healthy cattle and by flies on farms. Non-chlorinated water may also be a source of infections. However, properly cooking chicken, pasteurizing milk, and chlorinating drinking water will kill the bacteria.

6. Frequency of the Disease: *C. jejuni* is the leading cause of bacterial diarrhea in the U.S. There are probably numbers of cases in excess of the estimated cases of salmonellosis (2- to 4,000,000/year).

7. Complications: Complications are relatively rare, but infections have been associated with reactive arthritis, hemolytic uremic syndrome, and following septicemia, infections of nearly any organ. The estimated case/fatality ratio for all *C. jejuni* infections is 0.1, meaning one death per 1,000 cases. Fatalities are rare in healthy individuals and usually occur in cancer patients or in the otherwise debilitated. Only 20 reported cases of septic abortion induced by *C. jejuni* have been recorded in the literature.

Meningitis, recurrent colitis, acute cholecystitis and Guillain-Barre syndrome are very rare complications.

8. Target Populations: Although anyone can have a *C. jejuni* infection, children under 5 years and young adults (15-29) are more frequently afflicted than other age groups. Reactive arthritis, a rare complication of these infections, is strongly associated with people who have the human lymphocyte antigen B27 (HLA-B27).

9. Recovery from Foods: Isolation of *C. jejuni* from food is difficult because the bacteria are usually present in very low numbers (unlike the case of diarrheal stools in which 10/6 bacteria/gram is not unusual). The methods require an enrichment broth containing antibiotics, special antibiotic-containing plates and a microaerophilic atmosphere generally a microaerophilic atmosphere with 5% oxygen and an elevated concentration of carbon dioxide (10%). Isolation can take several days to a week.

10. Selected Outbreaks: Usually outbreaks are small (less than 50 people), but in Bennington, VT a large outbreak involving about 2,000 people occurred while the town was temporarily using an non-chlorinated water source as a water supply. Several small outbreaks have been reported among children who were taken on a class trip to a dairy and given raw milk to drink. An outbreak was also associated with consumption of raw clams. However, a survey showed that about 50% of infections are associated with either eating inadequately cooked or recontaminated chicken meat or handling chickens. It is the leading bacterial cause of sporadic (non-clustered cases) diarrheal disease in the U.S.

In April, 1986, an elementary school child was cultured for bacterial pathogens (due to bloody diarrhea), and *C. jejuni* was isolated. Food consumption/gastrointestinal illness questionnaires were administered to other students and faculty at the school. In all, 32 of 172 students reported symptoms of diarrhea (100%), cramps (80%), nausea (51%), fever (29%), vomiting (26%), and bloody stools (14%). The food questionnaire clearly implicated milk as the common source, and a dose/response was evident (those drinking more milk were more likely to be ill). Investigation of the dairy supplying the milk showed that they vat pasteurized the milk at 135°F for 25 minutes rather than the required 145°F for 30 minutes. The dairy processed surplus raw milk for the school, and this milk had a high somatic cell count. Cows from the herd supplying the dairy had *C. jejuni* in their feces. This outbreak points out the variation in symptoms which may occur with campylobacteriosis and the absolute need to adhere to pasteurization time/temperature standards.

Although other *Campylobacter* spp. have been implicated in human gastroenteritis (e.g. *C. laridis*, *C. hyointestinalis*), it is believed that 99% of the cases are caused by *C. jejuni*.

Information regarding an outbreak of Campylobacter in New Zealand is found in this MMWR 40(7):1991 Feb 22.

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

11. Education: The Food Safety Inspection Service of the U.S. Department of Agriculture has produced a background document on *Campylobacter*.

12. Other Resources: A Loci index for genome *Campylobacter jejuni* is available from GenBank.

CDC/MMWR

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Yersinia enterocolitica

CDC/MMWR NIH/PubMed

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1. Name of the Organism: *Yersinia enterocolitica* (and *Yersinia pseudotuberculosis*)

Y. enterocolitica, a small rod-shaped, Gram-negative bacterium, is often isolated from clinical specimens such as wounds, feces, sputum and mesenteric lymph nodes. However, it is not part of the normal human flora. *Y. pseudotuberculosis* has been isolated from the diseased appendix of humans.

Both organisms have often been isolated from such animals as pigs, birds, beavers, cats, and dogs. Only *Y. enterocolitica* has been detected in environmental and food sources, such as ponds, lakes, meats, ice cream, and milk. Most isolates have been found not to be pathogenic.

2. Name of Disease: Yersiniosis

There are 3 pathogenic species in the genus Yersinia, but only *Y. enterocolitica* and *Y. pseudotuberculosis* cause gastroenteritis. To date, no foodborne outbreaks caused by *Y. pseudotuberculosis* have been reported in the United States, but human infections transmitted via contaminated water and foods have been reported in Japan. *Y. pestis*, the causative agent of " the plague," is genetically very similar to *Y. pseudotuberculosis* but infects humans by routes other than food.

3. Nature of Disease: Yersiniosis is frequently characterized by such symptoms as gastroenteritis with diarrhea and/or vomiting; however, fever and abdominal pain are the hallmark symptoms. *Yersinia* infections mimic appendicitis and mesenteric lymphadenitis, but the bacteria may also cause infections of other sites such as wounds, joints and the urinary tract.

4. Infective dose: Unknown.

Illness onset is usually between 24 and 48 hours after ingestion, which (with food or drink as vehicle) is the usual route of infection.

5. Diagnosis of Human Illness: Diagnosis of yersiniosis begins with isolation of the organism from the human host's feces, blood, or vomit, and sometimes at the time of appendectomy. Confirmation occurs with the isolation, as well as biochemical and serological identification, of *Y. enterocolitica* from both the human host and the ingested foodstuff. Diarrhea is reported to occur in about 80% of cases; abdominal pain and fever are the most reliable symptoms.

Because of the difficulties in isolating *yersiniae* from feces, several

countries rely on serology. Acute and convalescent patient sera are titered against the suspect serotype of *Yersinia spp.*

Yersiniosis has been misdiagnosed as Crohn's disease (regional enteritis) as well as appendicitis.

6. Associated Foods:

Strains of *Y. enterocolitica* can be found in meats (pork, beef, lamb, etc.), oysters, fish, and raw milk. The exact cause of the food contamination is unknown. However, the prevalence of this organism in the soil and water and in animals such as beavers, pigs, and squirrels, offers ample opportunities for it to enter our food supply. Poor sanitation and improper sterilization techniques by food handlers, including improper storage, cannot be overlooked as contributing to contamination.

7. Frequency of the Disease:

Yersiniosis does not occur frequently. It is rare unless a breakdown occurs in food processing techniques. CDC estimates that about 17,000 cases occur annually in the USA. Yersiniosis is a far more common disease in Northern Europe, Scandinavia, and Japan.

8. Complications:

The major "complication" is the performance of unnecessary appendectomies, since one of the main symptoms of infections is abdominal pain of the lower right quadrant.

Both *Y. enterocolitica* and *Y. pseudotuberculosis* have been associated with reactive arthritis, which may occur even in the absence of obvious symptoms. The frequency of such postenteritis arthritic conditions is about 2-3%.

Another complication is bacteremia (entrance of organisms into the blood stream), in which case the possibility of a disseminating disease may occur. This is rare, however, and fatalities are also extremely rare.

9. Target Populations:

The most susceptible populations for the main disease and possible complications are the very young, the debilitated, the very old and persons undergoing immunosuppressive therapy. Those most susceptible to postenteritis arthritis are individuals with the antigen HLA-B27 (or related antigens such as B7).

10. Food Analysis:

The isolation method is relatively easy to perform, but in some instances, cold enrichment may be required. *Y. enterocolitica* can be presumptively identified in 36-48 hours. However, confirmation may take 14-21 days or more. Determination of pathogenicity is more complex. The genes encoding for invasion of mammalian cells are located on the chromosome while a 40-50 MDal plasmid encodes most of the other virulence associated phenotypes. The 40-50 MDal plasmid is present in almost all the pathogenic *Yersinia* species, and the plasmids appear to be homologous.

11. Selected Outbreaks:

1976. A chocolate milk outbreak in Oneida County, N.Y. involving school children (first reported yersiniosis incident in the United States in which a food vehicle was identified). A research laboratory was set up by FDA to investigate and study *Y. enterocolitica* and *Y. pseudotuberculosis* in the human food supply.

Dec. 1981 - Feb. 1982. *Y. enterocolitica* enteritis in King County, Washington caused by ingestion of tofu, a soybean curd. FDA investigators and researchers determined the source of the infection to be an non-chlorinated water supply. Manufacturing was halted until uncontaminated product was produced.

June 11 to July 21, 1982. *Y. enterocolitica* outbreak in Arkansas, Tennessee, and Mississippi associated with the consumption of pasteurized milk. FDA personnel participated in the investigation, and presumptively identified the infection source to be externally contaminated milk containers.

A report of *Yersinia enterocolitica* incidents associated with raw chitterlings may be found in MMWR 39(45):1990 Nov 16

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

12. Other Resources:

A Loci index for genome *Yersinia enterocolitica* and Loci index for genome *Yersinia pseudotuberculosis* are available from GenBank.

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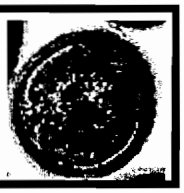

Listeria monocytogenes

Education | CDC/MMWR | NIH/PubMed

1. Name of the Organism: *Listeria monocytogenes*

This is a Gram-positive bacterium, motile by means of flagella. Some studies suggest that 1-10% of humans may be intestinal carriers of *L. monocytogenes*. It has been found in at least 37 mammalian species, both domestic and feral, as well as at least 17 species of birds and possibly some species of fish and shellfish. It can be isolated from soil, silage, and other environmental sources. *L. monocytogenes* is quite hardy and resists the deleterious effects of freezing, drying, and heat remarkably well for a bacterium that does not form spores. Most *L. monocytogenes* are pathogenic to some degree.

2. Name of Acute Disease:

Listeriosis is the name of the general group of disorders caused by *L. monocytogenes*.

3. Nature of Disease:

Listeriosis is clinically defined when the organism is isolated from blood, cerebrospinal fluid, or an otherwise normally sterile site (e.g. placenta, fetus).

The manifestations of listeriosis include septicemia, meningitis (or meningoencephalitis), encephalitis, and intrauterine or cervical infections in pregnant women, which may result in spontaneous abortion (2nd/3rd trimester) or stillbirth. The onset of the aforementioned disorders is usually preceded by influenza-like symptoms including persistent fever. It was reported that gastrointestinal symptoms such as nausea, vomiting, and diarrhea may precede more serious forms of listeriosis or may be the only symptoms expressed. Gastrointestinal symptoms were epidemiologically associated with use of antacids or cimetidine. The onset time to serious forms of listeriosis is unknown but may range from a few days to three weeks. The onset time to gastrointestinal symptoms is unknown but is probably greater than 12 hours.

The infective dose of *L. monocytogenes* is unknown but is believed to vary with the strain and susceptibility of the victim. From cases contracted through raw or supposedly pasteurized milk, it is safe to assume that in susceptible persons, fewer than 1,000 total organisms may cause disease. *L. monocytogenes* may invade the gastrointestinal epithelium. Once the bacterium enters the host's monocytes, macrophages, or polymorphonuclear leukocytes, it is bloodborne (septicemic) and can grow. Its presence intracellularly in phagocytic cells also permits access to the brain and probably transplacental migration to the fetus in pregnant women. The pathogenesis of *L. monocytogenes* centers on its ability to survive and multiply in phagocytic host cells.

4. Diagnosis of Human Illness: Listeriosis can only be positively diagnosed by culturing the organism from blood, cerebrospinal fluid, or stool (although the latter is difficult and of limited value).

5. Associated Foods: *L. monocytogenes* has been associated with such foods as raw milk, supposedly pasteurized fluid milk, cheeses (particularly soft-ripened varieties), ice cream, raw vegetables, fermented raw-meat sausages, raw and cooked poultry, raw meats (all types), and raw and smoked fish. Its ability to grow at temperatures as low as 3oC permits multiplication in refrigerated foods.

6. Frequency of the Disease: The 1987 incidence data prospectively collected by CDC suggests that there are at least 1600 cases of listeriosis with 415 deaths per year in the U.S. The vast majority of cases are sporadic, making epidemiological links to food very difficult.

7. Complications: Most healthy persons probably show no symptoms. The "complications" are the usual clinical expressions of the disease.

When listeric meningitis occurs, the overall mortality may be as high as 70%; from septicemia 50%, from perinatal/neonatal infections greater than 80%. In infections during pregnancy, the mother usually survives. Successful treatment with parenteral penicillin or ampicillin has been reported. Trimethoprim-sulfamethoxazole has been shown effective in patients allergic to penicillin.

8. Target Populations: The main target populations for listeriosis are:

- pregnant women/fetus - perinatal and neonatal infections;
- persons immunocompromised by corticosteroids, anticancer drugs, graft suppression therapy, AIDS;
- cancer patients - leukemic patients particularly;
- less frequently reported - diabetic, cirrhotic, asthmatic, and ulcerative colitis patients;
- the elderly;
- normal people--some reports suggest that normal, healthy people are at risk, although antacids or cimetidine may predispose. A listerosis outbreak in Switzerland involving cheese suggested that healthy uncompromised individuals could develop the disease, particularly if the foodstuff was heavily contaminated with the organism.

9. Food Analysis: The methods for analysis of food are complex and time consuming. The present FDA method, revised in September, 1990, requires 24 and 48 hours of enrichment, followed by a variety of other tests. Total time to identification is from 5 to 7 days, but the announcement of specific nonradiolabled DNA probes should soon allow a simpler and faster confirmation of suspect isolates.

Recombinant DNA technology may even permit 2-3 day positive analysis in the future. Currently, FDA is collaborating in adapting its methodology to quantitate very low numbers of the organisms in foods.

10. Selected Outbreaks:

Outbreaks include the California episode in 1985, which was due to Mexican-style cheese and led to numerous stillbirths. As a result of this episode, FDA has been monitoring domestic and imported cheeses and has taken numerous actions to remove these products from the market when *L. monocytogenes* is found.

There have been other clustered cases, such as in Philadelphia, PA, in 1987. Specific food linkages were only made epidemiologically in this cluster.

CDC has established an epidemiological link between consumption of raw hot dogs or undercooked chicken and approximately 20% of the sporadic cases under prospective study.

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

11. Education:

The FDA health alert for hispanic pregnant women concerns the risk of listeriosis from soft cheeses. The CDC provides similar information in spanish.

The Food Safety and Inspection Service of the U.S. Department of Agriculture has jointly produced with the FDA a background document on *Listeria* and Listeriosis. FSIS also has updated consumer information on Listeria dated February 1999.

The CDC produces an information brochure on preventing Listeriosis.

12. Other Resources:

A Loci index for genome *Listeria monocytogenes* is available from GenBank.

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Vibrio cholerae Serogroup O1

Education | CDC/MMWR | NIH/PubMed

1. Name of the Organism:
Vibrio cholerae Serogroup O1

This bacterium is responsible for Asiatic or epidemic cholera. No major outbreaks of this disease have occurred in the United States since 1911. However, sporadic cases occurred between 1973 and 1991, suggesting the possible reintroduction of the organism into the U.S. marine and estuarine environment. The cases between 1973 and 1991 were associated with the consumption of raw shellfish or of shellfish either improperly cooked or recontaminated after proper cooking. Environmental studies have demonstrated that strains of this organism may be found in the temperate estuarine and marine coastal areas surrounding the United States.

In 1991 outbreaks of cholera in Peru quickly grew to epidemic proportions and spread to other South American and Central American countries, including Mexico. Over 340,000 cases and 3,600 deaths have been reported in the Western Hemisphere since January 1991. However, only 24 cases of cholera have been reported in the United States. The U.S. cases were brought into the country by travelers returning from South America, or were associated with illegally smuggled, temperature-abused crustaceans.

2. Name of the Acute Disease:

Cholera is the name of the infection caused by *V. cholerae*.

3. Nature of the Disease:

Symptoms of Asiatic cholera may vary from a mild, watery diarrhea to an acute diarrhea, with characteristic rice water stools. Onset of the illness is generally sudden, with incubation periods varying from 6 hours to 5 days. Abdominal cramps, nausea, vomiting, dehydration, and shock; after severe fluid and electrolyte loss, death may occur. Illness is caused by the ingestion of viable bacteria, which attach to the small intestine and produce cholera toxin. The production of cholera toxin by the attached bacteria results in the watery diarrhea associated with this illness.

Infective dose -- Human volunteer feeding studies utilizing healthy individuals have demonstrated that approximately one million organisms must be ingested to cause illness. Antacid consumption markedly lowers the infective dose.

4. Diagnosis of Human Illness:

Cholera can be confirmed only by the isolation of the causative organism from the diarrheic stools of infected individuals.

5. Foods in which it Occurs:

Cholera is generally a disease spread by poor sanitation, resulting in contaminated water supplies. This is clearly the main mechanism for the spread of cholera in poor communities in South America. The excellent sanitation facilities in the U.S. are responsible for the near eradication of epidemic cholera. Sporadic cases occur when shellfish harvested from fecally polluted coastal waters are consumed raw. Cholera may also be transmitted by shellfish harvested from nonpolluted waters since *V. cholerae* O1 is part of the autochthonous microbiota of these waters.

6. Frequency of Disease:

Fewer than 80 proven cases of cholera have been reported in the U.S. since 1973. Most of these cases were detected only after epidemiological investigation. Probably more sporadic cases have occurred, but have gone undiagnosed or unreported.

7. The Usual Course of Disease and Some Complications:

Individuals infected with cholera require rehydration either intravenously or orally with a solution containing sodium chloride, sodium bicarbonate, potassium chloride, and dextrose (glucose). The illness is generally self-limiting. Antibiotics such as tetracycline have been demonstrated to shorten the course of the illness. Death occurs from dehydration and loss of essential electrolytes. Medical treatment to prevent dehydration prevents all complications.

8. Target Populations:

All people are believed to be susceptible to infection, but individuals with damaged or undeveloped immunity, reduced gastric acidity, or malnutrition may suffer more severe forms of the illness.

9. Analysis of Foods:

V. cholerae serogroup O1 may be recovered from foods by methods similar to those used for recovering the organism from the feces of infected individuals. Pathogenic and non- pathogenic forms of the organism exist, so all food isolates must be tested for the production of cholera enterotoxin.

10. Selected Outbreaks:

An incident of cholera in Indiana from imported food is reported in MMWR 44(20):1995 May 20 .

See MMWR 44(11):1995 Mar 24 for an updated report on *Vibrio cholerae* O1 in the Western Hemisphere 1991-1994 and on *V. cholerae* O139 in Asia, 1994.

Surveillance for cholera in Cochabamba Department, Bolivia is discussed in in this MMWR 42(33):1993 Aug 27.

The cholera outbreak in Burundi and Zimbabwe is detailed in the following MMWR 42(21):1993 Jun 04.

MMWR 40(49):1991 Dec 13 reports on a cholera outbreak associated with imported coconut milk.

A report of a cholera incident in New York is found in MMWR 40 (30):1991 Aug 01.

Similar incidents in New Jersey and Florida are reported in MMWR 40 (17):1991 May 03.

A case of importation of cholera from Peru to the United States is detailed in MMWR 40(15):1991 Apr 19.

The cholera outbreak in Peru is reported on in MMWR:40(6):1991 Feb 15, and the update of the South American endemic is in MMWR 40 (13):1991 Apr 5.

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

11. Education: The CDC has a brochure on the prevention of cholera

- in English
- in Spanish
- in Portuguese

12. Other Resources A Loci index for genome *Vibrio cholerae* is available from GenBank.

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Vibrio cholerae Serogroup Non-O1

CDC/MMWR NIH/PubMed

1. Name of the Organism: *Vibrio cholerae* Serogroup Non-O1	This bacterium infects only humans and other primates. It is related to *V. cholerae* Serogroup O1, the organism that causes Asiatic or epidemic cholera, but causes a disease less severe than cholera. Both pathogenic and nonpathogenic strains of the organism are normal inhabitants of marine and estuarine environments of the United States. This organism has been referred to as non-cholera vibrio (NCV) and nonagglutinable vibrio (NAG) in the past.
2. Name of Acute Disease:	Non-O1 *V. cholerae* gastroenteritis is the name associated with this illness.
3. Nature of the Disease:	Diarrhea, abdominal cramps, and fever are the predominant symptoms associated with this illness, with vomiting and nausea occurring in approximately 25% of infected individuals. Approximately 25% of infected individuals will have blood and mucus in their stools. Diarrhea may, in some cases, be quite severe, lasting 6-7 days. Diarrhea will usually occur within 48 hours following ingestion of the organism. It is unknown how the organism causes the illness, although an enterotoxin is suspected as well as an invasive mechanism. Disease is caused when the organism attaches itself to the small intestine of infected individuals and perhaps subsequently invades. Infective dose -- It is suspected that large numbers (more than one million) of the organism must be ingested to cause illness.
4. Diagnosis of Human Illness:	Diagnosis of a *V. cholerae* non-O1 infection is made by culturing the organism from an individual's diarrheic stool.
5. Foods in which it Occurs:	Shellfish harvested from U.S. coastal waters frequently contain *V. cholerae* serogroup non-O1. Consumption of raw, improperly cooked or cooked, recontaminated shellfish may lead to infection.
6. Relative Frequency of Disease:	No major outbreaks of diarrhea have been attributed to this organism. Sporadic cases occur frequently mainly along the coasts of the U.S., and are usually associated with the consumption of raw oysters during the warmer months.

7. The Usual Course of Disease and Some Complications:

Diarrhea resulting from ingestion of the organism usually lasts 7 days and is self-limiting. Antibiotics such as tetracycline shorten the severity and duration of the illness. Septicemia (bacteria gaining entry into the blood stream and multiplying therein) can occur. This complication is associated with individuals with cirrhosis of the liver, or who are immunosuppressed, but this is relatively rare. FDA has warned individuals with liver disease to refrain from consuming raw or improperly cooked shellfish.

8. Target Populations:

All individuals who consume raw shellfish are susceptible to diarrhea caused by this organism. Cirrhotic or immunosuppressed individuals may develop severe complications such as septicemia.

9. Analysis of Foods:

Methods used to isolate this organism from foods are similar to those used with diarrheic stools. Because many food isolates are nonpathogenic, pathogenicity of all food isolates must be demonstrated. All virulence mechanisms of this group have not been elucidated; therefore, pathogenicity testing must be performed in suitable animal models.

10. Selected Outbreaks:

Sporadic cases continue to occur all year, increasing in frequency during the warmer months.

An update report from CDC on *Vibrio cholerae* O139 in Asia may be found in MMWR 44(11):1995 Mar 24.

See MMWR 42(26):1993 Jul 09 for a report on the new O139 Non-O1 *Vibrio cholerae* (Bengal).

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

CDC/MMWR

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U.S. Food & Drug Administration
Center for Food Safety & Applied Nutrition
Foodborne Pathogenic Microorganisms and Natural Toxins Handbook

Vibrio parahaemolyticus

CDC/MMWR NIH/PubMed

1. Name of the Organism: *Vibrio parahaemolyticus* (and other marine *Vibrio* spp.**)	This bacterium is frequently isolated from the estuarine and marine environment of the United States. Both pathogenic and non-pathogenic forms of the organism can be isolated from marine and estuarine environments and from fish and shellfish dwelling in these environments.
2. Name of Acute Disease:	*V. parahaemolyticus*-associated gastroenteritis is the name of th infection caused by this organism.
3. Nature of the Disease:	Diarrhea, abdominal cramps, nausea, vomiting, headache, fever and chills may be associated with infections caused by this organism. The illness is usually mild or moderate, although some cases may require hospitalization. The median duration of the illness is 2.5 days. The incubation period is 4-96 hours after the ingestion of the organism, with a mean of 15 hours. Disease is caused when the organism attaches itself to an individuals' small intestine and excretes an as yet unidentified toxin. Infective dose -- A total dose of greater than one million organisms may cause disease; this is markedly lowered by antacids (or presumably by food with buffering capability).
4. Diagnosis of Human Illness:	Diagnosis of gastroenteritis caused by this organism is made by culturing the organism from the diarrheic stools of an individua
5. Associated Foods:	Infections with this organism have been associated with the consumption of raw, improperly cooked, or cooked, recontaminated fish and shellfish. A correlation exists between the probability of infection and warmer months of the year. Improper refrigeration of seafoods contaminated with this organism will allow its proliferation, which increases the possibility of infection.
6. Relative Frequency of Disease:	Major outbreaks have occurred in the U.S. during the warmer months of the year. Sporadic cases occur frequently along all coasts of the U.S.
7. The Usual Course of the Disease:	Diarrhea caused by this organism is usually self-limiting, with few cases requiring hospitalization and/or antibiotic treatment.
8. Target populations:	All individuals who consume raw or improperly cooked fish an shellfish are susceptible to infection by this organism.

