VIRGINIA
EPIDEMIOLOGY BULLETIN

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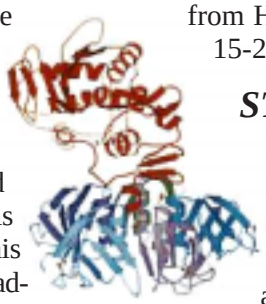
Shiga Toxin-producing *Escherichia coli* (STEC) in Virginia

The Commonwealth of Virginia offers an endless variety of activities to enjoy during the summer, including traveling, swimming, picnics and barbeques. However, what many of these activities have in common is the potential for acquiring a foodborne illness that could spoil the fun. This article reviews one of the more serious foodborne pathogens, Shiga Toxin-producing *Escherichia coli* (STEC), that people may inadvertently encounter.

Escherichia coli and Shiga Toxin

Escherichia coli (*E. coli*) is a normal inhabitant of the intestines of animals and humans, and helps both to suppress the growth of harmful bacterial species and to synthesize vitamins. However, of the over 700 antigenic types (serotypes) of *E. coli*, a few can cause illness in an otherwise healthy host. One set of these organisms are the Shiga Toxin-producing *E. coli* (STEC) that include *E. coli* serotype O157:H7, as well as other non-O157:H7 serotypes.

STEC infections cause a significant amount of illness. In the United States, the Centers for Disease Control and Prevention (CDC) estimates that STEC infects 73,000 people each year and causes over 2,000 hospitalizations and 61 deaths annually.[1] STEC strains cause illness through the production of large quantities of one of two distinct bacteriophage-encoded toxins: Shiga-like toxin 1 and Shiga-like toxin 2. Similar to the toxin produced by *Shigella dysenteriae*, the causative agent of bacillary dysentery, the STEC toxin has B subunits that bind to cell membranes, and allow A subunits to enter cells and inactivate ribosomes. This halts cell protein synthesis leading to cell death, a breakdown of the intestinal lining, and severe bleeding. Shiga-like toxins can also enter the bloodstream and act on the endothelium of small blood vessels, such as those in the digestive tract, the kidneys, and the lungs. In the kidneys, the damage to the vascular endothelium of the renal glomerulus can trigger hemolytic uremic syndrome (HUS).



Shiga Toxin

About 5%-10% of infections with STEC progress to HUS.[2] HUS typically develops in the second week of illness, and is more likely to occur in children < 5 years of age or adults > 60 years of age.[3] This multisystemic disorder characterized by thrombocytopenia, microangiopathic hemolytic anemia, and organ ischemia (e.g., acute renal failure) results from platelet agglutination in the arterial microvasculature. Patients may also develop neurologic impairment (e.g., seizures or stroke). And while the overall case fatality rate of HUS is 3-5%, the elderly are at particular risk, with mortality from HUS in the elderly as high as 15-23%.[4]

STEC in Virginia

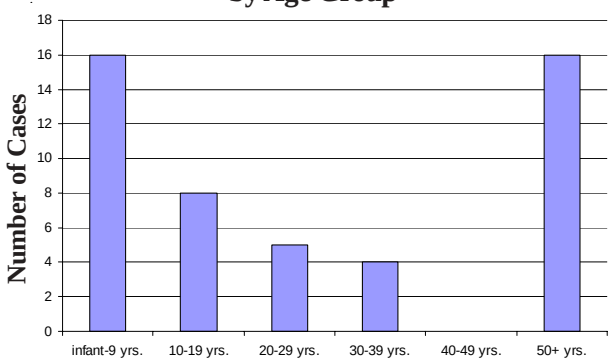
Since 1999 when STEC infection became a **reportable disease** in Virginia, a median of 70 cases have been reported annually. Infants and persons 1-9 years of age in Virginia have been at greatest risk for symptomatic STEC infection (Figure 1) and have averaged 3 cases/100,000 population/year; all other age groups averaged ≤ 1 case/100,000 population/year. Last year Virginia healthcare providers reported 49 cases of STEC infection. At least four of the culture-confirmed cases reported in Virginia in 2003 listed HUS as a complication of infection, and all of the cases of HUS in 2003 were under 5 years of age. One case of HUS was reported in a 3 year old from whom no pathogen was isolated.

