

Food Industry Watch



Food Toxins

2010

New Insights & Technologies Guide Management of a Safer Food Supply

Watch List

- Fear that botulism could be used as a terrorist weapon has driven a flurry of recent research. An antitoxin practical for mass manufacture has proved effective in animal tests, according to Tufts University researchers (“Novel antitoxin strategy,” 2010).
- Strict regulatory limits on mycotoxins in grains, fruits, and vegetables are pushing the development of advanced detection technologies, from enzyme-linked testers that change color to electronic “noses.”
- New farming practices can reduce the prevalence of toxin-producing molds. The deliberate introduction of a benign mold, for example, can crowd out toxic species.
- Climate change may alter global distribution of marine algae, including toxin-producing species. New areas of risk for paralytic shellfish poisoning may require expanded monitoring.
- The use of antibiotics in livestock is likely to come under increasing scrutiny as research shows that antibiotics can stimulate the production of bacterial toxins.

Key Takeaways

- A toxin is a poison produced by an organism. A toxin may persist in food even after cooking has killed the organism.
- Treatment is impractical against most food toxins. The emphasis is on prevention and early diagnosis to stop the spread of poisoning to others.
- Bacteria that attack the intestines often deploy multiple toxins. Moreover, different bacteria can produce essentially the same toxin (for example, CDT), which suggests genetic borrowing.
- Botulinum toxin and other paralyzing neurotoxins are rare but highly dangerous, and require vigilance.
- Paralytic toxins in certain fish and shellfish are originally produced by algae, an occurrence that demonstrates accumulation through a food chain.
- Fungal toxins such as aflatoxin and patulin are under control in developed countries but remain a burden on health and food production throughout the rest of the world.

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Executive Summary

A toxin is a poison produced by an organism. Most toxins in food come from a few well-known bacteria, with *Salmonella* leading the list. Fungal toxins are well-controlled in developed countries but pose a wide range of threats — to crops as well as unlucky mushroom hunters. Plant sources of toxins include undercooked red kidney beans and alkaloid-containing weeds that livestock will graze if preferred pasturage is not available. Antitoxins are often not available or practical against food toxins, but faster, lower-cost detection methods and treatments are on the horizon.

Bacteria often produce more than one toxin — *C. perfringens*, the “cafeteria germ,” produces 12. In addition, many bacteria have toxins in common, such as cytolethal distending toxin (CDT). Released in the intestines, CDT penetrates host cells and interferes with DNA copying, causing different

effects in different types of cells. A recent study shows that veterinary use of antibiotics in cattle can stimulate intestinal bacteria to produce toxin.

Botulinum toxin is the most powerful toxin known; causes paralysis and death at infinitesimal doses. Though botulism is rare, its potential as a terrorist weapon has driven much recent research. Vaccination could prevent botulism, but the development of an effective antidote would involve fewer people and preserve beneficial uses of the toxin. DNA-based biomanufacturing strategies show great promise.

Paralytic shellfish poisoning (PSP) can occur after eating clams, scallops, or mussels but the toxin originates with algae, eaten by the shellfish. Fish such as sea bass and snapper can also accumulate neurotoxins from algae, causing ciguatera. Monitoring algal blooms helps assure the safety of seafood, including shrimp and other species cultivated in coastal farms. Global climate change may lead to new patterns of algal activity, broadening needs for monitoring in the future.

Mycotoxins, from various mold species, can damage grains and other crops and exert effects on human health through milk, eggs, and meat. In developed nations, strict monitoring prevents outbreaks of aflatoxicosis and other illnesses. In developing nations, mycotoxin exposure is widespread and sustained over long periods; costs and health effects are unquantified. Mycotoxin detection methods require time-consuming laboratory work, but a mix of faster, lower-cost technologies aids in initial screening. Cultivation practices can reduce crop vulnerability to fungal toxins.



Overview

Most Food Toxins Are Produced by Bacteria, Fungi & Plants

A *toxin* is a poison produced by an organism. Chemically, most toxins are proteins that interfere with normal functioning in host cells. A toxin may be heat-stable, meaning it can cause illness even after the organism has been killed by cooking. Organisms that produce toxins include bacteria, fungi, and plants, which get into food by different paths and cause a variety of effects, from short-term gastrointestinal illness to muscular paralysis (sometimes fatal) as well as hallucinations, chronic arthritis, and liver cancer.