9. Analysis of Foods:

Methods used to isolate this organism from foods are similar to those used with diarrheic stools. Because many food isolates ar nonpathogenic, pathogenicity of all food isolates must be demonstrated. Although the demonstration of the Kanagawa hemolysin was long considered indicative of pathogenicity, this is now uncertain.

10. Selected Outbreaks:

Sporadic outbreaks of gastroenteritis caused by this organism have occurred in the U.S. and cases are more common during th warmer months. It is very common in Japan, where large outbreaks occur with regularity.

**OTHER MARINE VIBRIOS IMPLICATED IN FOODBORNE DISEASE:

Several other marine vibrios have been implicated in human disease. Some may cause wound or ear infections, and others, gastroenteritis. The amount of evidence for certain of these organisms as being causative of human gastroenteritis is small. Nonetheless, several have been isolated from human feces from diarrhea patients from which no other pathogens could be isolated. Methods for recovery of these organisms from foods are similar to those used for recovery of *V. parahaemolyticus*. The species implicated in human disease include:

Vibrio alginolyticus
Vibrio carchariae
Vibrio cincinnatiensis
Vibrio damsela
Vibrio fluvialis
Vibrio furnissii
Vibrio hollisae
Vibrio metschnikovi
Vibrio mimicus

For more information on recent outbreaks see the Morbidit and Mortality Weekly Reports from CDC.

11. Other Resources:

A Loci index for genome *Vibrio parahaemolyticus* is available from GenBank.

CDC/MMWR

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Foodborne Pathogenic Microorganisms and Natural Toxins Handbook

Vibrio vulnificus

Education | CDC/MMWR | NIH/PubMed

1. Name of the Organism:
Vibrio vulnificus

This bacterium infects only humans and other primates. It has been isolated from a wide range of environmental sources, including water, sediment, plankton, and shellfish (oysters, clams, and crabs) and a variety of locations, including the Gulf of Mexico, the Atlantic Coast as far north as Cape Cod, and the entire U.S. west coast. Cases of illness have also been associated with brackish lakes in New Mexico and Oklahoma.

2. Name of the Acute Disease:

This organism causes wound infections, gastroenteritis, or a syndrome known as "primary septicemia."

3. Nature of the Disease:

Wound infections result either from contaminating an open wound with sea water harboring the organism, or by lacerating part of the body on coral, fish, etc., followed by contamination with the organism. The ingestion of *V. vulnificus* by healthy individuals can result in gastroenteritis. The "primary septicemia" form of the disease follows consumption of raw seafood containing the organism by individuals with underlying chronic disease, particularly liver disease (see below). In these individuals, the microorganism enters the blood stream, resulting in septic shock, rapidly followed by death in many cases (about 50%). Over 70% of infected individuals have distinctive bulbous skin lesions.

Infective dose -- The infective dose for gastrointestinal symptoms in healthy individuals is unknown but for predisposed persons, septicemia can presumably occur with doses of less than 100 total organisms.

4. Diagnosis of Human Illness:

The culturing of the organism from wounds, diarrheic stools, or blood is diagnostic of this illness.

5 .Associated Foods:

This organism has been isolated from oysters, clams, and crabs. Consumption of these products raw or recontaminated may result in illness.

6. Relative Frequency of Disease:

No major outbreaks of illness have been attributed to this organism. Sporadic cases occur frequently, becoming more prevalent during the warmer months.

In a survey of cases of *V. vulnificus* infections in Florida from 1981 to 1987, Klontz et al. (Annals of Internal Medicine 109:318-23;1988) reported that 38 cases of primary septicemia (ingestion), 17 wound infections, and 7 cases gastroenteritis were associated with the organism. Mortality from infection varied from 55% for primary septicemia cases,

to 24% with wound infections, to no deaths associated with gastroenteritis. Raw oyster consumption was a common feature of primary septicemia and gastroenteritis, and liver disease was a feature of primary septicemia.

7. The Usual Course of Disease and Some Complications:

In healthy individuals, gastroenteritis usually occurs within 16 hours of ingesting the organism. Ingestion of the organism by individuals with some type of chronic underlying disease [such as diabetes, cirrhosis, leukemia , lung carcinoma, acquired immune deficiency syndrome (AIDS), AIDS- related complex (ARC), or asthma requiring the use of steroids] may cause the "primary septicemia" form of illness. The mortality rate for individuals with this form of the disease is over 50%.

8. Target Populations:

All individuals who consume foods contaminated with this organism are susceptible to gastroenteritis. Individuals with diabetes, cirrhosis, or leukemia, or those who take immunosuppressive drugs or steroids are particularly susceptible to primary septicemia. These individuals should be strongly advised not to consume raw or inadequately cooked seafood, as should AIDS/ARC patients.

9. Analysis of Foods:

Methods used to isolate this organism from foods are similar to those used with diarrheic stools. To date, all food isolates of this organism have been pathogenic in animal models.

FDA has a genetic probe for *V. vulnificus*; its target is a cytotoxin gene which appears not to correlate with the organism's virulence.

10. Selected Outbreaks:

Sporadic cases continue to occur all year, increasing in frequency during the warmer months.

MMWR 45(28):1996 Jul 26 reports on three incidents of *V. vulnificus* infection in Los Angeles, California.

A multi-year summary of *V. vulnificus* incidents associated with the consumption of raw oysters is reported in MMWR 42(21):1993 Jun 04

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

11. Education:

More information for consumers of raw shellfish is available in the FDA brochure If You Eat Raw Oysters, You Need to Know . . ..

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U.S. Food & Drug Administration
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Clostridium perfringens

CDC/MMWR NIH/PubMed

1. Name of Organism:
Clostridium perfringens

Clostridium perfringens is an anaerobic, Gram-positive, sporeforming rod (anaerobic means unable to grow in the presence of free oxygen). It is widely distributed in the environment and frequently occurs in the intestines of humans and many domestic and feral animals. Spores of the organism persist in soil, sediments, and areas subject to human or animal fecal pollution.

2. Name of Acute Disease:

Perfringens food poisoning is the term used to describe the common foodborne illness caused by *C. perfringens*. A more serious but rare illness is also caused by ingesting food contaminated with Type C strains. The latter illness is known as enteritis necroticans or pig-bel disease.

3. Nature of Disease:

The common form of perfringens poisoning is characterized by intense abdominal cramps and diarrhea which begin 8-22 hours after consumption of foods containing large numbers of those *C. perfringens* bacteria capable of producing the food poisoning toxin. The illness is usually over within 24 hours but less severe symptoms may persist in some individuals for 1 or 2 weeks. A few deaths have been reported as a result of dehydration and other complications.

Necrotic enteritis (pig-bel) caused by *C. perfringens* is often fatal. This disease also begins as a result of ingesting large numbers of the causative bacteria in contaminated foods. Deaths from necrotic enteritis (pig-bel syndrome) are caused by infection and necrosis of the intestines and from resulting septicemia. This disease is very rare in the U.S.

Infective dose--The symptoms are caused by ingestion of large numbers (greater than 10 to the 8th) vegetative cells. Toxin production in the digestive tract (or in test tubes) is associated with sporulation. This disease is a food infection; only one episode has ever implied the possibility of intoxication (i.e., disease from preformed toxin).

4. Diagnosis of Human Illness:

Perfringens poisoning is diagnosed by its symptoms and the typical delayed onset of illness. Diagnosis is confirmed by detecting the toxin in the feces of patients. Bacteriological confirmation can also be done by finding exceptionally large numbers of the causative bacteria in implicated foods or in the feces of patients.

5. Associated Foods and Food Handling:

In most instances, the actual cause of poisoning by *C. perfringens* is temperature abuse of prepared foods. Small numbers of the organisms are often present after cooking and multiply to food poisoning levels during cool down and storage of prepared foods. Meats, meat products, and gravy are the foods most frequently implicated.

6. Frequency:

Perfringens poisoning is one of the most commonly reported foodborne illnesses in the U.S. There were 1,162 cases in 1981, in 28 separate outbreaks. At least 10-20 outbreaks have been reported annually in the U.S. for the past 2 decades. Typically, dozens or even hundreds of person are affected. It is probable that many outbreaks go unreported because the implicated foods or patient feces are not tested routinely for *C. perfringens* or its toxin. CDC estimates that about 10,000 actual cases occur annually in the U.S.

7. Usual Course of Disease and Complications:

The disease generally lasts 24 hours. In the elderly or infirm, symptoms may last 1-2 weeks. Complications and/or death only very rarely occur.

8. Target Populations:

Institutional feeding (such as school cafeterias, hospitals, nursing homes, prisons, etc.) where large quantities of food are prepared several hours before serving is the most common circumstance in which perfringens poisoning occurs. The young and elderly are the most frequent victims of perfringens poisoning. Except in the case of pig-bel syndrome, complications are few in persons under 30 years of age. Elderly persons are more likely to experience prolonged or severe symptoms.

9. Analysis of Food and Feces:

Standard bacteriological culturing procedures are used to detect the organism in implicated foods and in feces of patients. Serological assays are used for detecting enterotoxin in the feces of patients and for testing the ability of strains to produce toxin. The procedures take 1-3 days.

10. Selected Outbreaks:

Since December 1981, FDA has investigated 10 outbreaks in 5 states. In two instances, more than one outbreak occurred in the same feeding facility within a 3-week period. One such outbreak occurred on 19 March 1984, involving 77 prison inmates. Roast beef served as a luncheon meat was implicated as the food vehicle and *C. perfringens* was confirmed as the cause by examining stools of 24 patients. Most of the patients became ill 8-16 hours after the meal. Eight days later, on 27 March 1984, a second outbreak occurred involving many of the same persons. The food vehicle was ham. Inadequate refrigeration and insufficient reheating of the implicated foods caused the outbreaks. Most of the other outbreaks occurred in institutional feeding environments: a hospital, nursing home, labor camp, school cafeteria, and at a fire house luncheon.

In November, 1985, a large outbreak of *C. perfringens* gastroenteritis occurred among factory workers in Connecticut. Forty-four percent of the 1,362 employees were affected. Four main-course foods served at an employee banquet were associated

with illness, but gravy was implicated by stratified analysis. The gravy had been prepared 12-24 hours before serving, had been improperly cooled, and was reheated shortly before serving. The longer the reheating period, the less likely the gravy was to cause illness.

A outbreak of *C. perfringens* in corned beef was reported in MMWR 43(8):1994 Mar 04.

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

CDC/MMWR

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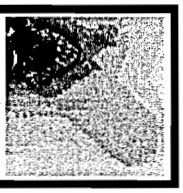

Bacillus cereus and other *Bacillus* spp.

CDC/MMWR NIH/PubMed

1. Name of the Organism: *Bacillus cereus* and other *Bacillus* spp.

Bacillus cereus is a Gram-positive, facultatively aerobic sporeformer whose cells are large rods and whose spores do not swell the sporangium. These and other characteristics, including biochemical features, are used to differentiate and confirm the presence *B. cereus*, although these characteristics are shared with *B. cereus* var. mycoides, *B. thuringiensis* and *B. anthracis*. Differentiation of these organisms depends upon determination of motility (most *B. cereus* are motile), presence of toxin crystals (*B. thuringiensis*), hemolytic activity (*B. cereus* and others are beta hemolytic whereas *B. anthracis* is usually nonhemolytic), and rhizoid growth which is characteristic of *B. cereus* var. mycoides.

2. Name of Illness:

B. cereus food poisoning is the general description, although two recognized types of illness are caused by two distinct metabolites. The diarrheal type of illness is caused by a large molecular weight protein, while the vomiting (emetic) type of illness is believed to be caused by a low molecular weight, heat-stable peptide.

3. Nature of Illness:

The symptoms of *B. cereus* diarrheal type food poisoning mimic those of *Clostridium perfringens* food poisoning. The onset of watery diarrhea, abdominal cramps, and pain occurs 6-15 hours after consumption of contaminated food. Nausea may accompany diarrhea, but vomiting (emesis) rarely occurs. Symptoms persist for 24 hours in most instances.

The emetic type of food poisoning is characterized by nausea and vomiting within 0.5 to 6 h after consumption of contaminated foods. Occasionally, abdominal cramps and/or diarrhea may also occur. Duration of symptoms is generally less than 24 h. The symptoms of this type of food poisoning parallel those caused by *Staphylococcus aureus* foodborne intoxication. Some strains of *B. subtilis* and *B. licheniformis* have been isolated from lamb and chicken incriminated in food poisoning episodes. These organisms demonstrate the production of a highly heat-stable toxin which may be similar to the vomiting type toxin produced by *B. cereus*.

The presence of large numbers of *B. cereus* (greater than 10^6 organisms/g) in a food is indicative of active growth and proliferation of the organism and is consistent with a potential hazard to health.

4. Diagnosis of Human Illness: Confirmation of *B. cereus* as the etiologic agent in a foodborne outbreak requires either (1) isolation of strains of the same serotype from the suspect food and feces or vomitus of the patient, (2) isolation of large numbers of a *B. cereus* serotype known to cause foodborne illness from the suspect food or from the feces or vomitus of the patient, or (3) isolation of *B. cereus* from suspect foods and determining their enterotoxigenicity by serological (diarrheal toxin) or biological (diarrheal and emetic) tests. The rapid onset time to symptoms in the emetic form of disease, coupled with some food evidence, is often sufficient to diagnose this type of food poisoning.

5. Foods Incriminated: A wide variety of foods including meats, milk, vegetables, and fish have been associated with the diarrheal type food poisoning. The vomiting-type outbreaks have generally been associated with rice products; however, other starchy foods such as potato, pasta and cheese products have also been implicated. Food mixtures such as sauces, puddings, soups, casseroles, pastries, and salads have frequently been incriminated in food poisoning outbreaks.

6. Relative Frequency of Illness: In 1980, 9 outbreaks were reported to the Centers for Disease Control and included such foods as beef, turkey, and Mexican foods. In 1981, 8 outbreaks were reported which primarily involved rice and shellfish. Other outbreaks go unreported or are misdiagnosed because of symptomatic similarities to *Staphylococcus aureus* intoxication (*B. cereus* vomiting-type) or *C. perfringens* food poisoning (B. cereus diarrheal type).

7. Complications: Although no specific complications have been associated with the diarrheal and vomiting toxins produced by *B. cereus*, other clinical manifestations of *B. cereus* invasion or contamination have been observed. They include bovine mastitis, severe systemic and pyogenic infections, gangrene, septic meningitis, cellulitis, panophthalmitis, lung abscesses, infant death, and endocarditis.

8. Target Populations: All people are believed to be susceptible to *B. cereus* food poisoning.

9. Food Analysis: A variety of methods have been recommended for the recovery, enumeration and confirmation of *B. cereus* in foods. More recently, a serological method has been developed for detecting the putative enterotoxin of *B. cereus* (diarrheal type) isolates from suspect foods. Recent investigations suggest that the vomiting type toxin can be detected by animal models (cats, monkeys) or possibly by cell culture.

10. Selected Outbreaks: On September 22, 1985, the Maine Bureau of Health was notified of gastrointestinal illness among patrons of a Japanese restaurant. Because the customers were exhibiting symptoms of illness while still on the restaurant premises, and because uncertainty existed as to the etiology of the problem, the local health department, in concurrence with the restaurant owner, closed the restaurant at 7:30 p.m. that same day.

Eleven (31%) of the approximately 36 patrons reportedly served on the evening of September 22, were contacted in an effort to determine the etiology of the outbreak. Those 11 comprised the last three dining parties served on September 22. Despite extensive publicity, no additional cases were reported.

A case was defined as anyone who demonstrated vomiting or diarrhea within 6 hours of dining at the restaurant. All 11 individuals were interviewed for symptoms, time of onset of illness, illness duration, and foods ingested. All 11 reported nausea and vomiting; nine reported diarrhea; one reported headache; and one reported abdominal cramps. Onset of illness ranged from 30 minutes to 5 hours (mean 1 hour, 23 minutes) after eating at the restaurant. Duration of illness ranged from 5 hours to several days, except for two individuals still symptomatic with diarrhea 2 weeks after dining at the restaurant. Ten persons sought medical treatment at local emergency rooms on September 22; two ultimately required hospitalization for rehydration.

Analysis of the association of specific foods with illness was not instructive, since all persons consumed the same food items; chicken soup, fried shrimp, stir-fried rice, fried zucchini, onions, bean sprouts, cucumber, cabbage, and lettuce salad, ginger salad dressing, hibachi chicken and steak, and tea. Five persons ordered hibachi scallops, and one person ordered hibachi swordfish. However, most individuals sampled each other's entrees. One vomitus specimen and two stool specimens from the three separate individuals yielded an overgrowth of *B. cereus*, although an accurate bacterial count could not be made because an inadequate amount of the steak remained for laboratory analysis. No growth of *B. cereus* was reported from the fried rice, mixed fried vegetables, or hibachi chicken.

According to the owner, all meat was delivered 2-3 times a week from a local meat supplier and refrigerated until ordered by restaurant patrons. Appropriate-sized portions for a dining group were taken from the kitchen to the dining area and diced or sliced, then sauteed at the table directly in front of restaurant patrons. The meat was seasoned with soy sauce salt and white pepper, open containers of which had been used for at least 2 months by the restaurant. The hibachi steak was served immediately after cooking.

The fried rice served with the meal was customarily made from leftover boiled rice. It could not be established whether the boiled rice had been stored refrigerated or at room temperature.

Fresh, rapidly cooked meat, eaten immediately, seems an unlikely vehicle of *B. cereus* food poisoning. The laboratory finding of *B. cereus* in a foodstuff without quantitative cultures and without accompanying epidemiologic data is insufficient to establish its role in the outbreak. Although no viable *B. cereus* organisms were isolated from the fried rice eaten with the meal, it does not exclude this food as the common vehicle. Reheating during preparation may have eliminated the bacteria in the food without decreasing the activity of the heat-stable toxin. While the question of the specific vehicle remains incompletely resolved, the clinical and laboratory findings substantially support *B. cereus* as the cause of the outbreak.

Most episodes of food poisoning undoubtedly go unreported, and in most of those reported, the specific pathogens are never identified. Alert recognition of the clinical syndrome and appropriate laboratory work permitted identification of the role of *B. cereus* in this outbreak.

For a report on a *B. cereus* outbreak in northern Virginia see this MMWR 43(10):1994 Mar 18.

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

CDC/MMWR

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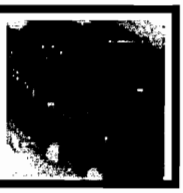

Shigella spp.

CDC/MMWR | NIH/PubMed

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1. Name of the Organism: *Shigella* spp. (*Shigella sonnei*, *S. boydii*, *S. flexneri*, and *S. dysenteriae*)

Shigella are Gram-negative, nonmotile, nonsporeforming rod-shaped bacteria. The illness caused by *Shigella* (shigellosis) accounts for less than 10% of the reported outbreaks of foodborne illness in this country. *Shigella* rarely occurs in animals; principally a disease of humans except other primates such as monkeys and chimpanzees. The organism is frequently found in water polluted with human feces.

2. Name of Disease:

Shigellosis (bacillary dysentery).

3. Nature of Disease:

Symptoms -- Abdominal pain; cramps; diarrhea; fever; vomiting; blood, pus, or mucus in stools; tenesmus.

Onset time -- 12 to 50 hours.

Infective dose -- As few as 10 cells depending on age and condition of host. The *Shigella spp.* are highly infectious agents that are transmitted by the fecal-oral route.

The disease is caused when virulent *Shigella* organisms attach to, and penetrate, epithelial cells of the intestinal mucosa. After invasion, they multiply intracellularly, and spread to contiguous epitheleal cells resulting in tissue destruction. Some strains produce enterotoxin and Shiga toxin (very much like the verotoxin of *E. coli* O157:H7).

4. Diagnosis of Human Illness:

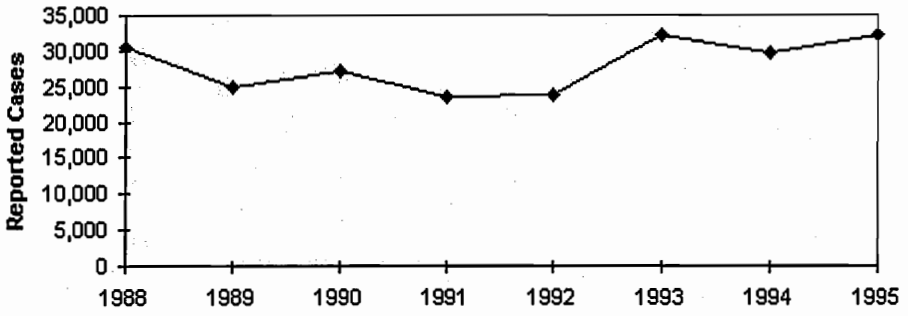
Serological identification of culture isolated from stool.

5. Associated Foods:

Salads (potato, tuna, shrimp, macaroni, and chicken), raw vegetables, milk and dairy products, and poultry. Contamination of these foods is usually through the fecal-oral route. Fecally contaminated water and unsanitary handling by food handlers are the most common causes of contamination.

6. Relative Frequency of Disease: An estimated 300,000 cases of shigellosis occur annually in the U.S. The number attributable to food is unknown, but given the low infectious dose, it is probably substantial.

Reported cases of Shigellosis, United States 1988-1995



Summary of Notifiable Diseases, United States MMWR 44(53): 1996 October 25

7. Complications: Infections are associated with mucosal ulceration, rectal bleeding, drastic dehydration; fatality may be as high as 10-15% with some strains. Reiter's disease, reactive arthritis, and hemolytic uremic syndrome are possible sequelae that have been reported in the aftermath of shigellosis.

8. Target Populations: Infants, the elderly, and the infirm are susceptible to the severest symptoms of disease, but all humans are susceptible to some degree. Shigellosis is a very common malady suffered by individuals with acquired immune deficiency syndrome (AIDS) and AIDS-related complex, as well as non-AIDS homosexual men.

9. Food Analysis: Organisms are difficult to demonstrate in foods because methods are not developed or are insensitive. A genetic probe to the virulence plasmid has been developed by FDA and is currently under field test. However, the isolation procedures are still poor.

10. Selected Outbreaks: In 1985, a huge outbreak of foodborne shigellosis occurred in Midland-Odessa, Texas, involving perhaps as many as 5,000 persons. The implicated food was chopped, bagged lettuce, prepared in a central location for a Mexican restaurant chain. FDA research subsequently showed that *S. sonnei*, the isolate from the lettuce, could survive in chopped lettuce under refrigeration, and the lettuce remained fresh and appeared to be quite edible.

In 1985-1986, several outbreaks of shigellosis occurred on college campuses, usually associated with fresh vegetables from the salad bar. Usually an ill food service worker was shown to be the cause.

In 1987, several very large outbreaks of shigellosis (*S. sonnei*) occurred involving thousands of persons, but no specific food vector could be proven.

In 1988, numerous individuals contracted shigellosis from food consumed aboard Northwest Airlines flights; food on these flights had been prepared in one central commisary. No specific food item was implicated, but various sandwiches were suspected.

**NOTE - Although all *Shigella* spp. have been implicated in foodborne

outbreaks at some time, *S. sonnei* is clearly the leading cause of shigellosis from food. The other species are more closely associated with contaminated water. One in particular, *S. flexneri*, is now thought to be in large part sexually transmitted.

For information on the outbreak of *Shigella* on a cruise ship, see MMWR 43 (35):1994 Sep 09

MMWR 40(25):1991 Jun 28 reports on a *Shigella dysenteriae* Type 1 outbreak in Guatemala, 1991.

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

CDC/MMWR

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Streptococcus spp.