STEC infections occur

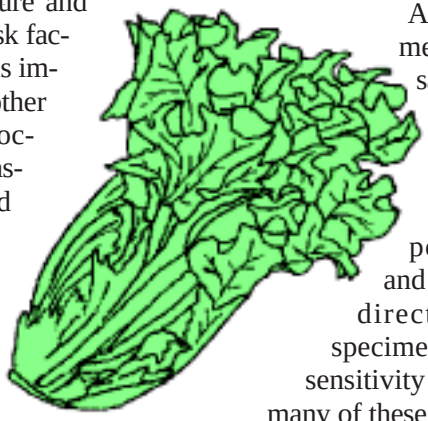
In This Issue:

throughout the year, but peak activity tends to occur during the warmer months (April to September)(Figure 2). This may be related to the mode of transmission; many cases have been associated with eating ground beef contaminated during slaughter that has not been cooked sufficiently to kill the bacteria. As a result, STEC infection used to be referred to as "hamburger disease."

Figure 1. *E coli* O157:H7 cases in Virginia in 2003 by Age Group



However, at least 22% of the cases of *E. coli* O157:H7 reported in Virginia in 2003 were known to have no ground beef exposure in the 8 days prior to illness. Similarly, at least 25 of the 49 reported cases had no known exposure to farm animals in the week prior to illness. Therefore, while both undercooked ground beef exposure and farm visits are significant risk factors for STEC infection[5], it is important to remember that other risks exist. Infection has occurred by drinking raw (unpasteurized) milk contaminated by the bacteria from the cow's udders or on equipment, consumption of foods that can be contaminated by bovine or human feces (e.g., alfalfa sprouts, lettuce), salami, unpasteurized juice and cider, and swimming in or drinking sewage-contaminated water. Because the infectious dose is so low (approximately 100 organisms) person-to-person transmission through the fecal-oral route can occur readily.



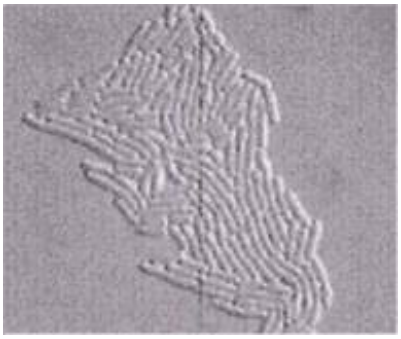
Diagnosis

STEC infection should be considered in any person who has a sudden onset of diarrhea with blood. STEC infection is characterized by severe abdominal cramps and diarrhea that is initially watery but becomes grossly bloody. Occasionally, vomiting occurs. Usually little or no fever is present, however temperatures as high as 39°C (102.2°F) have been reported. In uncomplicated STEC infections, diarrhea usually resolves in 5 to 10 days.

STEC infection is confirmed by detecting the bacterium in the stool. Most laboratories that culture stool do not routinely test for STEC, so it is important to **specifically request testing for STEC so that the laboratory can culture the stool specimen on sorbitol-MacConkey (SMAC) agar**.

A number of commercial immunoassays are also available for the rapid detection of Shiga-like toxins, O157 lipopolysaccharide and other O-antigens directly from stool specimens.[6] Although the sensitivity and specificity of many of these tests can be high, correlations with respect to clinical presentation have not been clearly established.

In addition, all STEC isolates received or isolated by the Virginia Division of Consolidated Laboratory Services (state laboratory) are subjected to pulsed-field gel electrophoresis (PFGE) DNA fingerprinting. DNA fingerprint patterns are compared locally and also electronically uploaded to the PulseNet National Molecular Subtyping Network Database managed by the CDC in Atlanta. Clusters of STEC isolates possessing indistinguishable PFGE DNA fingerprint patterns identified locally or at the national level are immediately reported to VDH for further investigation. **Therefore, even if a rapid Shiga-like toxin test is used, clinicians should also request stool cultures to confirm the patient's diagnosis and to allow for further strain characterization to support public health disease surveillance and outbreak investigations.**



Treatment

For patient management, the key objective is fluid and electrolyte replacement to prevent dehydration. Anti-diarrheal medications are not indicated. For the acute diarrheal illness, antibiotics have not proven useful. The *2003 American Academy of Pediatrics Red Book* states that while some studies have suggested that children with STEC treated with antimicrobial agents have an increased risk of developing HUS, meta-analysis of studies has failed to confirm this increased risk or to show a benefit from antimicrobial agents. Therefore, at the present time most experts would not treat children with STEC enteritis with an antimicrobial agent.[2]

In addition, proper disposal of the feces of infected persons, good hygiene, and careful hand washing with soap and water are important in limiting the further spread of STEC infection.