Bacterial toxins associated with foodborne illness come mostly from a few well-known names. *Salmonella* is by far the most common, causing 10 times as many cases as the number two bug, *Escherichia coli* (Olsen et al., 2000). Others include *Clostridium perfringens* (the “cafeteria germ”), *Shigella* species, *Staphylococcus aureus*, *Bacillus cereus*, and *Campylobacter* species. These bacteria exist in livestock, poultry, and crop fertilizer as well as in humans, and may be introduced into food at any stage of handling. Bacteria multiply and produce toxin when conditions are favorable — as a rule, at temperatures between 40 and 140 degrees Fahrenheit (4 and 60 Celsius) — while food is in dishes waiting to be served or later in the intestines.

Fungal toxins are a less frequent cause of food-related problems but pose a range of threats — to crops as well as unlucky mushroom hunters. Molds on peanuts, corn, wheat, and other cereals produce mycotoxins, including aflatoxin which attacks the liver and can cause cancer. Moldy apples, grapes, and other fruits that end up in juice may be laced with patulin, which causes stomach irritation and diarrhea. A wrong selection of mushrooms can offer up a smorgasbord of toxins, including neurotoxins, gastrointestinal toxins, organ-killing protoplasmic toxins, and disulfiram-like toxins (which cause brief acute illness but only if the subject drinks alcohol within 72 hours).

Plant toxin sources include undercooked red kidney beans, alkaloid-containing weeds, and others. The toxin from red kidney beans is phytohemagglutinin, an example of a poison with clinical uses: Stimulating reproduction of human T cells. Weeds such as common heliotrope and ragwort are sometimes used as herbal remedies but can cause pyrrolizidine alkaloids poisoning. These weeds get into animal feed by infesting cereal crops or may be grazed by cattle, horses, and sheep if better-tasting pasturage is not available.

An *antitoxin* is a substance, drawn from previously exposed animals or humans that targets and neutralizes a specific toxin. Antitoxins are often unavailable or impractical for treatment of food poisoning. For example, there is an antitoxin for Death Cap and other *Amanita* mushrooms, but a two-day lag before symptoms appear is problematic for early diagnosis and effective treatment. There is no antitoxin for the paralytic toxins sometimes found in seafood; treatment consists of pain management and respiratory support if needed. For the usually less severe gastrointestinal illnesses caused by bacteria, treatment is directed via antibiotics at the bacteria rather than the toxins. Generally, the emphasis in foodborne illness has to be on prevention rather than treatment. A listing of major food-related pathogens is maintained by the US Food and Drug Administration (FDA) in the *Bad Bug Book*.

How Toxins Work in the Intestines

Different Bacteria Produce Similar Toxins

Foodborne bacteria multiply in the intestines and release toxins, with symptoms typically appearing six or more hours later (earlier symptoms point to toxin already in the food at the time it was eaten). Bacteria may produce more than one kind of toxin — for example, *C. perfringens* produces 12 (Sirous et al., 2009). Different kinds of bacteria may produce essentially the same toxin. *Shiga* toxin

is produced by *Shigella* species but also by *E. coli* serotype O157:H7, notorious for far-reaching outbreaks of illness due to contaminated ground beef and for a tainted-spinach outbreak that made headlines in 2006.

Numerous toxins have been identified chemically and traced to the bacterial genes that encode them, but gaps remain in our knowledge of the exact roles played by specific toxins. A toxin may be associated with intestinal lesions and bloody diarrhea in one type of bacterial infection, but a different type of bacteria using the same toxin may not display the same symptom. It has been suggested toxins have different effects in different combinations. For example, Shiga toxin might interact with cytolethal distending toxin (CDT) to enable *E. coli* O157:H7 to escape the intestines and enter the bloodstream (Smith & Bayles, 2006). CDT is deployed by several food poisoning bacteria, including *Salmonella enterica* serovar Typhi, *E. coli*, *S. dysenteriae*, and *Campylobacter* species. The toxin is made up of three proteins, which carry out a three-step attack — adhere, penetrate, disrupt — against cells of the intestinal lining. Once inside the cell nucleus, the toxic CdtB protein cuts into DNA being assembled for cell division. Sensing DNA faults, the cell halts mitosis and dies. CDT also attacks cells of the immune system, including T and B cells, and fibroblasts involved in healing, which suggests that CDT has multiple roles in suppressing host defenses and buying time for bacterial growth (Smith & Bayles, 2006).

The gene responsible for a virulence factor (such as CDT) tends to be identical from one type of bacteria to the next. These genes appear as “pathogenicity islands,” with unstable sequences before and after the virulent code. This structure suggests the ability to produce CDT or Shiga toxin is transferable as a block from one organism to another — which explains how different bacteria have arrived at the same virulent chemistry, and how a few strains of *E. coli*, a mostly benign species, became pathogenic (LeBlanc, 2003).