CDC/MMWR NIH/PubMed

1. Name of the Organism: *Streptococcus* spp.	The genus Streptococcus is comprised of Gram-positive, microaerophilic cocci (round), which are not motile and occur in chains or pairs. The genus is defined by a combination of antigenic, hemolytic, and physiological characteristics into Groups A, B, C, D, F, and G. Groups A and D can be transmitted to humans via food. Group A: one species with 40 antigenic types (*S. pyogenes*). Group D: five species (*S. faecalis*, *S. faecium*, *S. durans*, *S. avium*, and *S. bovis*).
2. Name of Acute Disease:	Group A: Cause septic sore throat and scarlet fever as well as other pyogenic and septicemic infections. Group D: May produce a clinical syndrome similar to staphylococcal intoxication.
3. Nature of Illness/Disease:	Group A: Sore and red throat, pain on swallowing, tonsilitis, high fever, headache, nausea, vomiting, malaise, rhinorrhea; occasionally a rash occurs, onset 1-3 days; the infectious dose is probably quite low (less than 1,000 organisms). Group D: Diarrhea, abdominal cramps, nausea, vomiting, fever, chills, dizziness in 2-36 hours. Following ingestion of suspect food, the infectious dose is probably high (greater than 107 organisms).
4. Diagnosis of Human Disease:	Group A: Culturing of nasal and throat swabs, pus, sputum, blood, suspect food, environmental samples. Group D: Culturing of stool samples, blood, and suspect food.
5. Associated Foods:	Group A: Food sources include milk, ice cream, eggs, steamed lobster, ground ham, potato salad, egg salad, custard, rice pudding, and shrimp salad. In almost all cases, the foodstuffs were allowed to stand at room temperature for several hours between preparation and consumption. Entrance into the food is the result of poor hygiene, ill food handlers, or the use of unpasteurized milk. Group D: Food sources include sausage, evaporated milk, cheese, meat croquettes, meat pie, pudding, raw milk, and pasteurized milk. Entrance into the food chain is due to underprocessing and/or poor and unsanitary food preparation.

6. Relative Frequency of Infection:	Group A infections are low and may occur in any season, whereas Group D infections are variable.
7. Usual Course of Disease and Complications:	Group A: Streptococcal sore throat is very common, especially in children. Usually it is successfully treated with antibiotics. Complications are rare and the fatality rate is low. Group D: Diarrheal illness is poorly characterized, but is acute and self-limiting.
8. Target Population:	All individuals are susceptible. No age or race susceptibilities have been found.
9. Analysis of Foods:	Suspect food is examined microbiologically by selective enumeration techniques which can take up to 7 days. Group specificities are determined by Lancefield group-specific antisera.
10. Selected Outbreaks:	Group A: Outbreaks of septic sore throat and scarlet fever were numerous before the advent of milk pasteurization. Salad bars have been suggested as possible sources of infection. Most current outbreaks have involved complex foods (i.e., salads) which were infected by a food handler with septic sore throat. One ill food handler may subsequently infect hundreds of individuals. Group D: Outbreaks are not common and are usually the result of preparing, storing, or handling food in an unsanitary manner. **For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.**

CDC/MMWR

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U.S. Food & Drug Administration
Center for Food Safety & Applied Nutrition
Foodborne Pathogenic Microorganisms and Natural Toxins Handbook

Enterotoxigenic *Escherichia coli*

CDC/MMWR NIH/PubMed

1. Name of the Organism: Enterotoxigenic *Escherichia coli* (ETEC)	Currently, there are four recognized classes of enterovirulent *E. coli* (collectively referred to as the EEC group) that cause gastroenteritis in humans. Among these are the enterotoxigenic (ETEC) strains. They comprise a relatively small proportion of the species and have been etiologically associated with diarrheal illness of all age groups from diverse global locations. The organism frequently causes diarrhea in infants in less developed countries and in visitors there from industrialized countries. The etiology of this cholera-like illness has been recognized for about 20 years.
2. Name of Acute Disease:	Gastroenteritis is the common name of the illness caused by ETEC, although travelers' diarrhea is a frequent sobriquet.
3. Nature of Disease:	The most frequent clinical syndrome of infection includes watery diarrhea, abdominal cramps, low-grade fever, nausea and malaise. Infective dose--Volunteer feeding studies indicate that a relatively large dose (100 million to 10 billion bacteria) of enterotoxigenic *E. coli* is probably necessary to establish colonization of the small intestine, where these organisms proliferate and produce toxins which induce fluid secretion. With high infective dose, diarrhea can be induced within 24 hours. Infants may require fewer organisms for infection to be established.
4. Diagnosis of Human Illness:	During the acute phase of infection, large numbers of enterotoxigenic cells are excreted in feces. These strains are differentiated from nontoxigenic *E. coli* present in the bowel by a variety of in vitro immunochemical, tissue culture, or gene probe tests designed to detect either the toxins or genes that encode for these toxins. The diagnosis can be completed in about 3 days.
5. Associated Foods:	ETEC is not considered a serious foodborne disease hazard in countries having high sanitary standards and practices. Contamination of water with human sewage may lead to contamination of foods. Infected food handlers may also contaminate foods. These organisms are infrequently isolated from dairy products such as semi-soft cheeses.

6. Relative Frequency of Disease: Only four outbreaks in the U.S. have been documented, one resulting from consumption of water contaminated with human sewage, another from consumption of Mexican food prepared by an infected food handler. In two others, one in a hospital cafeteria and one aboard a cruise ship, food was the probable cause. The disease among travelers to foreign countries, however, is common.

7. Complications: The disease is usually self-limiting. In infants or debilitated elderly persons, appropriate electrolyte replacement therapy may be necessary.

8. Target Populations: Infants and travelers to underdeveloped countries are most at-risk of infection.

9. Analysis of Food: With the availability of a gene probe method, foods can be analyzed directly for the presence of enterotoxigenic *E. coli*, and the analysis can be completed in about 3 days. Alternative methods which involve enrichment and plating of samples for isolation of *E. coli* and their subsequent confirmation as toxigenic strains by conventional toxin assays may take at least 7 days.

10. Selected Outbreaks: In the last decade, four major common-source outbreaks of ETEC gastroenteritis occurred in the U.S. In late 1975 one-third of the passengers on two successive cruises of a Miami-based ship experienced diarrheal illness. A CDC investigation found ETEC to be the cause, presumably linked to consumption of crabmeat cocktail. In early 1980, 415 persons eating at a Mexican restaurant experienced diarrhea. The source of the causative organism was an ill food handler. In 1981, 282 of 3,000 personnel at a Texas hospital acquired ETEC gastroenteritis after eating in the hospital cafeteria. No single food was identified by CDC.

Outbreaks of ETEC in Rhode Island and New Hampshire are reported in this MMWR 43(5):1994 Feb 11.

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

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Enteropathogenic *Escherichia coli*

NIH/PubMed

1. Name of the Organism: Enteropathogenic *Escherichia coli* (EPEC)	Currently, there are four recognized classes of enterovirulent *E. coli* (collectively referred to as the EEC group) that cause gastroenteritis in humans. Among these are the enteropathogenic (EPEC) strains. EPEC are defined as *E. coli* belonging to serogroups epidemiologically implicated as pathogens but whose virulence mechanism is unrelated to the excretion of typical *E. coli* enterotoxins. *E. coli* are Gram-negative, rod-shaped bacteria belonging the family Enterobacteriaceae. Source(s) and prevalence of EPEC are controversial because foodborne outbreaks are sporadic. Humans, bovines, and swine can be infected, and the latter often serve as common experimental animal models. *E. coli* are present in the normal gut flora of these mammals. The proportion of pathogenic to nonpathogenic strains, although the subject of intense research, is unknown.
2. Name of Acute Disease:	Infantile diarrhea is the name of the disease usually associated with EPEC.
3. Nature of Disease:	EPEC cause either a watery or bloody diarrhea, the former associated with the attachment to, and physical alteration of, the integrity of the intestine. Bloody diarrhea is associated with attachment and an acute tissue-destructive process, perhaps caused by a toxin similar to that of *Shigella dysenteriae*, also called verotoxin. In most of these strains the shiga-like toxin is cell-associated rather than excreted. Infective dose -- EPEC are highly infectious for infants and the dose is presumably very low. In the few documented cases of adult diseases, the dose is presumably similar to other colonizers (greater than 10^6 total dose).
4. Diagnosis of Human Illness:	The distinction of EPEC from other groups of pathogenic *E. coli* isolated from patients' stools involves serological and cell culture assays. Serotyping, although useful, is not strict for EPEC.
5. Associated Foods:	Common foods implicated in EPEC outbreaks are raw beef and chicken, although any food exposed to fecal contamination is strongly suspect.
6. Relative Frequency of Disease:	Outbreaks of EPEC are sporadic. Incidence varies on a worldwide basis; countries with poor sanitation practices have the most frequent outbreaks.

7. Usual Course of Disease and Some Complications:	Occasionally, diarrhea in infants is prolonged, leading to dehydration, electrolyte imbalance and death (50% mortality rates have been reported in third world countries).
8. Target Populations:	EPEC outbreaks most often affect infants, especially those that are bottle fed, suggesting that contaminated water is often used to rehydrate infant formulae in underdeveloped countries.
9. Analysis of Foods:	The isolation and identification of *E. coli* in foods follows standard enrichment and biochemical procedures. Serotyping of isolates to distinguish EPEC is laborious and requires high quality, specific antisera, and technical expertise. The total analysis may require from 7 to 14 days.
10. Selected Outbreaks:	Sporadic outbreaks of EPEC diarrhea have occurred for half a century in infant nurseries, presumably derived from the hospital environment or contaminated infant formula. Common-source outbreaks of EPEC diarrhea involving healthy young adults were reported in the late 1960s. Presumably a large inoculum was ingested. **For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.**

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Escherichia coli O157:H7

Education | CDC/MMWR | NIH/PubMed

1. Name of the Organism:
Escherichia coli O157:H7 (enterohemorrhagic *E. coli* or EHEC)

Currently, there are four recognized classes of enterovirulent *E. coli* (collectively referred to as the EEC group) that cause gastroenteritis in humans. Among these is the enterohemorrhagic (EHEC) strain designatec *E. coli* O157:H7. *E. coli* is a normal inhabitant of the intestines of all animals, including humans. When aerobic culture methods are used, *E. cc* is the dominant species found in feces. Normally *E. coli* serves a useful function in the body by suppressing the growth of harmful bacterial speci and by synthesizing appreciable amounts of vitamins. A minority of *E. co* strains are capable of causing human illness by several different mechanisms. *E. coli* serotype O157:H7 is a rare variety of *E. coli* that produces large quantities of one or more related, potent toxins that cause severe damage to the lining of the intestine. These toxins [verotoxin (VT) shiga-like toxin] are closely related or identical to the toxin produced by *Shigella dysenteriae*.

2. Name of Acute Disease:

Hemorrhagic colitis is the name of the acute disease caused by *E. coli* O157:H7.

3. Nature of Disease:

The illness is characterized by severe cramping (abdominal pain) and diarrhea which is initially watery but becomes grossly bloody. Occasional vomiting occurs. Fever is either low-grade or absent. The illness is usuall self-limited and lasts for an average of 8 days. Some individuals exhibit watery diarrhea only.

Infective dose -- Unknown, but from a compilation of outbreak data, including the organism's ability to be passed person-to-person in the day-care setting and nursing homes, the dose may be similar to that of *Shigell* spp. (10 organisms).

4. Diagnosis of Human Illness:

Hemorrhagic colitis is diagnosed by isolation of *E. coli* of serotype O157:H7 or other verotoxin-producing *E. coli* from diarrheal stools. Alternatively, the stools can be tested directly for the presence of verotox Confirmation can be obtained by isolation of *E. coli* of the same serotype from the incriminated food.

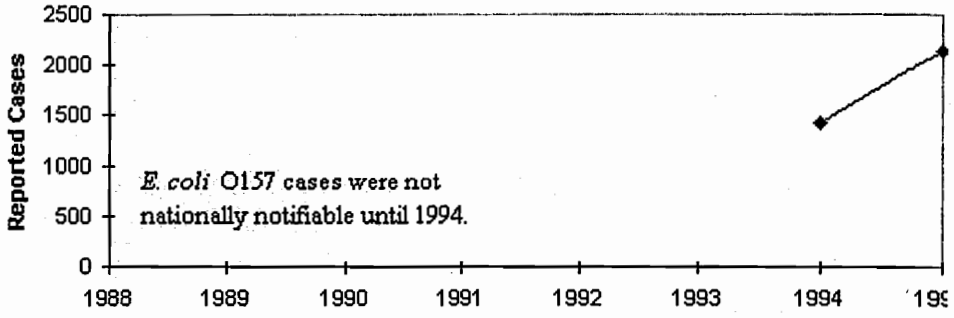
5. Associated Foods:

Undercooked or raw hamburger (ground beef) has been implicated in nea all documented outbreaks and in other sporadic cases. Raw milk was the vehicle in a school outbreak in Canada. These are the only two demonstrated food causes of disease, but other meats may contain *E. coli* O157:H7.

6. Relative Frequency of Disease:

Hemorrhagic colitis infections are not too common, but this is probably n reflective of the true frequency. In the Pacific Northwest, *E. coli* O157:H7 is thought to be second only to Salmonella as a cause of bacterial diarrhea Because of the unmistakable symptoms of profuse, visible blood in severe cases, those victims probably seek medical attention, but less severe cases are probably more numerous.

Reported Cases of *E. coli* O157, United States 1994-1995



Summary of Notifiable Diseases, United States MMWR 44(53): 1996 October 25

7. Usual Course of Disease and Some Complications:

Some victims, particularly the very young, have developed the hemolytic uremic syndrome (HUS), characterized by renal failure and hemolytic anemia. From 0 to 15% of hemorrhagic colitis victims may develop HUS. The disease can lead to permanent loss of kidney function.

In the elderly, HUS, plus two other symptoms, fever and neurologic symptoms, constitutes thrombotic thrombocytopenic purpura (TTP). This illness can have a mortality rate in the elderly as high as 50%.

8. Target Populations:

All people are believed to be susceptible to hemorrhagic colitis, but larger outbreaks have occurred in institutional settings.

9. Analysis of Foods:

E. coli 0157:H7 will form colonies on agar media that are selective for *E. coli*. However, the high temperature growth procedure normally performe to eliminate background organisms before plating cannot be used because of the inability of these organisms to grow at temperatures of 44.0 - 45.5° that support the growth of most *E. coli*. The use of DNA probes to detect genes encoding for the production of verotoxins (VT1 and VT2) is the mc sensitive method devised.

10. Selected Outbreaks:

Three outbreaks occurred in 1982. Two of them, one in Michigan and one in Oregon, involved hamburgers from a national fast-food chain. The thir occurred in a home for the aged in Ottawa, Ontario; club sandwiches were implicated, and 19 people died. More recently, several outbreaks in nursir homes and a day-care center have been investigated. Two large outbreaks occurred in 1984, one in 1985, three in 1986. Larger outbreaks have occurred in the Northwest U.S. and Canada.

In October-November, 1986, an outbreak of hemorrhagic colitis caused b *E. coli* O157:H7 occurred in Walla Walla, WA. Thirty-seven people, age 11 months to 78 years developed diarrhea caused by the organism. All isolates from patients (14) had a unique plasmid profile and produced Shiga-like toxin II. In addition to diarrhea, 36 persons reported grossly

bloody stools and 36 of the 37 reported abdominal cramps. Seventeen patients were hospitalized. One patient developed HUS (4 years old) and three developed TTP (70, 78, and 78 years old). Two patients with TTP died. Ground beef was the implicated food vehicle.

An excellent summary of nine *E. coli* O157:H7 outbreaks appeared in the Annals of Internal Medicine, 1 November, 1988, pp. 705-712.

There was a recall of frozen hamburger underway (12 Aug 1997). For mo information, see the USDA announcement and follow-up announcement (Aug 1997) on the U.S. Department of Agriculture web site concerning the recall of Hudson frozen ground beef.

The Centers for Disease Control and Prevention have reported on the abo outbreak in preliminary (MMWR 45(44):975, 1996 November 8) and in updated (MMWR 46(1):4-8, 1997 January 10) form.

The FDA has issued on 31 October 1996 a press release concerning an outbreak of *E. coli* O157:H7 associated with Odwalla brand apple juice products.

A non-food related outbreak of *E. coli* O157:H7 is reported in MMWR 4 (21):1996 May 31. While, the source of the outbreak is thought to be waterborne, the article is linked to this chapter to provide updated referen information on enterohemorrhagic *E. coli*.

MMWR 45(12):1996 Mar 29 reports on an outbreak of O157:H7 that occured in Georgia and Tennessee in June of 1995.

A community outbreak of hemolytic uremic syndrome attributable to *Escherichia coli* O111:NM in southern Australia in 1995 is reported in MMWR 44(29):1995 Jul 28.

A report on enhanced detection of sporadic *E. coli* O157:H7 infections in New Jersey and on an *E. coli* O157:H7 outbreak at a summer camp are in MMWR 44(22): 1995 Jun 9.

An outbreak of *E. coli* O157:H7 in Washington and California associated with dry-cured salami is reported in MMWR 44(9):1995 Mar 10.

Information concerning an outbreak that occured because of home-cooke hamburger can be found in this MMWR 43(12):1994 Apr 01.

MMWR 43(10):1994 Mar 18 reports on laboratory screening for *E. coli* O157 in Connecticut.

The outbreak of EHEC in the western states of the US is reported in preliminary form in this MMWR 42(4):1993 Feb 5, and in updated form i this MMWR 42(14):1993 Apr 16.

An outbreak of *E. coli* O157 in 1990 in North Dakota is reported in the MMWR 40(16):1991 Apr 26.

The Centers for Disease Control and Prevention has reissued the 5 November 1982 MMWR report that was the first to describe the diarrheal illness of *E. coli* O157:H7. This reissue is a part of the commemoration of CDC's 50th anniversary.

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

11. Education: USDA Urges Consumers To Use Food Thermometer When Cooking Ground Beef Patties (Aug 11 1998)

The CDC has an information brochure on preventing *Escherichia coli* O157:H7 infections.

12. Other Resources: Dr. Feng of FDA/CFSAN has written a monograph on *E. coli* O157:H7 which appeared in the CDC journal Emerging Infectious Diseases Vol. 1 No. 2, April-June 1995.

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Giardia lamblia

CDC/MMWR NIH/PubMed

1. Name of the organism:
Giardia lamblia

Giardia lamblia (intestinalis) is a single celled animal, i.e., a protozoa, that moves with the aid of five flagella. In Europe, it is sometimes referred to as Lamblia intestinalis.

2. Disease Name:

Giardiasis is the most frequent cause of non-bacterial diarrhea in North America.

3. Nature of the disease:

Organisms that appear identical to those that cause human illness have been isolated from domestic animals (dogs and cats) and wild animals (beavers and bears). A related but morphologically distinct organism infects rodents, although rodents may be infected with human isolates in the laboratory. Human giardiasis may involve diarrhea within 1 week of ingestion of the cyst, which is the environmental survival form and infective stage of the organism. Normally illness lasts for 1 to 2 weeks, but there are cases of chronic infections lasting months to years. Chronic cases, both those with defined immune deficiencies and those without, are difficult to treat. The disease mechanism is unknown, with some investigators reporting that the organism produces a toxin while others are unable to confirm its existence. The organism has been demonstrated inside host cells in the duodenum, but most investigators think this is such an infrequent occurrence that it is not responsible for disease symptoms. Mechanical obstruction of the absorptive surface of the intestine has been proposed as a possible pathogenic mechanism, as has a synergistic relationship with some of the intestinal flora. Giardia can be excysted, cultured and encysted in vitro; new isolates have bacterial, fungal, and viral symbionts. Classically the disease was diagnosed by demonstration of the organism in stained fecal smears. Several strains of *G. lamblia* have been isolated and described through analysis of their proteins and DNA; type of strain, however, is not consistently associated with disease severity. Different individuals show various degrees of symptoms when infected with the same strain, and the symptoms of an individual may vary during the course of the disease.

Infectious Dose - Ingestion of one or more cysts may cause disease, as contrasted to most bacterial illnesses where hundreds to thousands of organisms must be consumed to produce illness.

4. Diagnosis of Human Illness: *Giardia lamblia* is frequently diagnosed by visualizing the organism, either the trophozoite (active reproducing form) or the cyst (the resting stage that is resistant to adverse environmental conditions) in stained preparations or unstained wet mounts with the aid of a microscope. A commercial fluorescent antibody kit is available to stain the organism. Organisms may be concentrated by sedimentation or flotation; however, these procedures reduce the number of recognizable organisms in the sample. An enzyme linked immunosorbant assay (ELISA) that detects excretory secretory products of the organism is also available. So far, the increased sensitivity of indirect serological detection has not been consistently demonstrated.

5. Associated Foods: Giardiasis is most frequently associated with the consumption of contaminated water. Five outbreaks have been traced to food contamination by infected or infested food handlers, and the possibility of infections from contaminated vegetables that are eaten raw cannot be excluded. Cool moist conditions favor the survival of the organism.

6. Relative Frequency of Disease: Giardiasis is more prevalent in children than in adults, possibly because many individuals seem to have a lasting immunity after infection. This organism is implicated in 25% of the cases of gastrointestinal disease and may be present asymptomatically. The overall incidence of infection in the United States is estimated at 2% of the population. This disease afflicts many homosexual men, both HIV-positive and HIV-negative individuals. This is presumed to be due to sexual transmission. The disease is also common in child day care centers, especially those in which diapering is done.

7. Complications: About 40% of those who are diagnosed with giardiasis demonstrate disaccharide intolerance during detectable infection and up to 6 months after the infection can no longer be detected. Lactose (i.e., milk sugar) intolerance is most frequently observed. Some individuals (less than 4%) remain symptomatic more than 2 weeks; chronic infections lead to a malabsorption syndrome and severe weight loss. Chronic cases of giardiasis in immunodeficient and normal individuals are frequently refractile to drug treatment. Flagyl is normally quite effective in terminating infections. In some immune deficient individuals, giardiasis may contribute to a shortening of the life span.

8. Target Populations: Giardiasis occurs throughout the population, although the prevalence is higher in children than adults. Chronic symptomatic giardiasis is more common in adults than children.

9. Food Analysis: Food is analyzed by thorough surface cleaning of the suspected food and sedimentation of the organisms from the cleaning water. Feeding to specific pathogen-free animals has been used to detect the organism in large outbreaks associated with municipal water systems. The precise sensitivity of these methods has not been determined, so that negative results are questionable. Seven days may be required to detect an experimental infection.

10. Selected outbreaks:	Major outbreaks are associated with contaminated water systems that do not use sand filtration or have a defect in the filtration system. The largest reported foodborne outbreak involved 24 of 36 persons who consumed macaroni salad at a picnic. **For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.**
11. FDA Regulations or Activity:	FDA is actively developing and improving methods of recovering parasitic protozoa and helminth eggs from foods. Current recovery methods are published in the FDA's Bacteriological Analytical Manual.

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Entamoeba histolytica

CDC/MMWR NIH/PubMed

1. Name of the Organism:
Entamoeba histolytica

This is a single celled parasitic animal, i.e., a protozoa, that infects predominantly humans and other primates. Diverse mammals such as dogs and cats can become infected but usually do not shed cysts (the environmental survival form of the organism) with their feces, thus do not contribute significantly to transmission. The active (trophozoite) stage exists only in the host and in fresh feces; cysts survive outside the host in water and soils and on foods, especially under moist conditions on the latter. When swallowed they cause infections by excysting (to the trophozoite stage) in the digestive tract.

2. Name of Acute Disease:

Amebiasis (or amoebiasis) is the name of the infection caused by *E. histolytica*.

3. Nature of the Acute Disease:

Infections that sometimes last for years may be accompanied by 1) no symptoms, 2) vague gastrointestinal distress, 3) dysentery (with blood and mucus). Most infections occur in the digestive tract but other tissues may be invaded. Complications include 4) ulcerative and abscess pain and, rarely, 5) intestinal blockage. Onset time is highly variable. It is theorized that the absence of symptoms or their intensity varies with such factors as 1) strain of amoeba, 2) immune health of the host, and 3) associated bacteria and, perhaps, viruses. The amoeba's enzymes help it to penetrate and digest human tissues; it secretes toxic substances.

Infectious Dose--Theoretically, the ingestion of one viable cyst can cause an infection.

4. Diagnosis of Human Illness:

Human cases are diagnosed by finding cysts shed with the stool; various flotation or sedimentation procedures have been developed to recover the cysts from fecal matter; stains (including fluorescent antibody) help to visualize the isolated cysts for microscopic examination. Since cysts are not shed constantly, a minimum of 3 stools should be examined. In heavy infections, the motile form (the trophozoite) can be seen in fresh feces. Serological tests exist for long-term infections. It is important to distinguish the *E. histolytica* cyst from the cysts of nonpathogenic intestinal protozoa by its appearance.