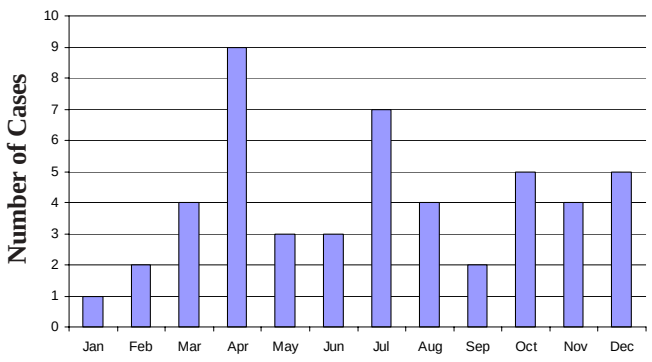
Public Health Response to the Reported Case

All suspected or confirmed cases of STEC infection must be reported to the local health department. Public health follow-up of reported cases of STEC cases attempts to identify the possible source and interrupt further transmission. Therefore, when a case of STEC comes to the attention of a local health department, the healthcare provider associated with the case may be contacted for further information. This will consist of confirming the diagnosis as well as obtaining the date of onset, symptoms, tests performed, and treatment. The healthcare provider may also be asked how much the patient has been told about the illness.

The local health department will then con-

tact the patient or the patient's guardian by telephone or make a home visit in order to conduct an interview and gather information using a standardized questionnaire. This questionnaire is designed to identify risk factors for STEC exposure through a detailed food history and questions about specific food items consumed in the seven days prior to disease onset. The questionnaire includes specific items on ground beef handling and consumption, as well as information related to other risk factors such as contact with children in diapers, recreational water exposure and contact with farm animals.

Figure 2. *E coli* O157:H7 cases per month in Virginia - 2003



Person-to-person contact is an important mode of transmission for STEC. Therefore, the interview with the patient will also help to establish whether the patient is engaged in a profession (e.g., food services, daycare worker) or situation (e.g., daycare attendant) that would pose a high risk for disease transmission. If a high risk situation does not exist, health education will be provided that includes proper handwashing techniques and foodhandling strategies (such as cooking ground beef thoroughly and avoiding cross-contamination of other foods).

If a high risk situation is identified, the same health education will be provided, but the patient will also be excluded from the high risk setting until diarrhea has ceased and, in most instances, until culture negative for STEC. Culture negativity is defined by the health department as **two** successive negative stool cultures taken 24 hours or more apart. If the patient received treatment, the specimens should not be collected any sooner than 48 hours following completion of antimicrobial therapy.

If, during the interview or through the disease reporting process, others are identified as being ill with similar symptoms, an outbreak investigation will be initiated to try to identify the source of the infection. Depending on the circumstances, this may take the form of either a cohort study (if the entire population at risk is known) or a case-control study (where cases are matched with uninfected controls).

Since the risk to children is so high, if more than one case is detected in a daycare the health department will inspect the facility to assess the extent of the contact that cases may have had with other children. Decisions concerning the management of a daycare experiencing a STEC outbreak are made on an individual basis, using the amount of contact that children in the facility have with each other as a guide. In general, children in the same classroom (or the entire facility if the situation warrants) are excluded from the daycare until they submit a negative stool specimen. This is done because typically there will be a high percentage of asymptomatic cases that continue to transmit the organism; young children may shed the organism in their feces for up to two weeks after their illness resolves. Any child with diarrhea or any asymptomatic child who tests positive for STEC would be excluded from the daycare until diarrhea ceases (if ill) and the stool culture is negative. Depending on the facility setup, a cohort system (where infected children and staff are housed together in a separate area away from other children and staff) may be implemented. Because it is critical to prevent the transfer of children to other childcare centers, a letter may be sent to nearby facilities warning them about the situation.

Conclusions

Shiga toxin-producing *Escherichia coli* cause a significant amount of disease in Virginia. Although most people suffer only a self-limited diarrheal illness from these organisms, STEC infections can cause long-term morbidity (e.g., chronic renal failure) and mortality. Primary prevention efforts, such as educating people about appropriate food preparation methods, are very important. However, healthcare providers will continue to have a critical role in preventing STEC-related illness through the rapid reporting of known or suspected cases to the local health department.

For more information on *E. coli* O157:H7 go to the CDC website at http://www.cdc.gov/ncidod/dbmd/diseaseinfo/escherichiacoli_g.htm.