The interactivity of *E. coli* O157:H7 (often called EHEC, for enterohemorrhagic *E. coli*) was the focus of a recent study on antibiotic use in cattle. Researchers found that low doses of enrofloxacin stimulated O157:H7 to produce Shiga toxin, indicating a potential increase in risk to human health from contaminated beef (Maurer et al., 2008). The widespread use of antibiotics in cattle, pigs, and poultry is already under review because of rising bacterial immunity. The authors suggest further that veterinary use of antibiotics may encourage the transfer of toxin-producing capability to new strains of bacteria.

Botulinum Neurotoxin

Low to High Tech Defenses against the World’s Most Dangerous Toxin

There are seven strains of *Botulinum* neurotoxin (BoNT), identified as A through G, four of which are a threat to humans (A, B, E, F). Serotype A is the most dangerous, requiring intubation in 67 percent of cases (Wenham & Cohen, 2008). The toxin binds to nerve cells, blocking release of the neurotransmitter acetylcholine, which results in muscles going limp and, in some cases, sudden respiratory failure. Symptoms begin with dry mouth or sore throat and then paralysis progresses down both sides of the body. The lethal dose is 100 nanograms, an inconceivably small amount. Researchers looking at BoNT as a terrorist weapon estimate that a gram of toxin could kill a million people. *Clostridium botulinum* spores are abundant in the environment but only multiply and produce toxin in the right combination of conditions — absence of oxygen, warm temperature, and low acidity. Preserved foods provide these conditions when home canning is done with insufficient heat and pressure to destroy bacterial spores. However, an outbreak in 2006 caused by commercially bottled carrot juice put a new light on how favorable conditions can come together. The carrot juice had been pasteurized (heated enough to kill most bacteria but not enough to destroy

spores). The bacteria may also have gotten into the carrot juice after the bottles were opened. The three people who were poisoned opened and then stored the bottles at room temperature. Refrigeration would have kept *C. botulinum* from producing toxin. For the manufacturer, another preventive measure would have been to increase the acidity of the drink — for example, by blending with another juice (Shuler et al., 2006).

A botulism antitoxin, cultured from horses, is available once the diagnosis has been made. The earlier the diagnosis the better, since the antitoxin cannot reach toxin that is already bound to nerve cells and cannot repair paralysis or damage already done. Alternatively, a preventive approach is possible, as a BoNT vaccine has been developed in Japan for protection of research workers. Immunization on a larger scale would raise a policy issue, given that botulism is rare and attendant costs and controversies would be substantial. Immunization would also take away clinical uses of *Botulinum* toxin, which include treatment for spastic nerve disorders as well as cosmetic uses of Botox (Cai & Singh, 2007).

Meanwhile, a flood of funding for antiterrorism research is likely to bring new options for dealing with botulism and other toxin-related challenges. As a bioweapon, BoNT has fearful potential; it is relatively easy to manufacture, has the capability to kill and cause long-term illness on a wide scale and could overwhelm hospital resources for months. Aerosol delivery has been speculated on, but poisoning milk or other foods would be a more practical and likely approach (Cai & Singh, 2007).

An innovative antitoxin strategy from Tufts University uses three binding agents that attach to the BoNT molecule and surround it, rendering it unable to attack nerve cells. The binding agents carry a chemical “tag” that is in turn recognized and swept up by an antibody, administered with the binding agents. While the binding agents are specific to BoNT, the tag

and antibody can be readily adapted for use against other kinds of toxin. By design, the tag and antibody are easy and cheap to manufacture in the laboratory. The binding agents have to be specially developed for the target toxin but are cheaper to produce and safer to handle than antitoxins cultivated in animals (“Novel antitoxin strategy,” 2010; Sepulveda et al., 2009).

Seafood Toxins

Toxins Accumulate in the Food Chain from Algae to Humans

Saxitoxin, the chemical agent in paralytic shellfish poisoning (PSP), cannot be seen, smelled, or cooked out of mussels, clams, and scallops. As a neurotoxin, it blocks communication between nerves and muscles, and a large dose can cause death by respiratory failure in two hours (Sobel et al., 2005). Less severe cases begin with a tingling sensation around the mouth, followed by headache, nausea, vomiting, and diarrhea. Saxitoxin originates with algae consumed by shellfish.

Algae that produce saxitoxin are common along US coasts, notably in areas that see red tides. Since there is no antitoxin for PSP, food safety depends on monitoring by states where outbreaks occur. The FDA sets guidelines for testing and threshold levels for a ban on harvesting. Between 1998 and 2002, CDC recorded seven outbreaks in the US involving 43 people (Sobel et al., 2005). Considering future incidence of PSP, a US and Canadian team hypothesizes that toxin-resistant shellfish, having a survival advantage, may become increasingly prevalent, causing long-term change in ecosystems and food safety requirements (Bricelj et al., 2005).