5. Transmission:

Amebiasis is transmitted by fecal contamination of drinking water and foods, but also by direct contact with dirty hands or objects as well as by sexual contact.

6. Frequency of Infections:	The infection is "not uncommon" in the tropics and arctics, but also in crowded situations of poor hygiene in temperate-zone urban environments. It is also frequently diagnosed among homosexual men.
7. Usual Course of the Disease and Some Complications:	In the majority of cases, amoebas remain in the gastrointestinal tract of the hosts. Severe ulceration of the gastrointestinal mucosal surfaces occurs in less than 16% of cases. In fewer cases, the parasite invades the soft tissues, most commonly the liver. Only rarely are masses formed (amoebomas) that lead to intestinal obstruction. Fatalities are infrequent.
8. Target Populations:	All people are believed to be susceptible to infection, but individuals with a damaged or undeveloped immunity may suffer more severe forms of the disease. AIDS/ ARC patients are very vulnerable.
9. Analysis of Foods:	*E. histolytica* cysts may be recovered from contaminated food by methods similar to those used for recovering *Giardia lamblia* cysts from feces. Filtration is probably the most practical method for recovery from drinking water and liquid foods. *E. histolytica* cysts must be distinguished from cysts of other parasitic (but nonpathogenic) protozoa and from cysts of free-living protozoa. Recovery procedures are not very accurate; cysts are easily lost or damaged beyond recognition, which leads to many falsely negative results in recovery tests. (See the FDA Bacteriological Analytical Manual.)
10. Selected Outbreaks:	The most dramatic incident in the USA was the Chicago World's Fair outbreak in 1933 caused by contaminated drinking water; defective plumbing permitted sewage to contaminate the drinking water. There were 1,000 cases (with 58 deaths). In recent times, food handlers are suspected of causing many scattered infections, but there has been no single large outbreak. **For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.**

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Cryptosporidium parvum

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1. Name of the organism: *Cryptosporidium parvum*

Cryptosporidium parvum, a single-celled animal, i.e., a protozoa, is an obligate intracellular parasite. It has been given additional species names when isolated from different hosts. It is currently thought that the form infecting humans is the same species that causes disease in young calves. The forms that infect avian hosts and those that infect mice are not thought capable of infecting humans. *Cryptosporidium* sp. infects many herd animals (cows, goats, sheep among domesticated animals, and deer and elk among wild animals). The infective stage of the organism, the oocyst is 3 um in diameter or about half the size of a red blood cell. The sporocysts are resistant to most chemical disinfectants, but are susceptible to drying and the ultraviolet portion of sunlight. Some strains appear to be adapted to certain hosts but cross-strain infectivity occurs and may or may not be associated with illness. The species or strain infecting the respiratory system is not currently distinguished from the form infecting the intestines.

2. Disease Name:

Intestinal, tracheal, or pulmonary cryptosporidiosis.

3. Nature of Acute Disease:

Intestinal cryptosporidiosis is characterized by severe watery diarrhea but may, alternatively, be asymptomatic. Pulmonary and tracheal cryptosporidiosis in humans is associated with coughing and frequently a low-grade fever; these symptoms are often accompanied by severe intestinal distress.

Infectious dose--Less than 10 organisms and, presumably, one organism can initiate an infection. The mechanism of disease is not known; however, the intracellular stages of the parasite can cause severe tissue alteration.

4. Diagnosis of Human Illness:

Oocysts are shed in the infected individual's feces. Sugar flotation is used to concentrate the organisms and acid fast staining is used to identify them. A commercial kit is available that uses fluorescent antibody to stain the organisms isolated from feces. Diagnosis has also been made by staining the trophozoites in intestinal and biopsy specimens. Pulmonary and tracheal cryptosporidiosis are diagnosed by biopsy and staining.

5. Food Occurence:

Cryptosporidium sp. could occur, theoretically, on any food touched by a contaminated food handler. Incidence is higher in child day care centers that serve food. Fertilizing salad vegetables with manure is another possible source of human infection. Large outbreaks are associated with contaminated water supplies.

6. Relative Frequency of the Disease:

Direct human surveys indicate a prevalence of about 2% of the population in North America. Serological surveys indicate that 80% of the population has had cryptosporidiosis. The extent of illness associated with reactive sera is not known.

7. Usual Course of the Disease and Complications:

Intestinal cryptosporidiosis is self-limiting in most healthy individuals, with watery diarrhea lasting 2-4 days. In some outbreaks at day care centers, diarrhea has lasted 1 to 4 weeks. To date, there is no known effective drug for the treatment of cryptosporidiosis. Immunodeficient individuals, especially AIDS patients, may have the disease for life, with the severe watery diarrhea contributing to death. Invasion of the pulmonary system may also be fatal.

8. Target Populations:

In animals, the young show the most severe symptoms. For the most part, pulmonary infections are confined to those who are immunodeficient. However, an infant with a presumably normal immune system had tracheal cryptosporidiosis (although a concurrent viremia may have accounted for lowered resistance). Child day care centers, with a large susceptible population, frequently report outbreaks.

9. Analysis of Foods:

The 7th edition of FDA's Bacteriological Analytical Manual will contain a method for the examination of vegetables for *Cryptosporidium* sp.

10. Selected Outbreaks:

Since 1984, cryptosporidiosis has been associated with outbreaks of diarrheal illness in child day care centers throughout the United States and Canada. During 1987 a waterborne outbreak in Georgia produced illness in an estimated 13,000 individuals, and exposure to contaminated drinking water was the major distinction between those that were ill and those that were not. This was the first report of disease transmission by a municipal water system that was in compliance with all state and federal standards for

An outbreak of cryptosporidiosis associated with the consumption of apple cider is reported in MMWR 46(1):1997 Jan 10.

MMWR 45(36):1996 Sep 13 reports on an outbreak of cryptosporidiosis associated with the consumption of home-made chicken salad in Minnesota.

A non-food outbreak of cryptosporidiosis in a day-camp is reported in MMWR 45(21):1995 May 31. This report is linked to this chapter to provide reference information.

MMWR 39(20):1990 May 25 reports on a non-food related outbreak of cryptosporidiosis, but contains useful information on *Cryptosporidium* sp.

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

11. FDA Regulations or Activity:

FDA is developing and improving methods for the recovery of cysts of parasitic protozoa from fresh vegetables. Current recovery methods are published in the Bacteriological Analytical Manual.

12. Education: The CDC has information on *Cryptosporidium*.

13. Other Resources: From GenBank there is a Loci index for genome *Cryptosporidium parvum*.

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Cyclospora cayetanensis

Education | CDC/MMWR | NIH/PubMed

1. Selected Outbreaks:

Outbreak of Cyclosporiasis -- Northern Virginia-Washington, DC-Baltimore, Maryland, Metropolitan Area, 1997 MMWR 46 (30):1997 Aug 1

MMWR 46(23):1997 June 13 Update: Outbreaks of Cyclosporiasis - United States and Canada, 1997

FDA has released a talk paper on outbreaks of cyclosporiasis and Guatemalan raspberries dated 10 June 1997.

MMWR 46(21):1997 May 30 Update: Outbreaks of Cyclosporiasis--United States, 1997

Report on an outbreak of cyclosporiasis in the United States in 1997 in MMWR 46(20):1997 May 23.

Updated Morbidity and Mortality Weekly Report on *Cyclospora cayentanensis* 19 July 1996

Morbidity and Mortality Weekly Report on *Cyclospora cayetanensis* 28 June 1996.

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

2. Education:

Information for the general public

Information for health professionals

3. Other Resources:

The FDA method *Cyclospora cayetanensis* Protocol: Concentration and Preparation of Oocysts from Produce for the Polymerase Chain Reaction (PCR) and Microscopy.

A FDA Laboratory Information Bulletin 4044 on "Differentiation of *Cyclospora* sp. and *Eimeria* spp. by Using the Polymerase Chain Reaction Amplification Products and Restriction Fragment Length Polymorphisms."

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Anisakis simplex and related worms

1. Name of the Organism: *Anisakis simplex* and related worms

Anisakis simplex (herring worm), *Pseudoterranova (Phocanema, Terranova) decipiens* (cod or seal worm), *Contracaecum* spp., and *Hysterothylacium (Thynnascaris)* spp. are anisakid nematodes (roundworms) that have been implicated in human infections caused by the consumption of raw or undercooked seafood. To date, only *A. simplex* and *P. decipiens* are reported from human cases in North America.

2. Name of Acute Disease:

Anisakiasis is generally used when referring to the acute disease in humans. Some purists utilize generic names (e.g., contracaeciasis) in referring to the disease, but the majority consider that the name derived from the family is specific enough. The range of clinical features is not dependent on species of anisakid parasite in cases reported to date.

3. Nature of the Acute Disease:

In North America, anisakiasis is most frequently diagnosed when the affected individual feels a tingling or tickling sensation in the throat and coughs up or manually extracts a nematode. In more severe cases there is acute abdominal pain, much like acute appendicitis accompanied by a nauseous feeling. Symptoms occur from as little as an hour to about 2 weeks after consumption of raw or undercooked seafood. One nematode is the usual number recovered from a patient. With their anterior ends, these larval nematodes from fish or shellfish usually burrow into the wall of the digestive tract to the level of the muscularis mucosae (occasionally they penetrate the intestinal wall completely and are found in the body cavity). They produce a substance that attracts eosinophils and other host white blood cells to the area. The infiltrating host cells form a granuloma in the tissues surrounding the penetrated worm. In the digestive tract lumen, the worm can detach and reattach to other sites on the wall. Anisakids rarely reach full maturity in humans and usually are eliminated spontaneously from the digestive tract lumen within 3 weeks of infection. Penetrated worms that die in the tissues are eventually removed by the host's phagocytic cells.

4. Diagnosis of Human Illness:

In cases where the patient vomits or coughs up the worm, the disease may be diagnosed by morphological examination of the nematode. (*Ascaris lumbricoides*, the large roundworm of humans, is a terrestrial relative of anisakines and sometimes these larvae also crawl up into the throat and nasal passages.) Other cases may require a fiber optic device that allows the attending physician to examine the inside of the stomach and the first part of the small intestine. These devices are equipped with a mechanical forceps that can be used to remove the worm. Other cases are diagnosed upon finding a granulomatous lesion with a worm on laparotomy. A specific radioallergosorbent test has been developed for anasakiasis, but is not yet commercially marketed.

5. Associated Foods:

Seafoods are the principal sources of human infections with these larval worms. The adults of *A. simplex* are found in the stomachs of whales and dolphins. Fertilized eggs from the female parasite pass out of the host with the host's feces. In seawater, the eggs embryonate, developing into larvae that hatch in sea water. These larvae are infective to copepods (minute crustaceans related to shrimp) and other small invertebrates. The larvae grow in the invertebrate and become infective for the next host, a fish or larger invertebrate host such as a squid. The larvae may penetrate through the digestive tract into the muscle of the second host. Some evidence exists that the nematode larvae move from the viscera to the flesh if the fish hosts are not gutted promptly after catching. The life cycles of all the other anisakid genera implicated in human infections are similar. These parasites are known to occur frequently in the flesh of cod, haddock, fluke, pacific salmon, herring, flounder, and monkfish.

6. Relative Frequency of the Disease:

Fewer than 10 cases are diagnosed in the U.S. annually. However, it is suspected that many other cases go undetected. The disease is transmitted by raw, undercooked or insufficiently frozen fish and shellfish, and its incidence is expected to increase with the increasing popularity of sushi and sashimi bars.

7. Usual Disease Course and Complications:

Severe cases of anisakiasis are extremely painful and require surgical intervention. Physical removal of the nematode(s) from the lesion is the only known method of reducing the pain and eliminating the cause (other than waiting for the worms to die). The symptoms apparently persist after the worm dies since some lesions are found upon surgical removal that contain only nematode remnants. Stenosis (a narrowing and stiffening) of the pyloric sphincter was reported in a case in which exploratory laparotomy had revealed a worm that was not removed.

8. Target Populations:

The target population consists of consumers of raw or underprocessed seafood.

9. Analysis of Foods:

Candling or examining fish on a light table is used by commercial processors to reduce the number of nematodes in certain white-flesh fish that are known to be infected frequently. This method is not totally effective, nor is it very adequate to remove even the majority of nematodes from fish with pigmented flesh.

10. Selected Outbreaks:

This disease is known primarily from individual cases. Japan has the greatest number of reported cases because of the large volume of raw fish consumed there.

A recent letter to the editor of the New England Journal of Medicine (319:1128-29, 1988) stated that approximately 50 cases of anisakiasis have been documented in the United States, to date. Three cases in the San Francisco Bay area involved ingestion of sushi or undercooked fish. The letter also points out that anasakiasis is easily misdiagnosed as acute appendicitis, Crohn's disease, gastric ulcer , or gastrointestinal cancer.

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

11. FDA Activity and Regulations:	FDA recommends that all fish and shellfish intended for raw (or semiraw such as marinated or partly cooked) consumption be blast frozen to -35°C (-31°F) or below for 15 hours, or be regularly frozen to -20°C (-4°F) or below for 7 days.

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Hepatitis A Virus

CDC/MMWR NIH/PubMed

1. Name of the Organism:
Hepatitis A Virus

Hepatitis A virus (HAV) is classified with the enterovirus group of the Picornaviridae family. HAV has a single molecule of RNA surrounded by a small (27 nm diameter) protein capsid and a buoyant density in CsCl of 1.33 g/ml. Many other picornaviruses cause human disease, including polioviruses, coxsackieviruses, echoviruses, and rhinoviruses (cold viruses).

2. Name of Acute Disease:

The term hepatitis A (HA) or type A viral hepatitis has replaced all previous designations: infectious hepatitis, epidemic hepatitis, epidemi jaundice, catarrhal jaundice, infectious icterus, Botkins disease, and M 1 hepatitis.

3. Nature of Disease:

Hepatitis A is usually a mild illness characterized by sudden onset of fever, malaise, nausea, anorexia, and abdominal discomfort, followed i several days by jaundice. The infectious dose is unknown but presumably is 10-100 virus particles.

4. Diagnosis of Human Illness:

Hepatitis A is diagnosed by finding IgM-class anti-HAV in serum collected during the acute or early convalescent phase of disease. Commercial kits are available.

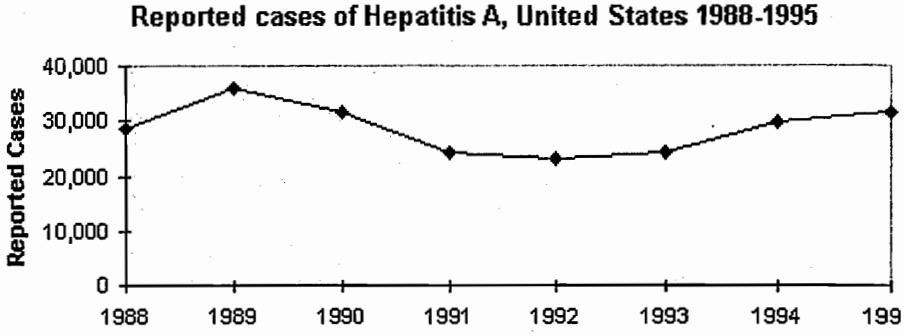
5. Associated Foods:

HAV is excreted in feces of infected people and can produce clinical disease when susceptible individuals consume contaminated water or foods. Cold cuts and sandwiches, fruits and fruit juices, milk and milk products, vegetables, salads, shellfish, and iced drinks are commonly implicated in outbreaks. Water, shellfish, and salads are the most frequent sources. Contamination of foods by infected workers in food processing plants and restaurants is common.

6. Frequency of Disease:

Hepatitis A has a worldwide distribution occurring in both epidemic ar sporadic fashions. About 22,700 cases of hepatitis A representing 38% of all hepatitis cases (5-year average from all routes of transmission) a reported annually in the U.S. In 1988 an estimated 7.3% cases were foodborne or waterborne. HAV is primarilly transmitted by person-to-person contact through fecal contamination, but common-source epidemics from contaminated food and water also occur. Poor sanitatic and crowding facilitate transmission. Outbreaks of HA are common in institutions, crowded house projects, and prisons and in military forces in adverse situations. In developing countries, the incidence of disease adults is relatively low because of exposure to the virus in childhood. Most individuals 18 and older demonstrate an immunity that provides lifelong protection against reinfection. In the U.S., the percentage of

adults with immunity increases with age (10% for those 18-19 years of age to 65% for those over 50). The increased number of susceptible individuals allows common source epidemics to evolve rapidly.



Summary of Notifiable Diseases, United States MMWR 44(53): 1996 October 25

7. Usual Course of Disease:

The incubation period for hepatitis A, which varies from 10 to 50 days (mean 30 days), is dependent upon the number of infectious particles consumed. Infection with very few particles results in longer incubatio periods. The period of communicability extends from early in the incubation period to about a week after the development of jaundice. The greatest danger of spreading the disease to others occurs during th middle of the incubation period, well before the first presentation of symptoms. Many infections with HAV do not result in clinical disease, especially in children. When disease does occur, it is usually mild and recovery is complete in 1-2 weeks. Occasionaly, the symptoms are severe and convalescence can take several months. Patients suffer fron feeling chronically tired during convalescence, and their inability to work can cause financial loss. Less than 0.4% of the reported cases in the U.S. are fatal. These rare deaths usually occur in the elderly.

8. Target Population:

All people who ingest the virus and are immunologically unprotected a susceptible to infection. Disease however, is more common in adults than in children.

9. Analysis of Foods:

The virus has not been isolated from any food associated with an outbreak. Because of the long incubation period, the suspected food is often no longer available for analysis. No satisfactory method is presently available for routine analysis of food, but sensitive molecula methods used to detect HAV in water and clinical specimens, should prove useful to detect virus in foods. Among those, the PCR amplification method seems particularly promising.

10. Selected Outbreaks:

Hepatitis A is endemic throughout much of the world. Major national epidemics occurred in 1954, 1961 and 1971. Although no major epidemic occurred in the 1980s, the incidence of hepatitis A in the U.S increased 58% from 1983 to 1989. Foods have been implicated in over 30 outbreaks since 1983. The most recent ones and the suspected contaminated foods include:

- 1987 - Louisville, Kentucky. Suspected source: imported lettuce
- 1988 - Alaska. Ice-slush beverage prepared in a local market. - North Carolina. Iced tea prepared in a restaurant. - Florida. Raw

oysters harvested from nonapproved bed.
- 1989 - Washington. Unidentified food in a restaurant chain.
- 1990 - North Georgia. Frozen strawberries. - Montana. Frozen strawberries. - Baltimore. Shellfish.

A summary of foodborne Hepatitis A outbreaks in Missouri, Wisconsi and Alaska is found in MMWR 42(27):1993 Jul 16.

MMWR 39(14):1990 Apr 13 summarizes foodborne outbreaks of Hepatitis A in Alaska, Florida, North Carolina, Washington.

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

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The Norwalk virus family

CDC/MMWR NIH/PubMed

1. Name of the Organism: The Norwalk virus family	Norwalk virus is the prototype of a family of unclassified small round structured viruses (SRSVs) which may be related to the caliciviruses. They contain a positive strand RNA genome of 7.5 kb and a single structural protein of about 60 kDa. The 27-32 nm viral particles have a buoyant density of 1.39-1.40 g/ml in CsCl. The family consists of several serologically distinct groups of viruses that have been named after the places where the outbreaks occurred. In the U.S., the Norwalk and Montgomery County agents are serologically related but distinct from the Hawaii and Snow Mountain agents. The Taunton, Moorcroft, Barnett, and Amulree agents were identified in the U.K., and the Sapporo and Otofuke agents in Japan. Their serological relationships remain to be determined.
2. Name of Acute Disease:	Common names of the illness caused by the Norwalk and Norwalk-like viruses are viral gastroenteritis, acute nonbacterial gastroenteritis, food poisoning, and food infection.
3. Nature of Disease:	The disease is self-limiting, mild, and characterized by nausea, vomiting, diarrhea, and abdominal pain. Headache and low-grade fever may occur. The infectious dose is unknown but presumed to be low.
4. Diagnosis of Human Illness:	Specific diagnosis of the disease can only be made by a few laboratories possessing reagents from human volunteer studies. Identification of the virus can be made on early stool specimens using immune electron microscopy and various immunoassays. Confirmation often requires demonstration of seroconversion, the presence of specific IgM antibody, or a four-fold rise in antibody titer to Norwalk virus on paired acute-convalescent sera.
5. Associated Foods:	Norwalk gastroenteritis is transmitted by the fecal-oral route via contaminated water and foods. Secondary person-to-person transmission has been documented. Water is the most common source of outbreaks and may include water from municipal supplies, well, recreational lakes, swiming pools, and water stored aboard cruise ships. Shellfish and salad ingredients are the foods most often implicated in Norwalk outbreaks. Ingestion of raw or insufficiently steamed clams and oysters poses a high risk for infection with Norwalk virus. Foods other than shellfish are contaminated by ill food handlers.

6. Frequency of Disease:

Only the common cold is reported more frequently than viral gastroenteritis as a cause of illness in the U.S. Although viral gastroenteritis is caused by a number of viruses, it is estimated that Norwalk viruses are responsible for about 1/3 of the cases not involving the 6-to-24-month age group. In developing countries the percentage of individuals who have developed immunity is very high at an early age. In the U.S. the percentage increases gradually with age, reaching 50% in the population over 18 years of age. Immunity, however, is not permanent and reinfection can occur.

7. Usual Course of Disease and Some Complications:

A mild and brief illness usually develops 24-48 h after contaminated food or water is consumed and lasts for 24-60 hours. Severe illness or hospitalization is very rare.

8. Target Populations:

All individuals who ingest the virus and who have not (within 24 months) had an infection with the same or related strain, are susceptible to infection and can develop the symptoms of gastroenteritis. Disease is more frequent in adults and older children than in the very young.

9. Analysis of Foods:

The virus has been identified in clams and oysters by radioimmunoassay. The genome of Norwalk virus has been cloned and development of gene probes and PCR amplification techniques to detect the virus in clinical specimens and possibly in food are under way.

10. Selected Outbreaks:

Foodborne outbreaks of gastroenteritis caused by Norwalk virus are often related to consumption of raw shellfish. Frequent and widespread outbreaks, reaching epidemic proportions, occurred in Australia (1978) and in the state of New York (1982) among consumers of raw clams and oysters. From 1983 to 1987, ten well documented outbreaks caused by Norwalk virus were reported in the U.S., involving a variety of foods: fruits, salads, eggs, clams, and bakery items.

Preliminary evidence suggests that large outbreaks of gastroenteritis which occurred in Pennsylvania and Delaware in September, 1987, were caused by Norwalk virus. The source of both outbreaks was traced to ice made with water from a contaminated well. In Pennsylvania, the ice was consumed at a football game, and in Delaware, at a cocktail party. Norwalk virus is also suspected to have caused an outbreak aboard a cruise ship in Hawaii in 1990. Fresh fruits were the probable vehicle of contamination.

Snow Mountain virus was implicated in an outbreak in a retirement community in California (1988) which resulted in two deaths. Illness was associated with consumption of shrimp probably contaminated by food handlers.

For outbreaks of Norwalk virus see MMWR 42(49):1993 Dec 17 and this MMWR 43(24):1994 Jun 24 as well.

The multistate outbreak of viral gastroenteritis associated with consumption of oysters from Apalachicola Bay, Florida, December 1994-January 1995 is reported in MMWR 44(2):1995 Jan 20.

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

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Other Gastroenteritis Viruses

1. Name of the Organism:
Other viruses associated with gastroenteritis

Although the rotavirus and the Norwalk family of viruses are the leading causes of viral gastroenteritis, a number of other viruses have been implicated in outbreaks, including astroviruses, caliciviruses, enteric adenoviruses and parvovirus. Astroviruses, caliciviruses, and the Norwalk family of viruses possess well-defined surface structures and are sometimes identified as "small round structured viruses" or SRSVs. Viruses with smooth edge and no discernible surface structure are designated "featureless viruses" or "small round viruses" (SRVs). These agents resemble enterovirus or parvovirus, and may be related to them.