Submitted by:
Julia Murphy, DVM, Division of Zoonotic and Environmental Epidemiology and
Denise Toney, PhD, Division of Consolidated Laboratory Services

References:

1. CDC: http://www.cdc.gov/ncidod/dbmd/diseaseinfo/escherichiacoli_t.htm
2. American Academy of Pediatrics. *Escherichia coli* diarrhea. In: Pickering LK, ed. Red Book: 2003 Report of the Committee on Infectious Diseases. 26th ed. Elk Grove Village, IL: American Academy of Pediatrics; 2003:275-9.
3. "*Escherichia coli* O157:H7 Infection." The Merck Manual of Diagnosis and Therapy. 17th ed. Ed. Mark H. Beers, M.D. and Robert Berkow, M.D. Rathway, NJ: Merck Research Laboratories, 1992: 285-6.
4. Karch H, Brelaszewska M, Brizon M, Schmidt. *Diag Microbiol Infect Dis* 1999; 34: 229-243.
5. Kassenborg HD, Hedberg CW, Hoekstra M, et al. *Clin Infect Dis* 2004 Apr 15;38 Suppl 3:S271-8.
6. Manual of Clinical Microbiology 8th Edition, volume 1. Patrick R. Murray, ed. (2003).

Harmful Algal Blooms (HABs)

Introduction

Phytoplankton are microscopic algae found in marine and fresh water and represent a major source of food and oxygen for wildlife inhabiting lakes, rivers, estuaries, and oceans. Phytoplankton are normally present within these waters in low concentrations but may proliferate to form dense concentrations of cells on water surfaces referred to as "blooms." These blooms are usually not harmful to animals or humans, but the high concentrations of pigment-containing phytoplankton may impart color to the water resulting in their description as "red tides," "brown tides," etc.

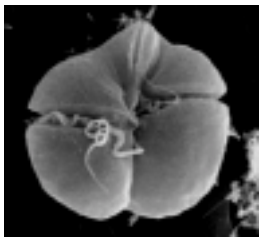


Figure 1. Dinoflagellate

Among the several thousand species of phytoplankton that exist world-wide, approximately 70 to 80 produce toxins. Toxin-producing phytoplankton species usually belong to one of the following phylogenetic categories: dinoflagellates (Figure1), diatoms (Figure 2), cyanobacteria, or raphidophytes. Blooms of toxin-producing algae are referred to as "harmful algal blooms" (HABs) and may pose an environmental, animal, and/or human health threat. At least 16 toxin-producing species have been identified in Virginia's Chesapeake Bay and/or its tributaries, and all have the potential to form blooms. For example, in June 2004, a blue-green toxin-producing algae (cyanobacteria), *Microcystis aeruginosa*, was identified in the Potomac River, leading to the temporary closure of a nearby beach as a public health precaution.

HABs have been documented in the coastal waters of both the eastern and western United States as well as other coastal areas throughout the world. During a HAB, toxin-producing phytoplankton may be consumed by shellfish and/or finfish where toxin is concentrated and, when ingested by humans and other animals, may produce illness. The most widely recognized human illnesses caused by HABs are (Table 1):

- Paralytic shellfish poisoning (PSP)
- Diarrhetic shellfish poisoning (DSP)
- Ciguatera fish poisoning (CFP)
- Neurotoxic shellfish poisoning (NSP)
- Amnestic shellfish poisoning (ASP)

Most of the harmful algae related to these illnesses produce neurotoxins that cause a range of neurological and gastrointestinal symptoms depending on the toxin produced. In addition, inhalation of certain aerosolized toxins associated with HABs has been implicated as a cause of respiratory irritation in humans.

One of the more recently identified microorganisms in Chesapeake Bay estuaries and other locations on the eastern coast of the United States is *Pfiesteria piscicida*. This organism may be linked to human illness characterized by fatigue, headache, respiratory irritation, skin lesions, disorientation, memory loss, and impairment of cognitive function. *P. piscicida* has also been associated with fish kills as well as ulcerative lesions on fish skin. The possibility that *P. piscicida* produces a toxin that may cause disease in humans and fish is under investigation. In addition, research on this organism has led to the discovery of other related phytoplankton species.

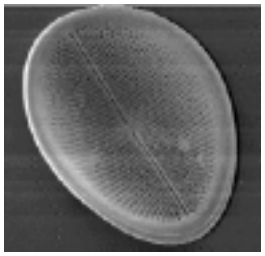


Figure 2. Diatom

This article will further describe the most widely recognized illnesses caused by HABS, their diagnosis and treatment, and monitoring for HABs in Virginia's coastal water and tributaries.