Effects of algal blooms on seafood farms have only recently come under study. Over the last decade, shrimp aquaculture has been established along the west coast of Mexico, and similar operations are likely to contribute significantly in future food production. In



addition to killing large numbers of cultured shrimp, algal toxins are known to reduce growth rates and disease resistance. Further study is needed to determine the risk of algal toxin being transmitted to human populations (Perez-Linares et al., 2008).

Ciguatera is one of the most common forms of seafood poisoning worldwide, causing illness in an estimated 20,000–50,000 cases annually. Between 1998 and 2002, there were 101 outbreaks in the US, affecting 374 people, with 30 hospitalizations and one death (Sobel et al., 2005). Ciguatera is caused by two toxins from algae that are eaten by small, plant-eating fish, which are in turn eaten by larger reef-dwellers such as barracuda, sea bass, and snapper. Ciguatoxin and maitotoxin become more concentrated at each step in the food chain before reaching the human consumer. Scientists anticipate that global climate change and rising ocean temperatures may affect the distribution of algae, introducing toxin-producing species to new areas with unknown effects on human health.

Mycotoxins in Crops

Management Capabilities Contrast in Developed Versus Developing Nations

Crop molds grow during storage and produce toxins such as aflatoxin, fumonisin, ochratoxin A, and deoxynivalenol. These mycotoxins affect human and animal health through direct consumption of crop foods and feed and may be concentrated in milk, eggs, and meat. The Joint Expert Committee on Food Additives (JECFA), combining resources of the United Nations Food and Agriculture Organization and World Health Organization, has set standards for safe levels of mycotoxins in food. In the US, the responsible agency is the Grain Inspection, Packers, and Stockyards Administration (GIPSA).

In developing nations, mycotoxin exposure tends to be greater because there is less variety of diet and poor quality food is less likely to be thrown away. A

typical diet in parts of Africa might include up to 500 grams of corn products per day (500 g = about one pound). At that rate, even moderate contamination with aflatoxin would exceed JECFA standards. Outbreaks of aflatoxicosis, a liver-damaging illness, occurred in Kenya in 1981, 2001, 2004, and 2005 (Shephard, 2008). The full burden of related health effects includes reduced resistance to infectious disease — aflatoxin is linked to suppression of the immune system — but secondary effects are difficult to quantify.

Control of mycotoxins depends critically on detection technology. Detection systems have to be *sensitive* to trace amounts and *specific* for target substances. Definitive laboratory techniques require time-consuming steps to separate mycotoxin from the sample. Increasingly, the trend is to use new, faster technologies for screening and then traditional labwork for analysis of positive results. With ELISA (enzyme-linked immunosorbent assay), graduated doses of antigen are dipped into an array of samples to get a quick measurement of a mycotoxin, while LC–MS/MS (liquid chromatography with tandem mass spectrometry) offers the ability to look for multiple mycotoxins in one pass.

Cultivation practices can reduce mold infestation of crops. In peanut farming, for example, kernel moisture is key to a crop's vulnerability, and late-season drought is the most significant opportunity for infestation. Irrigation (if available) and early harvesting are tactics to keep kernel moisture high and plant defenses strong. In Australia, a combination of aflatoxin penalties and free Web tools for estimating kernel moisture encourage farmers to manage peanut crops efficiently. Another cultivation strategy — deliberately introducing nontoxic strains of mold to compete against toxic strains — has also shown success. At packing facilities, toxin levels in peanuts can be managed efficiently by mechanical sorting for size and electronic sorting for color. Infested peanuts tend to be smaller and off-color compared to wholesome peanuts.



Related Entities

- Centers for Disease Control and Prevention (CDC)
- European Mycotoxin Awareness Network (EMAN)
- Food and Agriculture Organization of the United Nations (FAO)
- Food and Drug Administration (FDA)
- Grain Inspection, Packers and Stockyards Administration (GIPSA)
- Joint Expert Committee on Food Additives (JECFA)
- US Department of Agriculture (USDA)
- World Health Organization (WHO)

Acronyms

BoNT: *Botulinum* Neurotoxin

EHEC: Enterohemorrhagic *E. coli*

ELISA: Enzyme-linked Immunosorbent Assay

LC-MS/MS: Liquid Chromatography with Tandem Mass Spectrometry

PSP: Paralytic Shellfish Poisoning

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