Astroviruses are unclassified viruses which contain a single positive strand of RNA of about 7.5 kb surrounded by a protein capsid of 28-30 nm diameter. A five or six pointed star shape can be observed on the particles under the electron microscope. Mature virions contain two major coat proteins of about 33 kDa each and have a buoyant density in CsCl of 1.38 - 1.40 g/ml. At least five human serotypes have been identified in England. The Marin County agent found in the U.S. is serologically related to astrovirus type 5.

Caliciviruses are classified in the family Caliciviridae. They contain a single strand of RNA surrounded by a protein capsid of 31-40 nm diameter. Mature virions have cup-shaped indentations which give them a 'Star of David' appearance in the electron microscope. The particle contain a single major coat protein of 60 kDa and have a buoyant density in CsCl of 1.36 - 1.39 g/ml. Four serotypes have been identified in England.

Enteric adenoviruses represent serotypes 40 and 41 of the family Adenoviridae. These viruses contain a double-stranded DNA surrounded by a distinctive protein capsid of about 70 nm diameter. Mature virions have a buoyant density in CsCl of about 1.345 g/ml.

Parvoviruses belong to the family Parvoviridae, the only group of animal viruses to contain linear single-stranded DNA. The DNA genome is surrounded by a protein capsid of about 22 nm diameter. The buoyant density of the particle in CsCl is 1.39-1.42 g/ml. The Ditchling, Wollan, Paramatta, and cockle agents are candidate parvoviruses associated with human gastroenteritis.

2. Name of Acute Disease:

Common names of the illness caused by these viruses are acute nonbacterial infectious gastroenteritis and viral gastroenteritis.

3. Nature of Disease:

Viral gastroenteritis is usually a mild illness characterized by nausea, vomiting, diarrhea, malaise, abdominal pain, headache, and fever. The infectious dose is not known but is presumed to be low.

4. Diagnosis of Human Illness:

Specific diagnosis of the disease can be made by some laboratories possessing appropriate reagents. Identification of the virus present in early acute stool samples is made by immune electron microscopy and various enzyme immunoassays. Confirmation often requires demonstration of seroconversion to the agent by serological tests on acute and convalescent serum pairs.

5. Associated Foods:

Viral gastroenteritis is transmitted by the fecal-oral route via person-to-person contact or ingestion of contaminated foods and water. Ill food handlers may contaminate foods that are not further cooked before consumption. Enteric adenovirus may also be transmitted by the respiratory route. Shellfish have been implicated in illness caused by a parvo-like virus.

6. Frequency of Disease:

Astroviruses cause sporadic gastroenteritis in children under 4 years of age and account for about 4% of the cases hospitalized for diarrhea. Most American and British children over 10 years of age have antibodies to the virus.

Caliciviruses infect children between 6 and 24 months of age and account for about 3% of hospital admissions for diarrhea. By 6 years of age, more than 90% of all children have developed immunity to the illness.

The enteric adenovirus causes 5-20% of the gastroenteritis in young children, and is the second most common cause of gastroenteritis in this age group. By 4 years of age, 85% of all children have developed immunity to the disease. Parvo-like viruses have been implicated in a number of shellfish-associated outbreaks, but the frequency of disease is unknown.

7. Usual Course of Disease and Some Complications:

A mild, self limiting illness usually develops 10 to 70 hours after contaminated food or water is consumed and lasts for 2 to 9 days. The clinical features are milder but otherwise indistinguishable from rotavirus gastroenteritis. Co-infections with other enteric agents may result in more severe illness lasting a longer period of time.

8. Target Population:

The target populations for astro and caliciviruses are young children and the elderly. Only young children seem to develop illness caused by the enteric adenoviruses. Infection with these viruses is widespread and seems to result in development of immunity. Parvoviruses infect all age groups and probably do not ilicit a permanent immunity.

9. Analysis of Foods:

Only a parvovirus-like agent (cockle) has been isolated from seafood associated with an outbreak. Although foods are not routinely analyzed for these viruses, it may be possible to apply current immunological procedures to detect viruses in clinical specimens. Gene probes and PCR detection methods are currently being developed.

10. Selected Outbreaks:

Outbreaks of astrovirus and calicivirus occur mainly in child care settings and nursing homes. In the past decade, 7 outbreaks of calicivirus and 4 of astrovirus have been reported from England and Japan. In California, an outbreak caused by an astrovirus, the Marin County agent, occurred among elderly patients in a convalescent hospital. No typical calicivirus has been implicated in outbreaks in the U.S. However, if Norwalk and Norwalk-like viruses prove to be caliciviruses, they would account for most food and waterborne outbreaks of gastroenteritis in this country.

Outbreaks of adenovirus have been reported in England and Japan, all involving children in hospitals or day care centers.

The small featureless, parvo-like viruses caused outbreaks of gastroenteritis in primary and secondary schools in England (Ditchling and Wollan) and Australia (Paramatta). The cockle agent caused a large community-wide outbreak in England (1977) associated with consumption of contaminated seafood. Parvo-like viruses were also implicated in several outbreaks which occurred in the States of New York and Louisiana in 1982-1983.

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

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Center for Food Safety & Applied Nutrition
Foodborne Pathogenic Microorganisms and Natural Toxins Handbook

Ciguatera

CDC/MMWR NIH/PubMed

1. Name of Toxin: Ciguatera

2. Name of Disease: Ciguatera Fish Poisoning

Ciguatera is a form of human poisoning caused by the consumption of subtropical and tropical marine finfish which have accumulated naturally occurring toxins through their diet. The toxins are known to originate from several dinoflagellate (algae) species that are common to ciguatera endemic regions in the lower latitudes.

3. Nature of Disease: Manifestations of ciguatera in humans usually involves a combination of gastrointestinal, neurological, and cardiovascular disorders. Symptoms defined within these general categories vary with the geographic origin of toxic fish.

4. Normal Course of Disease: Initial signs of poisoning occur within six hours after consumption of toxic fish and include perioral numbness and tingling (paresthesia), which may spread to the extremities, nausea, vomiting, and diarrhea. Neurological signs include intensified paresthesia, arthralgia, myalgia, headache, temperature sensory reversal and acute sensitivity to temperature extremes, vertigo, and muscular weakness to the point of prostration. Cardiovascular signs include arrhythmia, bradycardia or tachycardia, and reduced blood pressure. Ciguatera poisoning is usually self-limiting, and signs of poisoning often subside within several days from onset. However, in severe cases the neurological symptoms are known to persist from weeks to months. In a few isolated cases neurological symptoms have persisted for several years, and in other cases recovered patients have experienced recurrence of neurological symptoms months to years after recovery. Such relapses are most often associated with changes in dietary habits or with consumption of alcohol. There is a low incidence of death resulting from respiratory and cardiovascular failure.

5. Diagnosis of Human Illness: Clinical testing procedures are not presently available for the diagnosis of ciguatera in humans. Diagnosis is based entirely on symptomology and recent dietary history. An enzyme immunoassay (EIA) designed to detect toxic fish in field situations is under evaluation by the Association of Official Analytical Chemists (AOAC) and may provide some measure of protection to the public in the future.

6. Associated Foods: Marine finfish most commonly implicated in ciguatera fish poisoning include the groupers, barracudas, snappers, jacks, mackerel, and triggerfish. Many other species of warm-water fishes harbor ciguatera toxins. The occurrence of toxic fish is sporadic, and not all fish of a given species or from a given locality will be toxic.

7. Relative Frequency of Disease: The relative frequency of ciguatera fish poisoning in the United States is not known. The disease has only recently become known to the general medical community, and there is a concern that incidence is largely under-reported because of the generally non-fatal nature and short duration of the disease.

8. Target Population: All humans are believed to be susceptible to ciguatera toxins. Populations in tropical/subtropical regions are most likely to be affected because of the frequency of exposure to toxic fishes. However, the increasing per capita consumption of fishery products coupled with an increase in interregional transportation of seafood products has expanded the geographic range of human poisonings.

9. Analysis of Foods: The ciguatera toxins can be recovered from toxic fish through tedious extraction and purification procedures. The mouse bioassay is a generally accepted method of establishing toxicity of suspect fish. A much simplified EIA method intended to supplant the mouse bioassay for identifying ciguatera toxins is under evaluation.

10. Selected Outbreaks: Isolated cases of ciguatera fish poisoning have occurred along the eastern coast of the United States from south Florida to Vermont. Hawaii, the U.S. Virgin Islands, and Puerto Rico experience sporadic cases with some regularity. A major outbreak of ciguatera occurred in Puerto Rico between April and June 1981 in which 49 persons were afflicted and two fatalities occurred. This outbreak prompted government officials of the Commonwealth of Puerto Rico to ban the sale of barracuda, amberjack, and blackjack.

In February-March of 1987 a large common-source outbreak of ciguatera occurred among Canadian vacationers returning from a Caribbean resort. Of 147 tourists, 61 ate a fish casserole shortly before departure, resulting in 57 identified cases of ciguatera.

In May of 1988 several hundred pounds of fish (primarily hogfish) from the Dry Tortuga Bank were responsible for over 100 human poisonings in Palm Beach County, Florida. The fish were sold to a seafood distributor after the fishermen (sport spearfishermen) themselves were first afflicted but dismissed their illness as seasickness and hangover. The poisonings resulted in a statewide warning against eating hogfish, grouper, red snapper, amberjack, and barracuda caught at the Dry Tortuga Bank.

For a report on Ciguatera poisoning in Florida, see this MMWR 42(21):1993 Jun 04.

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

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Various Shellfish-Associated Toxins

1. Name of Toxins: Various Shellfish-Associated

Shellfish poisoning is caused by a group of toxins elaborated by planktonic algae (dinoflagellates, in most cases) upon which the shellfish feed. The toxins are accumulated and sometimes metabolized by the shellfish. The 20 toxins responsible for paralytic shellfish poisonings (PSP) are all derivatives of saxitoxin. Diarrheic shellfish poisoning (DSP) is presumably caused by a group of high molecular weight polyethers, including okadaic acid, the dinophysis toxins, the pectenotoxins, and yessotoxin. Neurotoxic shellfish poisoning (NSP) is the result of exposure to a group of polyethers called brevetoxins. Amnesic shellfish poisoning (ASP) is caused by the unusual amino acid, domoic acid, as the contaminant of shellfish.

2. Name of the Acute Diseases:

Shellfish Poisoning:

Paralytic Shellfish Poisoning (PSP), Diarrheic Shellfish Poisoning (DSP), Neurotoxic Shellfish Poisoning (NSP), Amnesic Shellfish Poisoning (ASP).

3. Nature of the Diseases:

Ingestion of contaminated shellfish results in a wide variety of symptoms, depending upon the toxins(s) present, their concentrations in the shellfish and the amount of contaminated shellfish consumed. In the case of PSP, the effects are predominantly neurological and include tingling, burning, numbness, drowsiness, incoherent speech, and respiratory paralysis. Less well characterized are the symptoms associated with DSP, NSP, and ASP. DSP is primarily observed as a generally mild gastrointestinal disorder, i.e., nausea, vomiting, diarrhea, and abdominal pain accompanied by chills, headache, and fever. Both gastrointestinal and neurological symptoms characterize NSP, including tingling and numbness of lips, tongue, and throat, muscular aches, dizziness, reversal of the sensations of hot and cold, diarrhea, and vomiting. ASP is characterized by gastrointestinal disorders (vomiting, diarrhea, abdominal pain) and neurological problems (confusion, memory loss, disorientation, seizure, coma).

4. Normal Course of the Disease:

PSP: Symptoms of the disease develop fairly rapidly, within 0.5 to 2 hours after ingestion of the shellfish, depending on the amount of toxin consumed. In severe cases respiratory paralysis is common, and death may occur if respiratory support is not provided. When such support is applied within 12 hours of exposure, recovery usually is complete, with no lasting side effects. In unusual cases, because of the weak hypotensive action of the toxin, death may occur from cardiovascular collapse despite respiratory support.

NSP: Onset of this disease occurs within a few minutes to a few hours; duration is fairly short, from a few hours to several days. Recovery is complete with few after effects; no fatalities have been reported.

DSP: Onset of the disease, depending on the dose of toxin ingested, may be as little as 30 minutes to 2 to 3 hours, with symptoms of the illness lasting as long as 2 to 3 days. Recovery is complete with no after effects; the disease is generally not life threatening.

ASP: The toxicosis is characterized by the onset of gastrointestinal symptoms within 24 hours; neurological symptoms occur within 48 hours. The toxicosis is particularly serious in elderly patients, and includes symptoms reminiscent of Alzheimer's disease. All fatalities to date have involved elderly patients.

5. Diagnosis of Human Illnesses: Diagnosis of shellfish poisoning is based entirely on observed symptomatology and recent dietary history.

6. Associated Foods: All shellfish (filter-feeding molluscs) are potentially toxic. However, PSP is generally associated with mussels, clams, cockles, and scallops; NSP with shellfish harvested along the Florida coast and the Gulf of Mexico; DSP with mussels, oysters, and scallops, and ASP with mussels.

7. Relative Frequency of Disease: Good statistical data on the occurrence and severity of shellfish poisoning are largely unavailable, which undoubtedly reflects the inability to measure the true incidence of the disease. Cases are frequently misdiagnosed and, in general, infrequently reported. Of these toxicoses, the most serious from a public health perspective appears to be PSP. The extreme potency of the PSP toxins has, in the past, resulted in an unusually high mortality rate.

8. Target Populations: All humans are susceptible to shellfish poisoning. Elderly people are apparently predisposed to the severe neurological effects of the ASP toxin. A disproportionate number of PSP cases occur among tourists or others who are not native to the location where the toxic shellfish are harvested. This may be due to disregard for either official quarantines or traditions of safe consumption, both of which tend to protect the local population.

9. Analysis of Foods: The mouse bioassay has historically been the most universally applied technique for examining shellfish (especially for PSP); other bioassay procedures have been developed but not generally applied. Unfortunately, the dose-survival times for the DSP toxins in the mouse assay fluctuate considerably and fatty acids interfere with the assay, giving false-positive results; consequently, a suckling mouse assay that has been developed and used for control of DSP measures fluid accumulation after injection of the shellfish extract. In recent years considerable effort has been applied to development of chemical assays to replace these bioassays. As a result a good high performance liquid chromatography (HPLC) procedure has been developed to identify individual PSP toxins (detection limit for saxitoxin = 20 fg/100 g of meats; 0.2 ppm), an excellent HPLC procedure (detection limit for okadaic acid = 400 ng/g; 0.4 ppm), a commercially available immunoassay (detection limit for okadaic acid = 1 fg/100 g of meats; 0.01 ppm) for DSP and a totally satisfactory HPLC procedure for ASP (detection limit for domoic acid = 750 ng/g; 0.75 ppm).

10. Selected Outbreaks:

PSP is associated with relatively few outbreaks, most likely because of the strong control programs in the United States that prevent human exposure to toxic shellfish. That PSP can be a serious public health problem, however, was demonstrated in Guatemala, where an outbreak of 187 cases with 26 deaths, recorded in 1987, resulted from ingestion of a clam soup. The outbreak led to the establishment of a control program over shellfish harvested in Guatemala.

ASP first came to the attention of public health authorities in 1987 when 156 cases of acute intoxication occurred as a result of ingestion of cultured blue mussels (Mytilus edulis) harvested off Prince Edward Island, in eastern Canada; 22 individuals were hospitalized and three elderly patients eventually died.

The occurrence of DSP in Europe is sporadic, continuous and presumably widespread (anecdotal). DSP poisoning has not been confirmed in U.S. seafood, but the organisms that produce DSP are present in U.S. waters. An outbreak of DSP was recently confirmed in Eastern Canada. Outbreaks of NSP are sporadic and continuous along the Gulf coast of Florida and were recently reported in North Carolina and Texas.

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

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Scombrotoxin

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1. Name of Toxin: Scombrotoxin

2. Name of Acute Disease: Scombroid Poisoning (also called Histamine Poisoning)

Scombroid poisoning is caused by the ingestion of foods that contain high levels of histamine and possibly other vasoactive amines and compounds. Histamine and other amines are formed by the growth of certain bacteria and the subsequent action of their decarboxylase enzymes on histidine and other amino acids in food, either during the production of a product such as Swiss cheese or by spoilage of foods such as fishery products, particularly tuna or mahi mahi. However, any food that contains the appropriate amino acids and is subjected to certain bacterial contamination and growth may lead to scombroid poisoning when ingested.

3. Nature of Disease: Initial symptoms may include a tingling or burning sensation in the mouth, a rash on the upper body and a drop in blood pressure. Frequently, headaches and itching of the skin are encountered. The symptoms may progress to nausea, vomiting, and diarrhea and may require hospitalization, particularly in the case of elderly or impaired patients.

4. Normal Course of Disease: The onset of intoxication symptoms is rapid, ranging from immediate to 30 minutes. The duration of the illness is usually 3 hours, but may last several days.

5. Diagnosis of Human Illness: Diagnosis of the illness is usually based on the patient's symptoms, time of onset, and the effect of treatment with antihistamine medication. The suspected food must be analyzed within a few hours for elevated levels of histamine to confirm a diagnosis.

6. Associated Foods: Fishery products that have been implicated in scombroid poisoning include the tunas (e.g., skipjack and yellowfin), mahi mahi, bluefish, sardines, mackerel, amberjack, and abalone. Many other products also have caused the toxic effects. The primary cheese involved in intoxications has been Swiss cheese. The toxin forms in a food when certain bacteria are present and time and temperature permit their growth. Distribution of the toxin within an individual fish fillet or between cans in a case lot can be uneven, with some sections of a product causing illnesses and others not. Neither cooking, canning, or freezing reduces the toxic effect. Common sensory examination by the consumer cannot ensure the absence or presence of the toxin. Chemical testing is the only reliable test for evaluation of a product.

7. Relative Frequency of Disease: Scombroid poisoning remains one of the most common forms of fish poisoning in the United States. Even so, incidents of poisoning often go unreported because of the lack of required reporting, a lack of information by some medical personnel, and confusion with the symptoms of other illnesses. Difficulties with underreporting are a worldwide problem. In the United States from 1968 to 1980, 103 incidents of intoxication involving 827 people were reported. For the same period in Japan, where the quality of fish is a national priority, 42 incidents involving 4,122 people were recorded. Since 1978, 2 actions by FDA have reduced the frequency of intoxications caused by specific products. A defect action level for histamine in canned tuna resulted in increased industry quality control. Secondly, blocklisting of mahi mahi reduced the level of fish imported to the United States.

8. Target Population: All humans are susceptible to scombroid poisoning; however, the symptoms can be severe for the elderly and for those taking medications such as isoniazid. Because of the worldwide network for harvesting, processing, and distributing fishery products, the impact of the problem is not limited to specific geographical areas of the United States or consumption pattern. These foods are sold for use in homes, schools, hospitals, and restaurants as fresh, frozen, or processed products.

9. Analysis of Foods: An official method was developed at FDA to determine histamine, using a simple alcoholic extraction and quantitation by fluorescence spectroscopy. There are other untested procedures in the literature.

10. Selected Outbreaks: Several large outbreaks of scombroid poisoning have been reported. In 1970, some 40 children in a school lunch program became ill from imported canned tuna. In 1973, more than 200 consumers across the United States were affected by domestic canned tuna. In 1979-1980 more than 200 individuals became ill after consuming imported frozen mahi mahi. Symptoms varied with each incident. In the 1973 situation, of the interviewed patients, 86% experienced nausea, 55% diarrhea, 44% headaches and 32% rashes.

Other incidents of intoxication have resulted from the consumption of canned abalone-like products, canned anchovies, and fresh and frozen amberjack, bluefish sole, and scallops. In particular, shipments of unfrozen fish packed in refrigerated containers have posed a significant problem because of inadequate temperature control.

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

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Mushroom toxins

1. Name of Toxin(s): Amanitin, Gyromitrin, Orellanine, Muscarine, Ibotenic Acid, Muscimol, Psilocybin, Coprine

2. Name of Acute Disease: Mushroom Poisoning, Toadstool Poisoning

Mushroom poisoning is caused by the consumption of raw or cooked fruiting bodies (mushrooms, toadstools) of a number of species of higher fungi. The term toadstool (from the German Todesstuhl, death's stool) is commonly given to poisonous mushrooms, but for individuals who are not experts in mushroom identification there are generally no easily recognizable differences between poisonous and nonpoisonous species. Old wives' tales notwithstanding, there is no general rule of thumb for distinguishing edible mushrooms and poisonous toadstools. The toxins involved in mushroom poisoning are produced naturally by the fungi themselves, and each individual specimen of a toxic species should be considered equally poisonous. Most mushrooms that cause human poisoning cannot be made nontoxic by cooking, canning, freezing, or any other means of processing. Thus, the only way to avoid poisoning is to avoid consumption of the toxic species. Poisonings in the United States occur most commonly when hunters of wild mushrooms (especially novices) misidentify and consume a toxic species, when recent immigrants collect and consume a poisonous American species that closely resembles an edible wild mushroom from their native land, or when mushrooms that contain psychoactive compounds are intentionally consumed by persons who desire these effects.

3. Nature of Disease(s): Mushroom poisonings are generally acute and are manifested by a variety of symptoms and prognoses, depending on the amount and species consumed. Because the chemistry of many of the mushroom toxins (especially the less deadly ones) is still unknown and positive identification of the mushrooms is often difficult or impossible, mushroom poisonings are generally categorized by their physiological effects. There are four categories of mushroom toxins: protoplasmic poisons (poisons that result in generalized destruction of cells, followed by organ failure); neurotoxins (compounds that cause neurological symptoms such as profuse sweating, coma, convulsions, hallucinations, excitement, depression, spastic colon); gastrointestinal irritants (compounds that produce rapid, transient nausea, vomiting, abdominal cramping, and diarrhea); and disulfiram-like toxins. Mushrooms in this last category are generally nontoxic and produce no symptoms unless alcohol is consumed within 72 hours after eating them, in which case a short-lived acute toxic syndrome is produced.

4. Normal Course of Disease(s): The normal course of the disease varies with the dose and the mushroom species eaten. Each poisonous species contains one or more toxic compounds which are unique to few other species. Therefore, cases of mushroom poisonings generally do not resembles each other unless they are caused by the same or very closely related mushroom species. Almost all mushroom poisonings may be grouped in one of the categories outlined above.

PROTOPLASMIC POISONS

Amatoxins:

Several mushroom species, including the Death Cap or Destroying Angel (*Amanita phalloides*, *A. virosa*), the Fool's Mushroom (*A. verna*) and several of their relatives, along with the Autumn Skullcap (*Galerina autumnalis*) and some of its relatives, produce a family of cyclic octapeptides called amanitins. Poisoning by the amanitins is characterized by a long latent period (range 6-48 hours, average 6-15 hours) during which the patient shows no symptoms. Symptoms appear at the end of the latent period in the form of sudden, severe seizures of abdominal pain, persistent vomiting and watery diarrhea, extreme thirst, and lack of urine production. If this early phase is survived, the patient may appear to recover for a short time, but this period will generally be followed by a rapid and severe loss of strength, prostration, and pain-caused restlessness. Death in 50-90% of the cases from progressive and irreversible liver, kidney, cardiac, and skeletal muscle damage may follow within 48 hours (large dose), but the disease more typically lasts 6 to 8 days in adults and 4 to 6 days in children. Two or three days after the onset of the later phase, jaundice, cyanosis, and coldness of the skin occur. Death usually follows a period of coma and occasionally convulsions. If recovery occurs, it generally requires at least a month and is accompanied by enlargement of the liver. Autopsy will usually reveal fatty degeneration and necrosis of the liver and kidney.

Hydrazines:

Certain species of False Morel (*Gyromitra esculenta* and *G. gigas*) contain the protoplasmic poison gyromitrin, a volatile hydrazine derivative. Poisoning by this toxin superficially resembles *Amanita* poisoning but is less severe. There is generally a latent period of 6 - 10 hours after ingestion during which no symptoms are evident, followed by sudden onset of abdominal discomfort (a feeling of fullness), severe headache, vomiting, and sometimes diarrhea. The toxin affects primarily the liver, but there are additional disturbances to blood cells and the central nervous system. The mortality rate is relatively low (2-4%). Poisonings with symptoms almost identical to those produced by *Gyromitra* have also been reported after ingestion of the Early False Morel (*Verpa bohemica*). The toxin is presumed to be related to gyromitrin but has not yet been identified.