Illnesses Caused by Toxins Produced by HABs

Paralytic Shellfish Poisoning (PSP)

PSP has been reported worldwide for centuries. Globally, nearly 2,000 cases of PSP are reported annually with average mortality rates around 15%, but ranging from 9-50%. Unicellular dinoflagellates (*Alexandrium* spp., *Gymnodinium* spp., and *Pyrodinium* spp.) produce at least 12 heat and acid stable toxins which block neuronal and muscular sodium channels primarily in the peripheral nervous system. Saxotoxin (*Alexandrium* spp.) was the first identified and is the best characterized of the PSP toxins and may cause both neurologic and gastrointestinal illness in humans. Onset of illness following ingestion is rapid (5-30 minutes), and is usually characterized by perioral tingling progressing to numbness of the face and neck. In severe cases, these symptoms may spread to the extremities. Symptoms may also include incoordination, nausea, vomiting, headache, dizziness, and swallowing and respiratory difficulty. In severe cases, complete respiratory muscle paralysis may occur leading to death in the absence of ventilatory support. Regardless of the severity of illness, most patients gradually begin to recover within 12-18 hours of onset of illness and are without residual symptoms within a few days.

Diarrhetic Shellfish Poisoning (DSP)

DSP, first reported in the Netherlands in the 1960s, is now reported throughout the world, with Europe and Japan the most highly affected geographic areas. To date, no cases of

DSP have been reported in the United States although the causative organisms (the dinoflagellate species *Dinophysis* and *Prorocentrum*) have been identified throughout U.S. coastal waters.

Dinophysis spp. and *Prorocentrum* spp. produce the toxin okadaic acid and its derivatives. These toxins likely cause phophorylation of the proteins that control sodium secretion in intestinal cells, resulting in impaired water balance and loss of fluids. Compared to other forms of shellfish poisoning, DSP usually causes milder illness, most often characterized by diarrhea, nausea, and vomiting. Onset of illness is usually within 30 minutes - 12 hours after consuming contaminated shellfish and complete clinical recovery usually occurs within 3 days. Of note, okadaic acid is also recognized as a possible tumor promoter. This raises concerns about the potential harmful effects to humans and marine wildlife chronically exposed to DSP toxins.

Ciguatera Fish Poisoning (CFP)

CFP is the most commonly reported marine toxin disease world-wide. Endemic in the Caribbean, CFP accounts for more than 50,000 cases of seafood poisoning per year, although it is likely greatly underdiagnosed and under-reported (as are all HAB-related illnesses). The dinoflagellate *Gambierdiscus toxicus* produces toxin precursors. These precursors are consumed by herbivorous fish and invertebrates where they are biotransformed into ciguatoxins. In turn, contaminated herbivorous fish are consumed by larger carnivorous fish that have the potential to concentrate high levels of toxin in their tissues. Illness in humans is usually caused by the consumption of contaminated carnivorous tropical and subtropical coral reef fish (e.g., barracuda, grouper, snapper, and jacks), however, consumption of contaminated smaller herbivorous fish may also cause CFP.

Ciguatoxin opens sodium channels in cell membranes, inducing membrane depolarization. In general, CFP symptoms occur within several hours after ingesting contaminated fish and initially include gastrointestinal symptoms such as nausea, vomiting, and diarrhea. Neurologic symptoms usually have a later onset (over the course of 24 hours after exposure) and are characterized by parathesias, blurred vision, headache, itching, arrhythmias, heart block, and paralysis. The pathognomonic symptom of CFP is the reversal of hot and cold temperature sensation although not all patients report this. Rarely, CFP is fatal. In some cases, CFP can persist as a chronic illness following the acute phase. Symptoms of chronic CFP may include paresthesias, itching, headaches, depression, and general malaise.

Note that symptoms of CFP may vary by geographic region as well as among individuals, even with exposure to the same food source. In the Pacific regions neurologic symptoms usually dominate CFP. In the Caribbean, CFP more often presents with gastrointestinal symptoms followed by neurologic symptoms. This may be due, in part, to the same algal species producing different toxins based on the geographic area they inhabit.