Orellanine:

The final type of protoplasmic poisoning is caused by the Sorrel Webcap mushroom (*Cortinarius orellanus*) and some of its relatives. This mushroom produces orellanine, which causes a type of poisoning characterized by an extremely long asymptomatic latent period of 3 to 14 days. An intense, burning

thirst (polydipsia) and excessive urination (polyuria) are the first symptoms. This may be followed by nausea, headache, muscular pains, chills, spasms, and loss of consciousness. In severe cases, severe renal tubular necrosis and kidney failure may result in death (15%) several weeks after the poisoning. Fatty degeneration of the liver and severe inflammatory changes in the intestine accompany the renal damage, and recovery in less severe cases may require several months.

NEUROTOXINS

Poisonings by mushrooms that cause neurological problems may be divided into three groups, based on the type of symptoms produced, and named for the substances responsible for these symptoms.

Muscarine Poisoning:

Ingestion of any number of *Inocybe* or *Clitocybe* species (e.g., *Inocybe geophylla*, *Clitocybe dealbata*) results in an illness characterized primarily by profuse sweating. This effect is caused by the presence in these mushrooms of high levels (3- 4%) of muscarine. Muscarine poisoning is characterized by increased salivation, perspiration, and lacrimation within 15 to 30 minutes after ingestion of the mushroom. With large doses, these symptoms may be followed by abdominal pain, severe nausea, diarrhea, blurred vision, and labored breathing. Intoxication generally subsides within 2 hours. Deaths are rare, but may result from cardiac or respiratory failure in severe cases.

Ibotenic acid/Muscimol Poisoning:

The Fly Agaric (*Amanita muscaria*) and Panthercap (*Amanita pantherina*) mushrooms both produce ibotenic acid and muscimol. Both substances produce the same effects, but muscimol is approximately 5 times more potent than ibotenic acid. Symptoms of poisoning generally occur within 1 - 2 hours after ingestion of the mushrooms. An initial abdominal discomfort may be present or absent, but the chief symptoms are drowsiness and dizziness (sometimes accompanied by sleep), followed by a period of hyperactivity, excitability, illusions, and delirium. Periods of drowsiness may alternate with periods of excitement, but symptoms generally fade within a few hours. Fatalities rarely occur in adults, but in children, accidental consumption of large quantities of these mushrooms may cause convulsions, coma, and other neurologic problems for up to 12 hours.

Psilocybin Poisoning:

A number of mushrooms belonging to the genera *Psilocybe, Panaeolus, Copelandia, Gymnopilus, Conocybe*, and *Pluteus*, when ingested, produce a syndrome similar to alcohol intoxication (sometimes accompanied by hallucinations). Several of these mushrooms (e.g., *Psilocybe cubensis, P. mexicana, Conocybe cyanopus*) are eaten for their psychotropic effects in religious ceremonies of certain native American tribes, a practice which dates to the pre- Columbian era. The toxic effects are caused by psilocin and psilocybin. Onset of symptoms is usually rapid and the effects generally subside within 2 hours. Poisonings by these mushrooms are rarely fatal in adults and may be distinguished from ibotenic acid poisoning by the absence of

drowsiness or coma. The most severe cases of psilocybin poisoning occur in small children, where large doses may cause the hallucinations accompanied by fever, convulsions, coma, and death. These mushrooms are generally small, brown, nondescript, and not particularly fleshy; they are seldom mistaken for food fungi by innocent hunters of wild mushrooms. Poisonings caused by intentional ingestion of these mushrooms by people with no legitimate religious justification must be handled with care, since the only cases likely to be seen by the physician are overdoses or intoxications caused by a combination of the mushroom and some added psychotropic substance (such as PCP).

GASTROINTESTINAL IRRITANTS

Numerous mushrooms, including the Green Gill (*Chlorophyllum molybdites*), Gray Pinkgill (*Entoloma lividum*), Tigertop (*Tricholoma pardinum*), Jack O'Lantern (*Omphalotus illudens*), Naked Brimcap (*Paxillus involutus*), Sickener (*Russula emetica*), Early False Morel (*Verpa bohemica*), Horse mushroom (*Agaricus arvensis*) and Pepper bolete (*Boletus piperatus*), contain toxins that can cause gastrointestinal distress, including but not limited to nausea, vomiting, diarrhea, and abdominal cramps. In many ways these symptoms are similar to those caused by the deadly protoplasmic poisons. The chief and diagnostic difference is that poisonings caused by these mushrooms have a rapid onset, rather than the delayed onset seen in protoplasmic poisonings. Some mushrooms (including the first five species mentioned above) may cause vomiting and/or diarrhea which lasts for several days. Fatalities caused by these mushrooms are relatively rare and are associated with dehydration and electrolyte imbalances caused by diarrhea and vomiting, especially in debilitated, very young, or very old patients. Replacement of fluids and other appropriate supportive therapy will prevent death in these cases. The chemistry of the toxins responsible for this type of poisoning is virtually unknown, but may be related to the presence in some mushrooms of unusual sugars, amino acids, peptides, resins, and other compounds.

DISULFIRAM-LIKE POISONING

The Inky Cap Mushroom (*Coprinus atramentarius*) is most commonly responsible for this poisoning, although a few other species have also been implicated. A complicating factor in this type of intoxication is that this species is generally considered edible (i.e., no illness results when eaten in the absence of alcoholic beverages). The mushroom produces an unusual amino acid, coprine, which is converted to cyclopropanone hydrate in the human body. This compound interferes with the breakdown of alcohol, and consumption of alcoholic beverages within 72 hours after eating it will cause headache, nausea and vomiting, flushing, and cardiovascular disturbances that last for 2 - 3 hours.

MISCELLANEOUS POISONINGS

Young fruiting bodies of the sulfur shelf fungus *Laetiporus sulphureus* are considered edible. However, ingestion of this shelf fungus has caused digestive upset and other symptoms in adults and visual hallucinations and ataxia in a child.

5. Diagnosis of Human Illness:

A clinical testing procedure is currently available only for the most serious types of mushroom toxins, the amanitins. The commercially available method uses a 3H-radioimmunoassay (RIA) test kit and can detect sub-nanogram levels of toxin in urine and plasma. Unfortunately, it requires a 2-hour incubation period, and this is an excruciating delay in a type of poisoning which the clinician generally does not see until a day or two has passed. A 125I-based kit which overcomes this problem has recently been reported, but has not yet reached the clinic. A sensitive and rapid HPLC technique has been reported in the literature even more recently, but it has not yet seen clinical application. Since most clinical laboratories in this country do not use even the older RIA technique, diagnosis is based entirely on symptomology and recent dietary history. Despite the fact that cases of mushroom poisoning may be broken down into a relatively small number of categories based on symptomatology, positive botanical identification of the mushroom species consumed remains the only means of unequivocally determining the particular type of intoxication involved, and it is still vitally important to obtain such accurate identification as quickly as possible. Cases involving ingestion of more than one toxic species in which one set of symptoms masks or mimics another set are among many reasons for needing this information. Unfortunately, a number of factors (not discussed here) often make identification of the causative mushroom impossible. In such cases, diagnosis must be based on symptoms alone. In order to rule out other types of food poisoning and to conclude that the mushrooms eaten were the cause of the poisoning, it must be established that everyone who ate the suspect mushrooms became ill and that no one who did not eat the mushrooms became ill. Wild mushrooms eaten raw, cooked, or processed should always be regarded as prime suspects. After ruling out other sources of food poisoning and positively implicating mushrooms as the cause of the illness, diagnosis may proceed in two steps. The first step, outlined in Table 1, provides an early indication of the seriousness of the disease and its prognosis.

As described above, the protoplasmic poisons are the most likely to be fatal or to cause irreversible organ damage. In the case of poisoning by the deadly Amanitas, important laboratory indicators of liver (elevated LDH, SGOT, and bilirubin levels) and kidney (elevated uric acid, creatinine, and BUN levels) damage will be present. Unfortunately, in the absence of dietary history, these signs could be mistaken for symptoms of liver or kidney impairment as the result of other causes (e.g., viral hepatitis). It is important that this distinction be made as quickly as possible, because the delayed onset of symptoms will generally mean that the organ has already been damaged. The importance of rapid diagnosis is obvious: victims who are hospitalized and given aggressive support therapy almost immediately after ingestion have a mortality rate of only 10%, whereas those admitted 60 or more hours after ingestion have a 50-90% mortality rate. Table 2 provides more accurate diagnoses and appropriate therapeutic measures. A recent report indicates that amanitins are observable in urine well before the onset of any symptoms, but that laboratory tests for liver dysfunction do not appear until well after the organ has been damaged.

6. Associated Foods:

Mushroom poisonings are almost always caused by ingestion of wild mushrooms that have been collected by nonspecialists (although specialists have also been poisoned). Most cases occur when toxic species are confused with edible species, and a useful question to ask of the victims or their mushroom-picking benefactors is the identity of the mushroom they thought they were picking. In the absence of a well- preserved specimen, the answer to this question could narrow the possible suspects considerably. Intoxication has also occurred when reliance was placed on some folk method of distinguishing poisonous and safe species. Outbreaks have occurred after ingestion of fresh, raw mushrooms, stir-fried mushrooms, home-canned mushrooms, mushrooms cooked in tomato sauce (which rendered the sauce itself toxic, even when no mushrooms were consumed), and mushrooms that were blanched and frozen at home. Cases of poisoning by home-canned and frozen mushrooms are especially insidious because a single outbreak may easily become a multiple outbreak when the preserved toadstools are carried to another location and consumed at another time.

Specific cases of mistaken mushroom identity appears frequently. The Early False Morel *Gyromitra esculenta* is easily confused with the true Morel *Morchella esculenta*, and poisonings have occurred after consumption of fresh or cooked *Gyromitra*. *Gyromitra* poisonings have also occurred after ingestion of commercially available "morels" contaminated with *G. esculenta*. The commercial sources for these fungi (which have not yet been successfully cultivated on a large scale) are field collection of wild morels by semiprofessionals. Cultivated commercial mushrooms of whatever species are almost never implicated in poisoning outbreaks unless there are associated problems such as improper canning (which lead to bacterial food poisoning). A short list of the mushrooms responsible for serious poisonings and the edible mushrooms with which they are confused is presented in Table 3. Producers of mild gastroenteritis are too numerous to list here, but include members of many of the most abundant genera, including *Agaricus, Boletus, Lactarius, Russula, Tricholoma, Coprinus, Pluteus*, and others. The Inky Cap Mushroom (*Coprinus atrimentarius*) is considered both edible and delicious, and only the unwary who consume alcohol after eating this mushroom need be concerned. Some other members of the genus *Coprinus* (Shaggy Mane, *C. comatus*; Glistening Inky Cap, *C. micaceus*, and others) and some of the larger members of the *Lepiota* family such as the Parasol Mushroom (*Leucocoprinus procera*) do not contain coprine and do not cause this effect. The potentially deadly Sorrel Webcap Mushroom (*Cortinarius orellanus*) is not easily distinguished from nonpoisonous webcaps belonging to the same distinctive genus, and all should be avoided.

Most of the psychotropic mushrooms (*Inocybe* spp., *Conocybe* spp., *Paneolus* spp., *Pluteus* spp.) are in general appearance small, brown, and leathery (the so-called "Little Brown Mushrooms" or LBMs) and relatively unattractive from a culinary standpoint. The Sweat Mushroom (*Clitocybe dealbata*) and the Smoothcap Mushroom (*Psilocybe cubensis*) are small, white, and leathery. These small, unattractive mushrooms are distinctive, fairly unappetizing, and not easily confused with the fleshier fungi normally considered edible. Intoxications associated with them are less likely to be accidental, although both *C. dealbata* and *Paneolus foenisicii* have been found growing in the same fairy ring area as the edible (and choice) Fairy Ring Mushroom (*Marasmius oreades*) and the Honey Mushroom (*Armillariella mellea*), and have been

consumed when the picker has not carefully examined every mushroom picked from the ring. Psychotropic mushrooms, which are larger and therefore more easily confused with edible mushrooms, include the Showy Flamecap or Big Laughing Mushroom (*Gymnopilus spectabilis*), which has been mistaken for Chanterelles (*Cantharellus* spp.) and for *Gymnopilus ventricosus* found growing on wood of conifers in western North America. The Fly Agaric (*Amanita muscaria*) and Panthercap (*Amanita pantherina*) mushrooms are large, fleshy, and colorful. Yellowish cap colors on some varieties of the Fly Agaric and the Panthercap are similar to the edible Caesar's Mushroom (*Amanita caesarea*), which is considered a delicacy in Italy. Another edible yellow capped mushroom occasionally confused with yellow *A. muscaria* and *A. pantherina* varieties are the Yellow Blusher (*Amanita flavorubens*). Orange to yellow-orange *A. muscaria* and *A. pantherina* may also be confused with the Blusher (*Amanita rubescens*) and the Honey Mushroom (*Armillariella mellea*). White to pale forms of *A. muscaria* may be confused with edible field mushrooms (*Agaricus* spp.). Young (button stage) specimens of *A. muscaria* have also been confused with puffballs.

7. Relative Frequency of Disease:

Accurate figures on the relative frequency of mushroom poisonings are difficult to obtain. For the 5-year period between 1976 and 1981, 16 outbreaks involving 44 cases were reported to the Centers for Disease Control in Atlanta (Rattanvilay et al. MMWR 31(21): 287-288, 1982). The number of unreported cases is, of course, unknown. Cases are sporadic and large outbreaks are rare. Poisonings tend to be grouped in the spring and fall when most mushroom species are at the height of their fruiting stage. While the actual incidence appears to be very low, the potential exists for grave problems. Poisonous mushrooms are not limited in distribution as are other poisonous organisms (such as dinoflagellates). Intoxications may occur at any time and place, with dangerous species occurring in habitats ranging from urban lawns to deep woods. As Americans become more adventurous in their mushroom collection and consumption, poisonings are likely to increase.

8. Target Population:

All humans are susceptible to mushroom toxins. The poisonous species are ubiquitous, and geographical restrictions on types of poisoning that may occur in one location do not exist (except for some of the hallucinogenic LBMs, which occur primarily in the American southwest and southeast). Individual specimens of poisonous mushrooms are also characterized by individual variations in toxin content based on genetics, geographic location, and growing conditions. Intoxications may thus be more or less serious, depending not on the number of mushrooms consumed, but on the dose of toxin delivered. In addition, although most cases of poisoning by higher plants occur in children, toxic mushrooms are consumed most often by adults. Occasional accidental mushroom poisonings of children and pets have been reported, but adults are more likely to actively search for and consume wild mushrooms for culinary purposes. Children are more seriously affected by the normally nonlethal toxins than are adults and are more likely to suffer very serious consequences from ingestion of relatively smaller doses. Adults who consume mushrooms are also more likely to recall what was eaten and when, and are able to describe their symptoms more accurately than are children. Very old, very young, and debilitated persons of both sexes are more likely to become seriously ill from all types of mushroom poisoning, even those types which are generally considered to be mild.

Many idiosyncratic adverse reactions to mushrooms have been reported. Some

mushrooms cause certain people to become violently ill, while not affecting others who consumed part of the same mushroom cap. Factors such as age, sex, and general health of the consumer do not seem to be reliable predictors of these reactions, and they have been attributed to allergic or hypersensitivity reactions and to inherited inability of the unfortunate victim to metabolize certain unusual fungal constituents (such as the uncommon sugar, trehalose). These reactions are probably not true poisonings as the general population does not seem to be affected.

9. Analysis of Foods for Toxins: The mushroom toxins can with difficulty be recovered from poisonous fungi, cooking water, stomach contents, serum, and urine. Procedures for extraction and quantitation are generally elaborate and time-consuming, and the patient will in most cases have recovered by the time an analysis is made on the basis of toxin chemistry. The exact chemical natures of most of the toxins that produce milder symptoms are unknown. Chromatographic techniques (TLC, GLC, HPLC) exist for the amanitins, orellanine, muscimol/ibotenic acid, psilocybin, muscarine, and the gyromitrins. The amanitins may also be determined by commercially available 3H-RIA kits. The most reliable means of diagnosing a mushroom poisoning remains botanical identification of the fungus that was eaten. An accurate pre-ingestion determination of species will also prevent accidental poisoning in 100% of cases. Accurate post-ingestion analyses for specific toxins when no botanical identification is possible may be essential only in cases of suspected poisoning by the deadly *Amanitas*, since prompt and aggressive therapy (including lavage, activated charcoal, and plasmapheresis) can greatly reduce the mortality rate.

10. Selected Outbreaks: Isolated cases of mushroom poisoning have occurred throughout the continental United States. The occurred in Oregon in October,1988, and involved the intoxication of five people who consumed stir-fried *Amanita phalloides*. The poisonings were severe, and at this writing three of the five people had undergone liver transplants for treatment of amanitin-induced liver failure. Other recent cases have included the July, 1986, poisoning of a family in Philadelphia, by *Chlorophyllum molybdites*; the September, 1987, intoxication of seven men in Bucks County, PA, by spaghetti sauce which contained Jack O'Lantern mushroom (*Omphalotus illudens*); and of 14 teenage campers in Maryland by the same species (July, 1987). A report of a North Carolina outbreak of poisoning by False Morel (*Gyromitra* spp.) appeared in 1986. A 1985 report details a case of *Chlorophyllum molybdites* which occurred in Arkansas; a fatal poisoning case caused by an amanitin containing *Lepiota* was described in 1986. In 1981, two Berks County, PA, people were poisoned (one fatally) after ingesting *Amanita phalloides*, while in the same year, seven Laotian refugees living in California were poisoned by *Russula* spp. In separate 1981 incidents, several people from New York State were poisoned by *Omphalotus illudens*, *Amanita muscaria*, *Entoloma lividum*, and *Amanita virosa*. An outbreak of gastroenteritis during a banquet for 482 people in Vancouver, British Columbia, was reported by the Vancouver Health Department in June, 1991. Seventy-seven of the guests reported symptoms consisting of early onset nausea (15-30 min), diarrhea (20 min-13 h), vomiting (20-60 min), cramps and bloated feeling. Other symptoms included feeling warm, clamminess, numbness of the tongue and extreme thirst along with two cases of hive-like rash with onset of 3-7 days. Bacteriological tests were negative. This intoxication merits special attention because it involved consumption of species normally considered not only edible but choice. The fungi involved were the morels *Morchella esculenta* and *M. elata* (*M.*

angusticeps), which were prepared in a marinade and consumed raw. The symptoms were severe but not life threatening. Scattered reports of intoxications by these species and *M. conica* have appeared in anecodotal reports for many years.

Numerous other cases exist; however, the cases that appear in the literature tend to be the serious poisonings such as those causing more severe gastrointestinal symptoms, psychotropic reactions, and severe organ damage (deadly *Amanita*). Mild intoxications are probably grossly underreported, because of the lack of severity of symptoms and the unlikeliness of a hospital admission.

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

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Aflatoxins

CDC/MMWR NIH/PubMed

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1. Name of Toxin: Aflatoxins

2. Name of Acute Disease: Aflatoxicosis

Aflatoxicosis is poisoning that results from ingestion of aflatoxins in contaminated food or feed. The aflatoxins are a group of structurally related toxic compounds produced by certain strains of the fungi *Aspergillus flavus* and *A. parasiticus*. Under favorable conditions of temperature and humidity, these fungi grow on certain foods and feeds, resulting in the production of aflatoxins. The most pronounced contamination has been encountered in tree nuts, peanuts, and other oilseeds, including corn and cottonseed. The major aflatoxins of concern are designated B1, B2, G1, and G2. These toxins are usually found together in various foods and feeds in various proportions; however, aflatoxin B1 is usually predominant and is the most toxic. When a commodity is analyzed by thin-layer chromatography, the aflatoxins separate into the individual components in the order given above; however, the first two fluoresce blue when viewed under ultraviolet light and the second two fluoresce green. Aflatoxin M a major metabolic product of aflatoxin B1 in animals and is usually excreted in the milk and urine of dairy cattle and other mammalian species that have consumed aflatoxin-contaminated food or feed.

3. Nature of Disease: Aflatoxins produce acute necrosis, cirrhosis, and carcinoma of the liver in a number of animal species; no animal species is resistant to the acute toxic effects of aflatoxins; hence it is logical to assume that humans may be similarly affected. A wide variation in LD50 values has been obtained in animal species tested with single doses of aflatoxins. For most species, the LD50 value ranges from 0.5 to 10 mg/kg body weight. Animal species respond differently in their susceptibility to the chronic and acute toxicity of aflatoxins. The toxicity can be influenced by environmental factors, exposure level, and duration of exposure, age, health, and nutritional status of diet. Aflatoxin B1 is a very potent carcinogen in many species, including nonhuman primates, birds, fish, and rodents. In each species, the liver is the primary target organ of acute injury. Metabolism plays a major role in determining the toxicity of aflatoxin B1; studies show that this aflatoxion requires metabolic activation to exert its carcinogenic effect, and these effects can be modified by induction or inhibition of the mixed function oxidase system.

4. Normal Course of Disease:

In well-developed countries, aflatoxin contamination rarely occurs in foods at levels that cause acute aflatoxicosis in humans. In view of this, studies on human toxicity from ingestion of aflatoxins have focused on their carcinogenic potential. The relative susceptibility of humans to aflatoxins is not known, even though epidemiological studies in Africa and Southeast Asia, where there is a high incidence of hepatoma, have revealed an association between cancer incidence and the aflatoxin content of the diet. These studies have not proved a cause-effect relationship, but the evidence suggests an association.

One of the most important accounts of aflatoxicosis in humans occurred in more than 150 villages in adjacent districts of two neighboring states in northwest India in the fall of 1974. According to one report of this outbreak, 397 persons were affected and 108 persons died. In this outbreak, contaminated corn was the major dietary constituent, and aflatoxin levels of 0.25 to 15 mg/kg were found. The daily aflatoxin B1 intake was estimated to have been at least 55 ug/kg body weight for an undetermined number of days. The patients experienced high fever, rapid progressive jaundice, edema of the limbs, pain, vomiting, and swollen livers. One investigator reported a peculiar and very notable feature of the outbreak: the appearance of signs of disease in one village population was preceded by a similar disease in domestic dogs, which was usually fatal. Histopathological examination of humans showed extensive bile duct proliferation and periportal fibrosis of the liver together with gastrointestinal hemorrhages. A 10-year follow-up of the Indian outbreak found the survivors fully recovered with no ill effects from the experience.

A second outbreak of aflatoxicosis was reported from Kenya in 1982. There were 20 hospital admissions with a 60% mortality; daily aflatoxin intake was estimated to be at least 38 ug/kg body weight for an undetermined number of days.

In a deliberate suicide attempt, a laboratory worker ingested 12 ug/kg body weight of aflatoxin B1 per day over a 2-day period and 6 months later, 11 ug/kg body weight per day over a 14-day period. Except for transient rash, nausea and headache, there were no ill effects; hence, these levels may serve as possible no-effect levels for aflatoxin B1 in humans. In a 14-year follow-up, a physical examination and blood chemistry, including tests for liver function, were normal.

5. Diagnosis of Human Illnesses:

Aflatoxicosis in humans has rarely been reported; however, such cases are not always recognized. Aflatoxicosis may be suspected when a disease outbreak exhibits the following characteristics:

- the cause is not readily identifiable
- the condition is not transmissible
- syndromes may be associated with certain batches of food
- treatment with antibiotics or other drugs has little effect
- the outbreak may be seasonal, i.e., weather conditions may affect mold growth.

The adverse effects of aflatoxins in animals (and presumably in humans) have been categorized in two general forms.

A. (Primary) Acute aflatoxicosis is produced when moderate to high levels of aflatoxins are consumed. Specific, acute episodes of disease ensue may include hemorrhage, acute liver damage, edema, alteration in digestion, absorption and/or metabolism of nutrients, and possibly death.

B. (Primary) Chronic aflatoxicosis results from ingestion of low to moderate levels of aflatoxins. The effects are usually subclinical and difficult to recognize. Some of the common symptoms are impaired food conversion and slower rates of growth with or without the production of an overt aflatoxin syndrome.

7. Associated Foods: In the United States, aflatoxins have been identified in corn and corn products, peanuts and peanut products, cottonseed, milk, and tree nuts such as Brazil nuts, pecans, pistachio nuts, and walnuts. Other grains and nuts are susceptible but less prone to contamination.

8. Relative Frequency of Disease: The relative frequency of aflatoxicosis in humans in the United States is not known. No outbreaks have been reported in humans. Sporadic cases have been reported in animals.

9. Target Populations: Although humans and animals are susceptible to the effects of acute aflatoxicosis, the chances of human exposure to acute levels of aflatoxin is remote in well-developed countries. In undeveloped countries, human susceptibility can vary with age, health, and level and duration of exposure.

9. Analysis of Foods: Many chemical procedures have been developed to identify and measure aflatoxins in various commodities. The basic steps include extraction, lipid removal, cleanup, separation and quantification. Depending on the nature of the commodity, methods can sometimes be simplified by omitting unnecessary steps. Chemical methods have been developed for peanuts, corn, cottonseed, various tree nuts, and animal feeds. Chemical methods for aflatoxin in milk and dairy products are far more sensitive than for the above commodities because the aflatoxin M animal metabolite is usually found at much lower levels (ppb and ppt). All collaboratively studied methods for aflatoxin analysis are described in Chapter 26 of the AOAC Official Methods of Analysis.

10. Outbreaks: Very little information is available on outbreaks of aflatoxicosis in humans because medical services are less developed in the areas of the world where high levels of contamination of aflatoxins occur in foods, and, therefore, many cases go unnoticed.

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

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Pyrrolizidine Alkaloids

NIH/PubMed

1. Name of Toxin:	Pyrrolizidine Alkaloids
2. Name of Acute Disease:	Pyrrolizidine Alkaloids Poisoning Pyrrolizidine alkaloid intoxication is caused by consumption of plant material containing these alkaloids. The plants may be consumed as food, for medicinal purposes, or as contaminants of other agricultural crops. Cereal crops and forage crops are sometimes contaminated with pyrrolizidine-producing weeds, and the alkaloids find their way into flour and other foods, including milk from cows feeding on these plants. Many plants from the Boraginaceae, Compositae, and Leguminosae families contain well over 100 hepatotoxic pyrrolizidine alkaloids.
3. Normal Course of Disease	Most cases of pyrrolizidine alkaloid toxicity result in moderate to severe liver damage. Gastrointestinal symptoms are usually the first sign of intoxication, and consist predominantly of abdominal pain with vomiting and the development of ascites. Death may ensue from 2 weeks to more than 2 years after poisoning, but patients may recover almost completely if the alkaloid intake is discontinued and the liver damage has not been too severe.
4. Diagnosis of Human Illness:	Evidence of toxicity may not become apparent until sometime after the alkaloid is ingested. The acute illness has been compared to the Budd-Chiari syndrome (thrombosis of hepatic veins, leading to liver enlargement, portal hypertension, and ascites). Early clinical signs include nausea and acute upper gastric pain, acute abdominal distension with prominent dilated veins on the abdominal wall, fever, and biochemical evidence of liver disfunction. Fever and jaundice may be present. In some cases the lungs are affected; pulmonary edema and pleural effusions have been observed. Lung damage may be prominent and has been fatal. Chronic illness from ingestion of small amounts of the alkaloids over a long period proceeds through fibrosis of the liver to cirrhosis, which is indistinguishable from cirrhosis of other etiology.
5. Associated Foods:	The plants most frequently implicated in pyrrolizidine poisoning are members of the Borginaceae, Compositae, and Leguminosae families. Consumption of the alkaloid-containing plants as food, contaminants of food, or as medicinals has occurred.

6. Relative Frequency of Disease: Reports of acute poisoning in the United States among humans are relatively rare. Most result from the use of medicinal preparations as home remedies. However, intoxications of range animals sometimes occur in areas under drought stress, where plants containing alkaloids are common. Milk from dairy animals can become contaminated with the alkaloids, and alkaloids have been found in the honey collected by bees foraging on toxic plants. Mass human poisonings have occurred in other countries when cereal crops used to prepare food were contaminated with seeds containing pyrrolizidine alkaloid.

7. Target Population: All humans are believed to be susceptible to the hepatotoxic pyrrolizidine alkaloids. Home remedies and consumption of herbal teas in large quantities can be a risk factor and are the most likely causes of alkaloid poisonings in the United States.

8. Analysis in Foods: The pyrrolizidine alkaloids can be isolated from the suspect commodity by any of several standard alkaloid extraction procedures. The toxins are identified by thin layer chromatography. The pyrrolizidine ring is first oxidized to a pyrrole followed by spraying with Ehrlich reagent, which gives a characteristic purple spot. Gas-liquid chromatographic and mass spectral methods also are available for identifying the alkaloids.

9. Selected Outbreaks: There have been relatively few reports of human poisonings in the United States. Worldwide, however, a number of cases have been documented. Most of the intoxications in the USA involved the consumption of herbal preparations either as a tea or as a medicine. The first patient diagnosed in the USA was a female who had used a medicinal tea for 6 months while in Ecuador. She developed typical hepatic veno-occlusive disease, with voluminous ascites, centrilobular congestion of the liver, and increased portal vein pressure. Interestingly, the patient completely recovered within one year after ceasing to consume the tea. Another herbal tea poisoning occurred when Senecio longilobus was mistaken for a harmless plant (called "gordolobo yerba" by Mexican Americans) and used to make herbal cough medicine. Two infants were given this medication for several days. The 2-month-old boy was ill for 2 weeks before being admitted to the hospital and died 6 days later. His condition was first diagnosed as Reye's syndrome, but was changed when jaundice, ascites, and liver necrosis were observed. The second child, a 6-month-old female, had acute hepatocellular disease, ascites, portal hypertension, and a right pleural effusion. The patient improved with treatment; however, after 6 months, a liver biopsy revealed extensive hepatic fibrosis, progressing to cirrhosis over 6 months. Another case of hepatic veno-occlusive disease was described in a 47-year-old nonalcoholic woman who had consumed large quantities of comfrey (Symphytum species) tea and pills for more than one year. Liver damage was still present 20 months after the comfrey consumption ceased.

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

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Phytohaemagglutinin

1. Name of the Toxin:
Phytohaemagglutinin (Kidney Bean Lectin)

This compound, a lectin or hemagglutinin, has been used by immunologists for years to trigger DNA synthesis in T lymphocytes, and more recently, to activate latent human immunodeficiency virus type 1 (HIV-1, AIDS virus) from human peripheral lymphocytes. Besides inducing mitosis, lectins are known for their ability to agglutinate many mammalian red blood cell types, alter cell membrane transport systems, alter cell permeability to proteins, and generally interfere with cellular metabolism.

2. Name of the Acute Disease:

Red Kidney Bean (*Phaseolus vulgaris*) Poisoning, Kinkoti Bean Poisoning, and possibly other names.

3. Nature of the Acute Disease:

The onset time from consumption of raw or undercooked kidney beans to symptoms varies from between 1 to 3 hours. Onset is usually marked by extreme nausea, followed by vomiting, which may be very severe. Diarrhea develops somewhat later (from one to a few hours), and some persons report abdominal pain. Some persons have been hospitalized, but recovery is usually rapid (3 - 4 h after onset of symptoms) and spontaneous.

4. Diagnosis of Human Illness:

Diagnosis is made on the basis of symptoms, food history, and the exclusion of other rapid onset food poisoning agents (e.g., *Bacillus cereus*, *Staphylococcus aureus*, arsenic, mercury, lead, and cyanide).

5. Foods in Which It Occurs:

Phytohaemagglutinin, the presumed toxic agent, is found in many species of beans, but it is in highest concentration in red kidney beans (*Phaseolus vulgaris*). The unit of toxin measure is the hemagglutinating unit (hau). Raw kidney beans contain from 20,000 to 70,000 hau, while fully cooked beans contain from 200 to 400 hau. White kidney beans, another variety of *Phaseolus vulgaris*, contain about one-third the amount of toxin as the red variety; broad beans (*Vicia faba*) contain 5 to 10% the amount that red kidney beans contain.

The syndrome is usually caused by the ingestion of raw, soaked kidney beans, either alone or in salads or casseroles. As few as four or five raw beans can trigger symptoms. Several outbreaks have been associated with "slow cookers" or crock pots, or in casseroles which had not reached a high enough internal temperature to destroy the glycoprotein lectin. It has been shown that heating to 80°C may potentiate the toxicity five-fold, so that these beans are

more toxic than if eaten raw. In studies of casseroles cooked in slow cookers, internal temperatures often did not exceed 75°C.

6. Frequency of the Disease:

This syndrome has occurred in the United Kingdom with some regularity. Seven outbreaks occurred in the U.K. between 1976 and 1979 and were reviewed (Noah et al. 1980. Br. Med. J. 19 July, 236-7). Two more incidents were reported by Public Health Laboratory Services (PHLS), Colindale, U.K. in the summer of 1988. Reports of this syndrome in the United States are anecdotal and have not been formally published.

7. Usual Course of the Disease and Some Complications:

The disease course is rapid. All symptoms usually resolve within several hours of onset. Vomiting is usually described as profuse, and the severity of symptoms is directly related to the dose of toxin (number of raw beans ingested). Hospitalization has occasionally resulted, and intravenous fluids may have to be administered. Although of short duration, the symptoms are extremely debilitating.

8. Target Populations:

All persons, regardless of age or gender, appear to be equally susceptible; the severity is related only to the dose ingested. In the seven outbreaks mentioned above, the attack rate was 100%.

9. Analysis of Food:

The difficulty in food analysis is that this syndrome is not well known in the medical community. Other possible causes must be eliminated, such as *Bacillus cereus*, *staphylococcal* food poisoning, or chemical toxicity. If beans are a component of the suspected meal, analysis is quite simple, and based on hemagglutination of red blood cells (hau).

10. Selected Outbreaks:

As previously stated, no major outbreaks have occurred in the U.S. Outbreaks in the U.K. are far more common. The syndrome is probably sporadic, affecting small numbers of persons or individuals, and is easily misdiagnosed or never reported due to the short duration of symptoms. Differences in reporting between the U.S. and U.K. may be attributed to greater use of dried kidney beans in the U.K., or better physician awareness. The U.K. has established a reference laboratory for the quantitation of hemagglutinins from suspected foods.

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

11. Education:

NOTE: The following procedure has been recommended by the PHLS to render kidney, and other, beans safe for consumption:

Soak in water for at least 5 hours.
Pour away the water.
Boil briskly in fresh water for at least 10 minutes.
Undercooked beans may be more toxic than raw beans.

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Grayanotoxin

NIH/PubMed

1. Name of Toxin: Grayanotoxin (formerly known as andromedotoxin, acetylandromedol, and rhodotoxin)

2. Name of Acute Disease: Honey Intoxication

Honey intoxication is caused by the consumption of honey produced from the nectar of rhododendrons. The grayanotoxins cause the intoxication. The specific grayanotoxins vary with the plant species. These compounds are diterpenes, polyhydroxylated cyclic hydrocarbons that do not contain nitrogen. Other names associated with the disease is rhododendron poisoning, mad honey intoxication or grayanotoxin poisoning.

3. Nature of Disease: The intoxication is rarely fatal and generally lasts for no more than 24 hours. Generally the disease induces dizziness, weakness, excessive perspiration, nausea, and vomiting shortly after the toxic honey is ingested. Other symptoms that can occur are low blood pressure or shock, bradyarrhythima (slowness of the heart beat associated with an irregularity in the heart rhythm), sinus bradycardia (a slow sinus rhythm, with a heart rate less than 60), nodal rhythm (pertaining to a node, particularly the atrioventricular node), Wolff-Parkinson-White syndrome (anomalous atrioventricular excitation) and complete atrioventricular block.

4. Normal Course of the Disease: The grayanotoxins bind to sodium channels in cell membranes. The binding unit is the group II receptor site, localized on a region of the sodium channel that is involved in the voltage-dependent activation and inactivation. These compounds prevent inactivation; thus, excitable cells (nerve and muscle) are maintained in a state of depolarization, during which entry of calcium into the cells may be facilitated. This action is similar to that exerted by the alkaloids of veratrum and aconite. All of the observed responses of skeletal and heart muscles, nerves, and the central nervous system are related to the membrane effects.

Because the intoxication is rarely fatal and recovery generally occurs within 24 hours, intervention may not be required. Severe low blood pressure usually responds to the administration of fluids and correction of bradycardia; therapy with vasopressors (agents that stimulate contraction of the muscular tissue of the capillaries and arteries) is only rarely required. Sinus bradycardia and conduction defects usually respond to atropine therapy; however, in at least one instance the use of a temporary pacemaker was required.

5. Diagnosis of Human Illness: In humans, symptoms of poisoning occur after a dose-dependent latent period of a few minutes to two or more hours and include salivation, vomiting, and both circumoral (around or near the mouth) and extremity paresthesia (abnormal sensations). Pronounced low blood pressure and sinus bradycardia develop. In severe intoxication, loss of coordination and progressive muscular weakness result. Extrasystoles (a premature contraction of the heart that is independent of the normal rhythm and arises in response to an impulse in some part of the heart other than the sinoatrial node; called also premature beat) and ventricular tachycardia (an abnormally rapid ventricular rhythm with aberrant ventricular excitation, usually in excess of 150 per minute) with both atrioventricular and intraventricular conduction disturbances also may occur. Convulsions are reported occasionally.

6. Associated Foods: Grayanotoxin poisoning most commonly results from the ingestion of grayanotoxin-contaminated honey, although it may result from the ingestion of the leaves, flowers, and nectar of rhododendrons. Not all rhododendrons produce grayanotoxins. Rhododendron ponticum grows extensively on the mountains of the eastern Black Sea area of Turkey. This species has been associated with honey poisoning since 401 BC. A number of toxin species are native to the United States. Of particular importance are the western azalea (*Rhododendron occidentale*) found from Oregon to southern California, the California rosebay (*Rhododendron macrophyllum*) found from British Columbia to central California, and *Rhododendron albiflorum* found from British Columbia to Oregon and in Colorado. In the eastern half of the United States grayanotoxin-contaminated honey may be derived from other members of the botanical family Ericaceae, to which rhododendrons belong. Mountain laurel (*Kalmia latifolia*) and sheep laurel (*Kalmia angustifolia*) are probably the most important sources of the toxin.

7. Relative Frequency of Disease: Grayanotoxin poisoning in humans is rare. However, cases of honey intoxication should be anticipated everywhere. Some may be ascribed to a increase consumption of imported honey. Others may result from the ingestion of unprocessed honey with the increased desire of natural foods in the American diet.

8. Target Population: All people are believed to be susceptible to honey intoxication. The increased desire of the American public for natural (unprocessed) foods, may result in more cases of grayanotoxin poisoning. Individuals who obtain honey from farmers who may have only a few hives are at increased risk. The pooling of massive quantities of honey during commercial processing generally dilutes any toxic substance.

9. Analysis in Foods: The grayanotoxins can be isolated from the suspect commodity by typical extraction procedures for naturally occurring terpenes. The toxins are identified by thin layer chromatography.

10. Selected Outbreaks: Several cases of grayanotoxin poisonings in humans have been documented in the 1980s. These reports come from Turkey and Austria. The Austrian case resulted from the consumption of honey that was brought back from a visit to Turkey. From 1984 to 1986, 16 patients were treated for honey intoxication in Turkey. The symptoms started approximately 1 h after 50 g of honey was consumed. In an average of 24 h, all of the patients recovered. The case in Austria resulted in cardiac arrhythmia, which required a temporal pacemaker to prevent further decrease in heart rate. After a few hours, pacemaker

simulation was no longer needed. The Austrian case shows that with increased travel throughout the world, the risk of grayanotoxin poisoning is possible outside the areas of Ericaceae-dominated vegetation, namely, Turkey, Japan, Brazil, United States, Nepal, and British Columbia. In 1983 several British veterinarians reported a incident of grayanotoxin poisoning in goats. One of the four animals died. Post-mortem examination showed grayanotoxin in the rumen contents.

For more information on recent outbreaks see the Morbidity and Mortality Weekly Reports from CDC.

CDC/MMWR

The CDC/MMWR link will provide a list of Morbidity and Mortality Weekly Reports at CDC relating to this organism or toxin. The date shown is the date the item was posted on the Web, not the date of the MMWR. The summary statement shown are the initial words of the overall document. The specific article of interest may be just one article or item within the overall report.

NIH/PubMed

The NIH/PubMed button at the top of the page will provide a list of research abstracts contained in the National Library of Medicine's MEDLINE database for this organism or toxin.

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Infective Dose Information

Most chapters include a statement on infectious dose. These numbers should be viewed with caution for any of the following reasons:

- Often they were extrapolated from epidemiologic investigations.
- They were obtained by human feeding studies on healthy, young adult volunteers.
- They are best estimates based on a limited data base from outbreaks.
- They are worst case estimates.
- Because of the following variables they cannot be directly used to assess risk:

Variables of the Parasite or Microorganism

- Variability of gene expression of multiple pathogenic mechanism(s)
- Potential for damage or stress of the microorganism.
- Interaction of organism with food menstruum and environment
- pH susceptibility of organism
- Immunologic "uniqueness" of the organism
- Interactions with other organisms

Variables of the Host

- Age
- General health
- Pregnancy
- Medications--OTC or prescription
- Metabolic disorders
- Alcoholism, cirrhosis, hemochromatosis
- Malignancy
- Amount of food consumed
- Gastric acidity variation: antacids, natural variation, achlorhydria
- Genetic disturbances
- Nutritional status
- Immune competence
- Surgical history
- Occupation

Because of the complexity of factors involved in making risk decisions, the multidisciplinary Health Hazard Evaluation Board judges each situation on all available facts.

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Onset, Duration, and Symptoms of Foodborne Illness

Approximate onset time to symptoms	Predominant symptoms	Associated organism or toxin
Upper gastrointestinal tract symptoms (nausea, vomiting) occur first or predominate		
Less than 1 h	Nausea, vomiting, unusual taste, burning of mouth.	Metallic salts
1-2 h	Nausea, vomiting, cyanosis, headache, dizziness, dyspnea, trembling, weakness, loss of consciousness.	Nitrites
1-6 h mean 2-4 h	Nausea, vomiting, retching, diarrhea, abdominal pain, prostration.	*Staphylococcus aureus* and its enterotoxins
8-16 h (2-4 h emesis possible)	Vomiting, abdominal cramps, diarrhea, nausea.	*Bacillus cereus*
6-24 h	Nausea, vomiting, diarrhea, thirst, dilation of pupils, collapse, coma.	Amanita species mushrooms
Sore throat and respiratory symptoms occur		
12-72 h	Sore throat, fever, nausea, vomiting, rhinorrhea, sometimes a rash.	*Streptococcus pyogenes*
2-5 days	Inflamed throat and nose, spreading grayish exudate, fever, chills, sore throat, malaise, difficulty in swallowing, edema of cervical lymph node.	*Corynebacterium diphtheriae*
Lower gastrointestinal tract symptoms (abdominal cramps, diarrhea) occur first or predominate		

2-36 h, mean 6-12 h	Abdominal cramps, diarrhea, putrefactive diarrhea associated with *C. perfringens*, sometimes nausea and vomiting.	*Clostridium perfringens, Bacillus cereus, Streptococcus faecalis, S. faecium*
12-74 h, mean 18-36 h	Abdominal cramps, diarrhea, vomiting, fever, chills, malaise, nausea, headache, possible. Sometimes bloody or mucoid diarrhea, cutaneous lesions associated with V. vulnificus. Yersinia enterocolitica mimics flu and acute appendicitis.	*Salmonella* species (including *S. arizonae*), *Shigella*, enteropathogenic *Escherichia coli*, other *Enterobacteriacae*, *Vibrio parahaemolyticus*, *Yersinia enterocolitica*, ***Pseudomonas aeruginosa*** (?), *Aeromonas hydrophila*, *Plesiomonas shigelloides*, *Campylobacter jejuni*, ***Vibrio cholerae*** (O1 and non-O1) *V.vulnificus*, ***V. fluvialis***
3-5 days	Diarrhea, fever, vomiting abdominal pain, respiratory symptoms.	Enteric viruses
1-6 weeks	Mucoid diarrhea (fatty stools) abdominal pain, weight loss.	*Giardia lamblia*
1 to several weeks	Abdominal pain, diarrhea, constipation, headache, drowsiness, ulcers, variable -- often asymptomatic.	*Entamoeba histolytica*
3-6 months	Nervousness, insomnia, hunger pains, anorexia, weight loss, abdominal pain, sometimes gastroenteritis.	*Taenia saginata, T. solium*
Neurological symptoms (visual disturbances, vertigo, tingling, paralysis) occur		
Less than 1 h	*** SEE GASTROINTESTINAL AND/OR NEUROLOGIC SYMPTOMS (Shellfish Toxins) (this Appendix)	Shellfish toxin
	Gastroenteritis, nervousness, blurred vision, chest pain, cyanosis, twitching, convulsions.	Organic phosphate
	Excessive salivation, perspiration, gastroenteritis, irregular pulse, pupils constricted, asthmatic breathing.	Muscaria-type mushrooms

	Tingling and numbness, dizziness, pallor, gastro- hemmorrhage, and desquamation of skin, fixed eyes, loss of reflexes, twitching, paralysis.	Tetradon (tetrodotoxin) toxins
1-6 h	Tingling and numbness, gastroenteritis, dizziness, dry mouth, muscular aches, dilated pupils, blurred vision, paralysis.	Ciguatera toxin
	Nausea, vomiting, tingling, dizziness, weakness, anorexia, weight loss, confusion.	Chlorinated hydrocarbons
2 h to 6 days, usually 12-36 h	Vertigo, double or blurred vision, loss of reflex to light, difficulty in swallowing. speaking, and breathing, dry mouth, weakness, respiratory paralysis.	*Clostridium botulinum* and its neurotoxins
More than 72 h	Numbness, weakness of legs, spastic paralysis, impairment of vision, blindness, coma.	Organic mercury
	Gastroenteritis, leg pain, ungainly high-stepping gait, foot and wrist drop.	Triorthocresyl phosphate
Allergic symptoms (facial flushing, itching) occur		
Less than 1 h	Headache, dizziness, nausea, vomiting, peppery taste, burning of throat, facial swelling and flushing, stomach pain, itching of skin.	Histamine (scombroid)
	Numbness around mouth, tingling sensation, flushing, dizziness, headache, nausea.	Monosodium glutamate
	Flushing, sensation of warmth, itching, abdominal pain, puffing of face and knees.	Nicotinic acid
Generalized infection symptoms (fever, chills, malaise, prostration, aches, swollen lymph nodes) occur		

4-28 days, mean 9 days	Gastroenteritis, fever, edema about eyes, perspiration, muscular pain, chills, prostration, labored breathing.	*Trichinella spiralis*
7-28 days, mean 14 days	Malaise, headache, fever, cough, nausea, vomiting, constipation, abdominal pain, chills, rose spots, bloody stools.	*Salmonella typhi*
10-13 days	Fever, headache, myalgia, rash.	*Toxoplasma gondii*
10-50 days, mean 25-30 days	Fever, malaise, lassitude, anorexia, nausea, abdominal pain, jaundice.	Etiological agent not yet isolated -- probably viral
Varying periods (depends on specific illness)	Fever, chills, head- or joint ache, prostration, malaise, swollen lymph nodes, and other specific symptoms of disease in question.	*Bacillus anthracis, Brucella melitensis, B. abortus, B. suis, Coxiella burnetii, Francisella tularensis, Listeria monocytogenes, Mycobacterium tuberculosis, Mycobacterium* species, *Pasteurella multocida, Streptobacillus moniliformis, Campylobacter jejuni, Leptospira* species.
Gastrointestinal and/or Neurologic Symptoms - (Shellfish Toxins)		
0.5 to 2 h	Tingling, burning, numbness, drowsiness, incoherent speech, respiratory paralysis	Paralytic Shellfish Poisoning (PSP) (saxitoxins)
2-5 min to 3-4 h	Reversal of hot and cold sensation, tingling; numbness of lips, tongue & throat; muscle aches, dizziness, diarrhea, vomiting	Neurotoxic Shellfish Poisoning (NSP) (brevetoxins)
30 min to 2-3 h	Nausea, vomiting, diarrhea, abdominal pain, chills, fever	Diarrheic Shellfish Poisoning (DSP) (dinophysis toxin, okadaic acid, pectenotoxin, yessotoxin)
24 h (gastrointestinal) to 48 h (neurologic)	Vomiting, diarrhea, abdominal pain, confusion, memory loss, disorientation, seizure, coma	Amnesic Shellfish Poisoning (ASP) (domoic acid)

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Factors Affecting the Growth of Microorganisms in Foods

Food is a chemically complex matrix, and predicting whether, or how fast, microorganisms will grow in any given food is difficult. Most foods contain sufficient nutrients to support microbial growth. Several factors encourage, prevent, or limit the growth of microorganisms in foods, the most important are a_w, pH, and temperature.

a_w: (Water Activity or Water Availability). Water molecules are loosely oriented in pure liquid water and can easily rearrange. When other substances (solutes) are added to water, water molecules orient themselves on the surface of the solute and the properties of the solution change dramatically. The microbial cell must compete with solute molecules for free water molecules. Except for *Staphylococcus aureus*, bacteria are rather poor competitors, whereas molds are excellent competitors.

a_w varies very little with temperature over the range of temperatures that support microbial growth. A solution of pure water has an a_w of 1.00. The addition of solute decreases the a_w to less than 1.00.