Neurotoxic Shellfish Poisoning (NSP)

NSP was first recorded in 1880 on the western coast of Florida and historically has been associated with the presence of red tides. In 1987, over 48 human cases of NSP occurred in North Carolina during a red tide. In the 1990s, prolonged red tides in the Gulf Coast caused massive manatee, fish, and bird kills as well as the closing of beaches in Texas and shellfish beds from Florida to Texas. The dinoflagellate *Karenia brevis* (formerly *Gymnodinium breve*) produces brevetoxins. Brevetoxins cause clinical illness through their ability to bind to and open cell sodium channels leading to a persistent sodium influx. The clinical illness that results depends on the route of exposure. For example, ingestion of shellfish contaminated with brevetoxin produces gastrointestinal and neurological symptoms similar to those of PSP. Unlike PSP, however, NSP has not been known to cause

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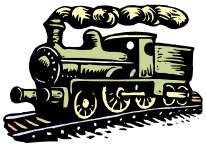

Sponsor: APIC-VA
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Program:

Sept. 28, 2004: Long-Term Care Pre-conference
Basic Training in Infection Surveillance, Prevention and Control, Gail Bennett, RN, MSN, CIC

Sept. 29 – Oct. 1, 2004: Conference

Topics will include: Outbreak investigation; Antibiograms; JCAHO and the 7th National Patient Safety Goal; Dialysis surveillance; Role of anesthesiology in infection control; DCLS - What the state micro lab can tell you; HIV - What's new for patients/employees; AND MUCH MORE!

For more information, contact: **Teresa Stowasser, RN, CIC**
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death. Because *Karenia brevis* is relatively fragile, it is easily lysed in turbulent water, releasing toxins which may become aerosolized and inhaled leading to respiratory irritation characterized by burning of the throat, conjunctivitis, and nonproductive cough.

Table 1. Summary of Harmful Algal Syndromes

Syndrome/Toxins	Dinoflagellates/Diatoms	Exposure(s)	Range of Symptoms	Incubation Period
Paralytic Shellfish Poisoning (PSP) -saxitoxins	*Alexandrium* spp., *Gymnodinium catenatum, Pyrodinium bahemense* (dinoflagellate)	Shellfish	• Perioral tingling • Numbness of face and neck • Headache • Nausea and vomiting • Respiratory difficulty • Death	30 min - 3 hours
Diarrhetic Shellfish Poisoning (DSP) -okadaic acid and derivatives	*Dinophysis* spp., *Prorocentrum* spp. (dinoflagellate)	Shellfish	• Diarrhea (most prominent) • Nausea and vomiting • Abdominal pain	30 min - 2 hours
Ciguatera Fish Poisoning (CFP) -ciguatoxins, maitotoxin	*Ganbierdiscus toxicus, Prorocentrum* spp. (dinoflagellate)	Reef fish	Early onset • Diarrhea • Nausea and vomiting Later onset • Reversal of hot and cold sensations • Paresthesias • Blurred vision • Headache • Arrhythmias • Paralysis	2 - 6 hours
Neurotoxic Shellfish Poisoning (NSP) -brevetoxin	*Karenia brevis* (dinoflagellate)	Shellfish Inhalation of aerosolized toxins	GI and neuro symtoms • Similar to those of PSP except NSP not known to cause death Respiratory irritation • Burning throat • Conjunctivitis • Non-productive cough	Few min - hours
Amnestic Shellfish Poisoning (ASP) -domoic acid	*Pseudo-nitzschia* spp. (diatom)	Shellfish	• Diarrhea • Nausea and vomiting • Confusion • Memory loss • Seizures • Coma • Death	24 - 48 hours

Amnestic Shellfish Poisoning (ASP)

ASP was first described in 1987 in Canada after an outbreak of approximately 150 cases of acute illness following the ingestion of mussels. Domoic acid, produced by the diatom *Pseudo-nitzschia multiseries*, was identified as the toxic agent, but at least 7 species of *Pseudo-nitzschia* have been identified from geographic regions worldwide. Domoic acid acts as a potent excitatory neurotransmitter leading to increased levels of intracellular calcium and continuous stimulation of neurons. Acute symptoms of ASP include nausea, vomiting, and diarrhea which may be followed by confusion, loss of memory, seizures, coma, and in rare instances, death. In some cases, especially among the elderly, those with poor renal function, and those who develop neurological symptoms within 48 hours after toxin exposure, permanent loss of short term memory may occur. Although no human outbreaks of ASP have been identified since 1987, domoic acid was identified as the causative agent of mass kills of pelicans and cormorants in Monterey Bay, California in 1991 and of sea lions in the same region in 1998. In both instances anchovies were the vectors for transfer of domoic acid.

Diagnosis, Management and Treatment of Human Illness Caused by HABs

Diagnosis**:** Toxic seafood poisoning should be suspected in all patients with a recent history (within the past 2-3 days) of seafood ingestion and either gastrointestinal symptoms alone or a combination of gastrointestinal and neurological signs and symptoms compatible with those of illnesses caused by toxin-producing HABs.

Ideally, definitive diagnosis is based on detecting and identifying specific toxins from contaminated tissues (shellfish or fish) and/or their source (water or algae species producing toxin) and identifying compatible signs and symptoms in humans. Virginia's public health laboratory, the Division of Consolidated Laboratory Services (DCLS), has the capacity to identify most toxins produced by HABs using High Performance Liquid Chromatography (HPLC). Toxins are usually identified in submitted shellfish or finfish tissue samples—detection of toxins in humans is possible, but not routinely performed. Other causes of illness due to ingestion of seafood (ex., *Vibrio cholerae*, scromboid poisoning, Puffer fish poisoning) should be considered and ruled out as part of the differential diagnosis.

Treatment and Management**:** Treatment of illness caused by toxins produced by HABs is mainly symp-

tomatic and supportive. In most instances of toxic seafood poisoning, the index case represents the tip of the iceberg and therefore should be reported to the local health department immediately. The local health department will notify the central office of the Virginia Department of Health (VDH) which will assist with follow-up on the report in order to confirm the diagnosis, detect other possible cases, identify possible source(s) of infection, and implement measures to prevent further illness. VDH will also notify appropriate state and federal agencies to institute food trace backs.

Monitoring of Virginia's Marine and Estuarine Waters for HABs

The Division of Shellfish Sanitation (DSS) within VDH, in collaboration with the United States Food and Drug Administration (FDA), routinely monitors shellfish for biotoxins from designated sampling sites in the Chesapeake Bay and its tributaries. Collected shellfish samples are sent for testing to FDA's Southeast Regional Laboratory in Atlanta, Georgia. Water samples are also collected from designated sites in the Chesapeake Bay and its tributaries and tested routinely for specific algae and related biotoxins by The Phytoplankton Laboratory at Old Dominion University in Norfolk, Virginia.

VDH is a member of the Interagency HAB Task Force (Table 2) (formerly the Pfiesteria Task Force) established in 1997 to monitor and respond to the potential public health effects of *Pfiesteria* spp. and other related dinoflagellates through environmental monitoring, passive and enhanced surveillance, a cohort study, and research on the algal species. Two cases of human illness possibly related to *Pfiesteria* spp. occurred in 1997; no cases have been detected since, although data collection and analysis is ongoing.

Table 2. Interagency HAB Task Force Participants

Virginia Department of Health (VDH)

Division of Zoonotic and Environmental Epidemiology (VDH/DZEE)

Division of Shellfish Sanitation (DSS)

Virginia Department of Environmental Quality (DEQ)

Virginia Institute of Marine Science (VIMS)

Old Dominion University (ODU)

Virginia Marine Resources Commission (VMRC)

Conclusions

Although the occurrence of HABs has been rare in Virginia's coastal waters, the occurrence of HABs worldwide appears to be increasing. The reason for the increase is likely a result of many factors including climatic changes, anomalous weather events, transport of nonindigenous marine species through the ballast water of ships, and pollution of coastal waters. Improvements in the detection of harmful algae and HABs may also contribute to their perceived increase. Because of the potential for HABs to occur in Virginia's marine and estuarine waters or for toxin-containing seafood to be imported into the state, healthcare providers need to be aware of the human illnesses that they may cause, especially among persons who are at risk for disease because of recreational and/or occupational exposure.

Submitted by: Susan Fischer Davis, MD, Division of Zoonotic and Environmental Epidemiology

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Water-related Skin Infections: Nontuberculous Mycobacteria and Non-cholera *Vibrio* Bacteria

Nontuberculous mycobacteria (NTMs) may be present in many water sources including swimming pools, aquariums, and coastal waters. NTMs have also been identified in fish. Skin infections in people may occur when NTM-containing water comes into contact with cuts or abrasions. Infections usually occur on extremities and appear as nodular erythematous lesions. Minor infections may heal spontaneously, but more serious infections may require antibiotic treatment and/or surgical debridement. *Mycobacterium marinum* is the most common causative organism of NTM water-related skin infections.