Water Activity of Various NaCl Solutions

Percent NaCl (w/v)	Molal	Water Activity (a_w)
0.9	0.15	0.995
1.7	0.30	0.99
3.5	0.61	0.98
7.0	1.20	0.96
10.0	1.77	0.94
13.0	2.31	0.92
16.0	2.83	0.90
22.0	3.81	0.86

The a_w of a solution may dramatically affect the ability of heat to kill a bacterium at a given

temperature. For example, a population of *Salmonella typhimurium* is reduced tenfold in 0.18 minutes at 60°C if the a_w of the suspending medium is 0.995. If the a_w is lowered to 0.94, 4.3 min are required at 60°C to cause the same tenfold reduction.

An a_w value stated for a bacterium is generally the minimum a_w which supports growth. At the minimum aw, growth is usually minimal, increasing as the a_w increases. At a_w values below the minimum for growth, bacteria do not necessarily die, although some proportion of the population does die. The bacteria may remain dormant, but infectious. Most importantly, a_w is only one factor, and the other factors (e.g., pH, temperature) of the food must be considered. It is the interplay between factors that ultimately determines if a bacterium will grow or not. The a_w of a food may not be a fixed value; it may change over time, or may vary considerably between similar foods from different sources.

pH: (hydrogen ion concentration, relative acidity or alkalinity). The pH range of a microorganism is defined by a minimum value (at the acidic end of the scale) and a maximum value (at the basic end of the scale). There is a pH optimum for each microorganism at which growth is maximal. Moving away from the pH optimum in either direction slows microbial growth.

A range of pH values is presented here, as the pH of foods, even those of a similar type, varies considerably. Shifts in pH of a food with time may reflect microbial activity, and foods that are poorly buffered (i.e., do not resist changes in pH), such as vegetables, may shift pH values considerably. For meats, the pH of muscle from a rested animal may differ from that of a fatigued animal.

A food may start with a pH which precludes bacterial growth, but as a result of the metabolism of other microbes (yeasts or molds), pH shifts may occur and permit bacterial growth.

Temperature. Temperature values for microbial growth, like pH values, have a minimum and maximum range with an optimum temperature for maximal growth. The rate of growth at extremes of temperature determines the classification of an organism (e.g., psychrotroph, thermotroph). The optimum growth temperature determines its classification as a thermophile, mesophile, or psychrophile.

INTERPLAY OF FACTORS AFFECTING MICROBIAL GROWTH IN FOODS: Although each of the major factors listed above plays an important role, the interplay between the factors ultimately determines whether a microorganism will grow in a given food. Often, the results of such interplay are unpredictable, as poorly understood synergism or antagonism may occur. Advantage is taken of this interplay with regard to preventing the outgrowth of *C. botulinum*. Food with a pH of 5.0 (within the range for *C. botulinum*) and an a_w of 0.935 (above the minimum for *C. botulinum*) may not support the growth of this bacterium. Certain processed cheese spreads take advantage of this fact and are therefore shelf stable at room temperature even though each individual factor would permit the outgrowth of *C. botulinum*.

Therefore, predictions about whether or not a particular microorganism will grow in a food can, in general, only be made through experimentation. Also, many microorganisms do not need to multiply in food to cause disease.

Factors affecting growth of pathogens in foods.

pH values of some foods

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pH Values of Various Foods

VEGETABLES	pH
Artichokes	5.6
Canned	5.7 - 6
Asparagus	4 - 6
Canned	5.2 - 5.3
Buds	6.7
Stalks	6.1
Beans	5.7 - 6.2
String	4.6
Lima	6.5
Kidney	5.4 - 6
Beets	4.9 - 5.6
S	4.2 - 4.4
Canned	4.9
Brussel sprouts	6.0 - 6.3
Cabbage	5.2 - 6.0
Green	5.4 - 6.9
White	6.2
Red	5.4 - 6.0
Savoy	6.3
Carrots	4.9 - 5.2
Canned	5.18-5.22
Juice	6.4
Cauliflower	5.6
Celery	5.7 - 6.0
Chives	5.2 - 6.1
Corn	6.0 - 7.5
Canned	6.0
Sweet	7.3
Cucumbers	5.1 - 5.7
Dill pickles	3.2 - 3.5
Eggplant	4.5 - 5.3
Hominy (cooked)	6.0
Horseradish	5.35
Kale (cooked)	6.4 - 6.8
Kohlrabi (cooked)	5.7 - 5.8
Leeks	5.5 - 6.0
Lettuce	5.8 - 6.0
Lentils (cooked)	6.3 - 6.8
Mushrooms (cooked)	6.2
Okra (cooked)	5.5 - 6.4
Olives (green)	3.6 - 3.8
(ripe)	6.0 - 6.5
Onions (red)	5.3 - 5.8
(white)	5.4 - 5.8
(yellow)	5.4 - 5.6
Parsley	5.7 - 6.0
Parsnip	5.3
Peas	5.8 - 7.0
Frozen	6.4 - 6.7
Canned	5.7 - 6.0
Dried	6.5 - 6.8
Pepper	5.15
Pimiento	4.6 - 4.9
Potatoes	6.1
Tubers	5.7
Sweet	5.3 - 5.6
Pumpkin	4.8 - 5.2
Radishes (red)	5.8 - 6.5
(white)	5.5 - 5.7
Rhubarb	3.1 - 3.4
Canned	3.4
Rice (all cooked)	
Brown	6.2 - 6.7
White	6.0 - 6.7
Wild	6.0 - 6.4
Sauerkraut	3.4 - 3.6
Sorrel	3.7
Spinach	5.5 - 6.8
Cooked	6.6 - 7.2
Frozen	6.3 - 6.5
Squash (all cooked)	
Yellow	5.8 - 6.0
White	5.5 - 5.7
Hubbard	6.0 - 6.2
Tomatoes (whole)	4.2 - 4.9
Paste	3.5 - 4.7
Canned	3.5 - 4.7
Juice	4.1 - 4.2
Turnips	5.2 - 5.5
Zucchini (cooked)	5.8 - 6.1
FRUITS	
Apples	
Delicious	3.9
Golden Delicious	3.6
Jonathan	3.33
McIntosh	3.34
Winesap	3.47
Juice	3.4 - 4.0
Sauce	3.3 - 3.6
Apricots	3.3 - 4.0
Dried	3.6 - 4.0
Canned	3.74
Bananas	4.5 - 5.2

FRUITS (contin.)	pH
Cantaloupe	6.17-7.13
Dates	6.3 - 6.6
Figs	4.6
Grapefruit	3.0 - 3.3
Canned	3.1 - 3.3
Juice	3.0
Lemons	2.2 - 2.4
Canned juice	2.3
Limes	1.8 - 2.0
Mangos	3.9 - 4.6
Melons	
Cassaba	5.5 - 6.0
Honey dew	6.3 - 6.7
Persian	6.0 - 6.3
Nectarines	3.9
Oranges	3.1 - 4.1
Juice	3.6 - 4.3
Marmalade	3.0
Papaya	5.2 - 5.7
Peaches	3.4 - 3.6
In jars	4.2
In cans	4.9
Persimmons	5.4 - 5.8
Pineapple	3.3 - 5.2
Canned	3.5
Juice	3.5
Plums	2.8 - 4.6
Pomegranates	3.0
Prunes	3.1 - 5.4
Juice	3.7
Quince (stewed)	3.1 - 3.3
Tangerines	4.0
Watermelon	5.2 - 5.8

BERRIES	
Blackberries	3.2 - 4.5
Blueberries	3.7
Frozen	3.1 - 3.35
Cherries	3.2 - 4.1
Cranberries	
Sauce	2.4
Juice	2.3 - 2.5
Currants (red)	2.9
Gooseberries	2.8 - 3.1
Grapes	3.4 - 4.5
Raspberries	3.2 - 3.7
Strawberries	3.0 - 3.5
Frozen	2.3 - 3.0

MEAT, POULTRY	pH
Beef	
Ground	5.1 - 6.2
Ripened	5.8
Unripened	7.0
Canned	6.6
Tongue	5.9
Ham	5.9 - 6.1
Lamb	5.4 - 6.7
Pork	5.3 - 6.9
Veal	6.0
Chicken	6.5 - 6.7
Turkey (roasted)	5.7 - 6.8
FISH	
Fish (most fresh)	6.6 - 6.8
Clams	6.5
Crabs	7.0
Oysters	4.8 - 6.3
Tuna fish	5.2 - 6.1
Shrimp	6.8 - 7.0
Salmon	6.1 - 6.3
Whitefish	5.5
Freshwater (most)	6.9 - 7.3
Sturgeon	5.5 - 6.0
Herring	6.1 - 6.4
DAIRY PRODUCTS/EGGS	
Butter	6.1 - 6.4
Buttermilk	4.5
Milk	6.3 - 8.5
Acidophilus	4.0
Cream	6.5
Cheeses	
Camembert	7.44
Cheddar	5.9
Cottage	5.0
Cream cheese	4.88
Edam	5.4
Roquefort	5.5 - 5.9
Swiss Gruyer	5.1 - 6.6
Eggs	
White	7.0 - 9.0
Yolk	6.4
Egg solids, whites	6.5 - 7.5
Whole	7.1 - 7.9
Frozen	8.5 - 9.5

BAKERY PRODUCTS	pH
Bread	5.3 - 5.8
Eclairs	4.4 - 4.5
Napoleons	4.4 - 4.5
Biscuits	7.1 - 7.3
Crackers	7.0 - 8.5
Cakes	
Angel food	5.2 - 5.6
Chocolate	7.2 - 7.6
Devil's food	7.5 - 8.0
Pound	6.6 - 7.1

Sponge	7.3 - 7.6
White layer	7.1 - 7.4
Yellow layer	6.7 - 7.1
Flour	6.0 - 6.3
MISCELLANEOUS	
Caviar (domestic)	5.4
Cider	2.9 - 3.3
Cocoa	6.3
Corn syrup	5.0
Corn starch	4.0 - 7.0
Ginger ale	2.0 - 4.0
Honey	3.9
Jams/Jellies	3.1 - 3.5
Mayonnaise	4.2 - 4.5
Molasses	5.0 - 5.5
Raisins	3.8 - 4.0
Sugar	5.0 - 6.0
Vinegar	2.0 - 3.4
Yeast	3.0 - 3.5

[(a)]pH values were derived from the following references:

Anon. 1962. pH values of food products. Food Eng. 34(3):98-99.

Bridges, M.A., and Mattice, M.R. 1939. Over two thousand estimations of the pH of representative foods. Am. J. Digest. Dis. Nutr. 9:440-449.

FDA Bacteriological Analytical Manual, 6th Ed. 1984. Chapter 23, Table 11.

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FOREIGN MATERIALS IN FOODS: ARE THEY REALLY HAZARDOUS?

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INTRODUCTION

Whenever anyone one of us eats or drinks something, we have certain expectations, especially that something that is a favorite. The product should look, smell and taste as expected. How a food is perceived is a combination of many factors. Flavor is a complex of sensations, which include taste, odor, sight, and tactile responses. While color or appearance may not be a component of flavor, they play a role in our response to food. Through the interrelationships of the sensations of taste, odor, and feeling, flavors of foods and beverages are identified in the mouth. Enjoyment of a food frequently is predicated on the appearance of the food and the aroma emitted from the same prior to actual tasting. So, what happens when something unexpected occurs? The unexpected can be an off flavor or the presence of a foreign material in the food. The fortunate ones discover the foreign material before they put the food into their mouth. The unfortunate.....well, everyone has heard the old joke, "What's worse than finding a worm in your apple?....Half a worm.....So, are foreign objects in foods a real area of concern? Are they legitimate food safety hazards????

FOOD SAFETY & PHYSICAL HAZARDS

Almost all HACCP (Hazard Analysis, Critical Control Point) classes taught throughout the United States and the world include a discussion of potential hazards in foods. The focus of this discussion is usually the biological hazards, which as they are the most important in terms of public health, makes complete sense. The lecturer would be remiss if physical hazards were ignored, however. Before entering into a discussion of physical hazards, let's review some of the basics. Food processors are mandated by law to produce safe foods. A safe food may be defined as;

> *a product which contains no physical, chemical, or microbial organisms or by-products of those organisms which if consumed by man will result in illness, injury, or death (an unacceptable consumer health risk.*

The definition purposely does not use the term contaminants because many of the potential hazards in food, which HACCP programs are designed to address, are normally found in or on the food. It is their concentration, numbers, or size that create potential safety problems. We must, therefore, understand what constitutes a hazard. A food hazard may be defined as;

> *Any biological, chemical, or physical property that may cause an unacceptable consumer health risk .*

This definition poses several questions. What are the potential physical hazards in

foods? How might these materials gain access to the processing system or the product? How can potential hazards be detected and removed? Are all foreign materials in foods truly hazards?

SOURCES OF FOREIGN MATERIALS FOODS

It is generally agreed that there are five basic sources of foreign materials in foods. The question still remains as to whether these foreign materials from these sources would be construed potential hazards. These five sources are;

1. Inadvertent from the field (stones, metal, insects, undesirable vegetable matter such as thorns or wood, dirt, or small animals).

2. Inadvertent resulting from processing and handling (bone, glass, metal, wood, nuts, bolts, screening, cloth, grease, paint chips, rust, etc.)

3. Materials entering the food during distribution - insects, metal, dirt, stones, or anything else.

4. Materials intentionally placed in food (employee sabotage)

5. Miscellaneous - struvite and other materials in this class.

The unit operations in processing plants should be designed to remove or eliminate the physical hazards described under numbers 1 and 2. Operations that process raw agricultural commodities, such as fruits and vegetables, or grains and seeds, would be remiss if they did not build such cleaning operations into their lines. In the United States, most crops are mechanically harvested. Mechanical harvesters have one major problem in that they often collect more than just the product. Stones, wire, and small birds, mammals or reptiles can be collected by the harvesters. I once watched a four foot long snake drop out of a load of green beans being deposited in a washer. The workers went one way and the snake the other. This is one of the reasons that processors include destoners, air cleaners, magnets, screens and washers in their lines. Another reason is economic. Removal of stones, scrap metal or other materials protects your investment, the equipment, from damage. Failure to remove a stone from a product before it enters a chopper or slicer could damage the unit, and create a secondary problem; metal in the system. Ironically, in less developed nations where mechanical harvesting is less common, these concerns are less of an a issue. The trade off is that hand harvesting is slower, and may compromise product quality.

Grain processors and manufacturers of four utilize screens throughout the process. The screens help them sift and size product, but they also remove undesirable foreign materials. Cleaning is an integral part of processing agricultural commodities. To understand the differences between handling agricultural products and processed, all one needs to do is look at what is found in raw coffee beans and processed coffee The raw beans may contain stones, metal fragments, and other "surprises."

Inadvertent contaminants from the processing operations may be another potential source of foreign materials. This is one of the reasons that preventive maintenance is

considered a "HACCP Prerequisite." Properly maintained equipment and lines usually do ***not*** cause problems.

The next question is whether there is a potential for contamination during distribution and storage. Distribution and storage practices and in-store handling practices should control and/or be designed to prevent the finished food product from being contaminated or affected. Once the container is sealed, the chances for physical contamination are greatly reduced, particularly if one is dealing with metal, glass, or one of the thermoplastics used for hermetically sealed foods. Food protection is one of the primary functions of the package. All packages should be designed to prevent tampering or be tamper evident. In fact, there is little chance of contamination of any packaged food becoming contaminated once it is in the package. On rare occasions, if grains, flours, dried fruits or other materials packaged in paper or cardboard are stored or held in an area that is infested with insects or rodents, the pests can get into the containers. Examination of infested containers will let an examiner know whether the insects came from the inside or outside. Experts can tell whether an insect chewed his way in or out. The greatest concern with contamination during distribution and storage is with bulk products.

Foreign material contamination resulting from employee sabotage are more insidious and are very difficult to monitor. Controlling employee sabotage is a function of good management and proper employee education. Implementation of an all encompassing quality assurance system whereby employees are educated on good food handling and HACCP principles, so food safety becomes everyone's responsibility can reduce the likelihood of this kind of problem. Management cannot watch everything, but line workers generally know exactly what goes on. These individuals are excellent sources of information. When examining a facility, they can provide an inspector or auditor with a great deal of useful information, provided he or she can gain their confidence.

The miscellaneous contaminants are also insidious. Struvite , an ammonium complex, is a prime example. This hard crystalline material may be formed in canned proteinaceous seafoods. The material resembles glass in appearance to the consumer. They may break a tooth if they bite it, but the material will not cut them like glass. It may, however, be considered a safety hazard.

SO, IS IT REALLY A HAZARD?

This then leads us to the ultimate question where HACCP is concerned. Do foreign materials in foods pose a real food safety hazard? They may cause varying degrees of injury, and in rare cases death. They may cause psychological trauma, some may cause physical illness, and others may never be noted. An example of the latter are insect fragments in a product which has a defect action level for that particular character. The following table describes some of the common foreign materials found in foods, their potential for causing injury and some sources for these materials (Table 1).

TABLE 1

MATERIAL	INJURY POTENTIAL	SOURCES
Glass	Cuts, bleeding; May require surgery to find or remove	Bottles, jars, light fixtures, utensils, gage covers

Wood	Cuts, infection	Fields, pallets, boxes, buildings
Stones	Choking, broken teeth choking, may require surgery to remove	Field, buildings
Metal	Cuts, infection, may require surgery to remove	Wire, employees, machinery, fields,
Insects	Illness, trauma, choking	Fields, plant, post-process entry
Insulation	Choking, long-term if asbestos	Insulation
Bone	Choking, trauma	Fields, plant, improper processing

The question that the HACCP team must ask themselves is ***"Is this a realistic problem, and is there a real chance of injury***?" In 1990, Corlett and Stier proposed a guide for helping processors assess potential chemical and physical hazards in foods. This guide may be seen in Figure 1. It was developed to provide a roadmap towards assessing risk, but was really never utilized following issuance of the National Advisory Committee's updated guidelines in 1992.

The United States Food and Drug Administration recently developed a policy guideline that may help the group answer the question posed above. The Agency has been monitoring injuries resulting from foreign materials since 1972. Over a twenty-five year period, they evaluated 190 cases involving hard or sharp foreign materials in foods. The Agency has developed criteria for determining whether a product is adulterated and could cause injury. Only hard or sharp foreign objects that measured 7 to 25 mm were determined to be hazardous. Objects less than 7 mm were determined to be too small to cause injury and those greater than 25 mm were so large that the Agency felt that there was little chance of a consumer eating the food. So, let's look at the foreign materials listed above and determine whether they would be realistic hazards.

Stones and wood will cause injury. Consumers might break a tooth or cut themselves. Are they realistic hazards, however? Personally, I have never been in a plant where I felt that these were real hazards. Cleaning of incoming materials, the application of good manufacturing practices, the use of screens and sorting in process operations and the nature of the contaminants themselves tend to minimize the potential for product contamination. Insulation could be placed in the same category. The bottom line is that these materials can be controlled by the application of your HACCP prerequisite programs. If you, as a processor, are concerned about these wood or stones, take a look at your consumer complaint files. I would wager that there have been very few, if any, alleged or real complaints regarding these materials.

What about glass? Glass is scary. It is hard to see because it is usually clear. It is sharp and will cause injury, but again, is it a realistic hazard. Processors who pack foods in glass almost always have a program in their plants addressing what should be done in the event of glass breakage. When I worked with juice-in-glass, our policy was to discard all containers within ten feet of any glass that shattered. Containers that had been filled and sealed were washed to remove shards. Our objective was to assure that there were no shards of glass of any size in the product. Prevention of glass contamination is one reason that both the regulations emphasize the importance of shielding lights. If a bulb blows or is broken, glass may fly all over. If there is any exposed product, it should be destroyed. Reconditioning or sorting to remove shards would be expensive and inefficient. Now there, are instruments that can be installed

"on-line" to scan for glass and other materials. These systems utilize x-rays or other sophisticated scanning tools. The problem is they are usually very expensive.

What about metal now? Metal is a common industry concern. The best way to assure that metal is not an issue is good preventive maintenance. Are bolts and screws being loosened by equipment vibration? Or, didn't there used to be a bolt in that hole? Ferrous metal can be removed using magnets that can be placed in-line. If an operation uses magnets, they should be monitored to determine what they are collecting. Metal detectors are becoming more and more common in processing operations in this day and age. In some industries, especially meat processing, metal detectors are almost a necessity. Why you may ask? For some perverse reason, people like shooting at cattle and many end up with pellets imbedded in the muscle. Also, on rare occasions an animal will "flinch" when being given a shot causing the needle to break off below the skin. Metal detectors will find these foreign materials. With increased use, they are becoming more sensitive, easier to maintain and calibrate and easier to use. In fact, there are many processors and buyers who mandate that everything that they manufacture or buy pass through a metal detector. Metal detectors can detect ferrous metals ranging down to 1-2 mm in size; non-ferrous metal sensitivity might be in the 2-3 mm range. Smaller pieces of metal would not be considered hazardous.

Finally, what about insects or insect parts? Is the presence of an insect in a food product a real hazard? If you eat that insect will you become sick? The answer is not really. There are many cultures where insects are considered delicacies. Psychological trauma is not considered to be a physical injury. So, does this mean that we should accept insect contamination in foods? The answer is no. Remember, we are in business to make money and in the food industry, this means repeat sales. The consumer who discovers insects or insect parts in their food will probably not buy that product again. It could also mean a visit from an Agency looking for insanitary operating conditions, or even worse, coverage by the media. The resulting adverse publicity can damage the company's reputation and adversely affect sales.

SUMMARY

As part of the hazard analysis, all food processors need to examine their products and processes to determine whether any biological, chemical or physical hazards exist. Physical hazards may the least common, but cannot be ignored. Evaluation of the potential for contamination of the product with foreign materials and whether those materials pose a realistic hazard should involve the following steps:

1) A plant audit aimed at evaluating systems for pest control, foreign object removal, plant condition, shipping and receiving practices, and plant maintenance procedures.
2) A review of packaging materials and container/package handling procedures, particularly when glass is the packaging material.
3) A review of agricultural practices.
4) A review of personnel practices, including those of maintenance staff.
5) Package evaluation to ensure that it is tamper proof, or tamper evident.

6) A review of consumer complaints to see whether foreign materials have been implicated in illness or injury.

Using these steps to assess physical hazards when developing and implementing a HACCP program should be more than adequate. As noted, the best means for assuring that physical hazards are properly controlled is through the use of a well-designed preventive maintenance program. It is one of the basic HACCP prerequisites.

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ASSESSMENT OF PHYSICAL AND CHEMICAL HAZARDS

HAZARD A: A special class that applies to products designed and intended for consumption by at-risk populations, e.g., infants, the aged, the infirm, or immunocompromised individuals.

Examples: Foods intended for persons who are sensitive to sulfites or those intended for infants and packaged in glass.

HAZARD B: The product contains ingredients known to be potential sources of toxic chemicals or dangerous physical hazards.

Examples: Aflatoxin/fumonisin in field corn, or stones in agricultural products.

HAZARD C: The process does not contain a controlled step(s) that effectively destroys or removes toxic chemicals or physical hazards.

Examples: Removal of lye, or screens for foreign objects.

HAZARD D: The product is subject to recontamination after processing, but before packaging.

Examples: Products bulk packed and shipped elsewhere for final packaging.

HAZARD E: There is substantial potential for chemical or physical recontamination in distribution or in consumer handling that could render the product harmful when consumed.

Examples: Foods transported in containers or vehicles which contained toxic materials or foreign objects; marketing of foods in open containers where the potential for tampering is high.

HAZARD F: There is no way for the consumer to detect, remove, or destroy a chemical or physical hazard.

Examples: Presence of toxic mushrooms or paralytic shellfish poisoning, or with physical hazards, sharp metal fragments buried within the food.

Corlett, D.A. and R.F. Stier (1990)