Non-cholera *Vibrio* species are most commonly found in salt water, but may also be present in fresh water. As with NTMs, skin infections in people may occur when non-cholera *Vibrio*-containing water comes into contact with cuts or abrasions. Infections typically appear on the extremities as ulcerations and cellulitis. Disseminated infections may occur, especially among immunocompromised patients. Non-cholera *Vibrio* skin infections require antibiotic treatment. *Vibrio vulnificus* is the most common causative organism of non-cholera *Vibrio* water-related skin infections.

Prevention

The most reliable form of prevention is avoiding water that could potentially be contaminated with NTMs or non-cholera *Vibrio* bacteria **when cuts or abrasions are present on skin**. Risk of infection may also be reduced by washing with soap and water after contact with water that could potentially be contaminated with NTMs or non-cholera *Vibrio* bacteria. Infection with NTMs may also be prevented by wearing gloves when fishing and handling fish.

The Virginia Department of Health (VDH) is monitoring reports of these infections and investigates any unusual increase in the number of positive tests. VDH has also developed a brochure, available from local health departments or on the internet at http://www.vdh.virginia.gov/whc/external_whc/ZEEpageExternal.asp, explaining NTMs and non-cholera *Vibrio* water-related skin infections. **Healthcare workers are encouraged to report cases of water-related skin infections due to NTMs and *Vibrio* species to their local health department.**

Cases of Selected Notifiable Diseases Reported in Virginia*

Disease	Total Cases Reported, May 2004: State	Regions: NW	N	SW	C	E	Total Cases Reported Statewide, January - May: This Year	Last Year	5 Yr Avg
AIDS	69	2	29	4	15	19	285	357	344
Campylobacteriosis	63	16	10	20	10	7	184	186	157
***E. coli* O157:H7**	0	0	0	0	0	0	1	17	13
Giardiasis	38	5	3	15	5	10	144	113	124
Gonorrhea	623	38	63	58	170	294	3,596	3,621	3,905
Hepatitis, viral									
A, acute	14	3	2	2	2	5	39	36	49
B, acute	22	1	0	6	5	10	82	58	60
C, acute	1	1	0	0	0	0	10	1	2
HIV Infection	86	4	21	7	16	38	352	323	327
Lead in Children†	65	6	7	12	32	8	248	240	201
Legionellosis	3	0	0	2	0	1	8	8	6
Lyme Disease	4	1	0	0	2	1	12	14	16
Measles	0	0	0	0	0	0	0	0	1
Meningococcal Infection	5	1	0	0	0	4	8	11	20
Mumps	0	0	0	0	0	0	1	1	4
Pertussis	18	12	0	2	2	2	57	33	28
Rabies in Animals	54	11	15	14	5	9	187	224	217
Rocky Mountain Spotted Fever	1	0	0	0	0	1	1	1	<1
Rubella	0	0	0	0	0	0	0	0	0
Salmonellosis	84	12	22	18	15	17	255	274	280
Shigellosis	6	1	1	1	2	1	36	127	127
Syphilis, Early§	27	1	5	3	7	11	69	75	107
Tuberculosis	25	1	17	2	5	0	80	87	100

Localities Reporting Animal Rabies This Month: Accomack 1 raccoon; Albemarle 1 fox, 1 raccoon; Alexandria 1 raccoon; Bedford 3 raccoons; Clarke 1 cat; Essex 1 fox; Fairfax 1 bat, 1 groundhog, 3 raccoons, 4 skunks; Franklin 1 raccoon; Giles 1 raccoon; Goochland 1 raccoon; Henry 1 bat; Isle of Wight 1 cat; King George 1 fox; Loudoun 3 raccoons; Lunenburg 1 raccoon; Lynchburg 1 raccoon; Madison 1 skunk; Norfolk 1 raccoon; Northampton 3 raccoons; Page 1 raccoon; Patrick 2 raccoons; Pittsylvania 1 cat, 1 raccoon; Powhatan 1 raccoon; Prince George 1 fox; Prince William 2 raccoons; Rockbridge 1 fox; Rockingham 1 skunk; Shenandoah 1 raccoon; Stafford 1 cat, 1 skunk; Surry 1 skunk; Tazewell 1 dog; Washington 1 raccoon; Westmoreland 1 raccoon; Wythe 1 skunk; York 1 raccoon.

Toxic Substance-related Illnesses: Adult Lead Exposure 11; Asbestosis 2; Pneumoconiosis 6.

*Data for 2004 are provisional. †Elevated blood lead levels ≥10µg/dL. §Includes primary, secondary, and early latent.

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