Fungi in Food: Natural Poisons and Gourmet Delicacies

CHAPTER 9

Countryside scenes of people eating foods unadulterated by the contaminating influences of modern agriculture are a popular image for advertisers. We perceive these foods as safe because they have been sampled and eaten over the millennia by foraging humans seeking nourishment. Even so, everyone knows that some of these plants are poisonous and should be avoided, although the specific folklore about the edibility of natural plants is familiar to few people in industrial societies. Most of us simply choose from among the foods displayed in supermarkets and farm stands.

Even plants commonly eaten are not necessarily nontoxic. Leaves and stems of potato plants contain high concentrations of toxic alkaloids. Tubers left in sunlight turn green and accumulate these same alkaloids; therefore, green tubers should not be eaten and potatoes should be stored in the dark. Cassava, or manioc (*Manihot esculenta*), a staple food for more than 300 million people in tropical regions, contains high levels of cyanogenic glucosides. The starchy root of this plant must be ground and washed thoroughly before being eaten to avoid cyanide poisoning. Many natural components of plants that we commonly eat are toxic, and many are carcinogenic. Research by Dr. Bruce Ames at the University of California at Berkeley has demonstrated mutagenic compounds in familiar foods, including raw mushrooms and many vegetables.

Over the years, humans have learned which plants or parts of plants are safe to eat. Our bodies have also developed many detoxification mechanisms that allow us to eat some toxic compounds with little or no harm. Plant breeders who select plants with resistance to insects or pathogens test the resistant cultivars for increased levels of toxic compounds before releasing them for human consumption. Many compounds used by plants to resist these pests may cause toxic reactions in us as well. Such compounds could be present at high levels throughout the plant tissues, resulting in a health risk far greater than low-level pesticide residues on plant surfaces, where they can be washed away.

**Ergotism and the Holy Fire**

Once the edibility of a plant was established, humans began to select plants that were high yielding and suitable for agriculture. As a result,
most people today eat a limited number of plants containing only a few major calorie sources. As this specialization developed, the potential health threat from any particular plant increased greatly because of the disproportionate role these few species play in our diet. One particular risk that is receiving renewed attention in the interpretation of historical records is associated with the cultivation of the cereal rye (Secale cereale).

In the Middle Ages, a frightening disease known as holy fire or St. Anthony's fire was common but unpredictable. Unlike most diseases, which were rampant in the overcrowded cities, holy fire was most common among the rural poor. It did not seem to be contagious and could strike one family or even members of a family without necessarily infecting neighbors. Children and feeble people were more susceptible to the disease. Nursing mothers might see the effects in their babies. A folk cure for these victims was to have the nursing mother eat white bread rather than the coarse and inexpensive rye bread.

This scourge of the Middle Ages has an unclear history. Medical diagnostics were more primitive then, and some of the described symptoms could be attributed to several different medical problems, making historical records difficult to interpret. The common symptoms included strange mental aberrations and hallucinations. People described the feeling of burning skin or insects crawling under their skin. Women frequently miscarried when the disease occurred; fertility was generally reduced during outbreaks. Severe cases resulted in gangrene infections due to constriction of blood vessels in the extremities; many victims lost hands and feet and became permanently crippled. Hospitals dedicated to St. Anthony took in such patients, caring for them until their painful and prolonged suffering ceased. In art work from that period, one can see the symbols of St. Anthony on medals and clothing worn by people hoping to ward off the effects of the disease.

Ancient European records of the disease before the Middle Ages are missing, and the Greeks and Romans do not seem to have described the malady. The disease became prominent following the introduction of rye as an agricultural grain after the invasion of eastern Europe by nomadic groups such as the Vandals. Disease outbreaks were sporadic in the Middle Ages, particularly in years in which cold winters put the rye under stress and cool, wet springs prolonged the flowering period.

France was the center of many severe epidemics, primarily because rye was grown as the staple crop of the poor, and the cool, wet climate was conducive to the development of the fungus that caused the mysterious malady. We now know that alkaloids produced by a fungus parasitic on rye were the source of the varied symptoms observed in human beings. Harvested along with the regular rye grains were hard, purplish black, grainlike structures (called ergots or sclerotia) produced by the fungus, Claviceps purpurea. When the grain and the ergots were ground together during milling, the flour became contaminated by the potent toxic alkaloids of the fungus.

It takes only a few ergots mixed in the flour to produce the frightening symptoms of the holy fire. Hundreds of thousands of humans as well as cattle and horses died or became seriously ill in the years before its cause was understood. Not until well into the 18th century was the direct relationship between the disease and the fungal parasite of rye widely recognized. Once the ergots were known to be the source of the misery, they could easily be separated from the grain before milling to prevent the poisoning, but accidental ergot poisonings, especially among domestic animals, have been recorded into the 20th century. Today, government standards prevent the sale of grain containing more than 0.3% ergot by weight for human consumption. A cultural preference for wheat bread over rye bread remains in France and among many people in other parts of Europe and in the United States.

As the rye grain matures, the ergots fall to the soil. These masses of mycelium with a dark rind are survival structures for the fungus similar

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Fig. 9-1. Saint Anthony. Redrawn from a woodcut made in Germany about 1215 A.D.
to those produced by many other fungi. They help protect the fungus from desiccation and ensure its survival through the winter. In the spring, small mushroomlike structures are produced from the ergots on the soil surface. They forcibly eject the microscopic ascospores (sexual spores) of the fungus upward, to be picked up by wind currents in the field of rye. Rye, like many grasses, is wind pollinated. The stigma of the rye flower is large and featherlike to help trap the windborne pollen. This same mechanism helps trap the airborne ascospores expelled from reproductive structures of the fungus on the soil surface.

If water and temperature conditions are favorable, the ascospores germinate and infect the ovaries of the flowers. Like many fungi, *C. purpurea* then produces asexual conidia to increase the number of infections in the rye field. This must happen quickly because the flowers remain susceptible to infection for only a short time. To help spread the conidia, the fungus also secretes a sweet, sticky *honeydew* over the infected flower, which attracts flies and other insects. As they feed on the honeydew, conidia become attached to their feet and bodies. The insects move among the rye plants searching for more honeydew, effectively disseminating the conidia. Depending on the weather conditions, this honeydew stage may last for a few or many days.

Rather than producing a rye grain, infected flowers may be sterile, which directly reduces yield. Other infected grains are of reduced quality, while still others are totally replaced by the sclerotium, or ergot, of the fungus. Control of the disease is very successful if the stubble is plowed under after harvest to bury the remaining ergots and the rye crop is rotated to different fields.

In a recent book by historian M. K. Matossian, *Poisons of the Past: Molds, Epidemics, and History*, the author builds quite a strong case for the theory that ergot toxicity played a multifaceted role of in European history. She challenges a common theory that the introduction of the potato from the New World was responsible for the European population explosion between 1750 and 1850. She presents convincing evidence that the population growth was directly related to the removal of toxic compounds (including ergot alkaloids and other mycotoxins discussed below) from the human diet when the potato replaced rye and other susceptible grains as the preferred food of poor people. Her population studies of individual countries also provide evidence for the role of ergotism in demographic depression, particularly in France, where rye remained the predominant food of the poor long after the potato had become more popular in other countries.

She also presents evidence that ergot poisoning had a role in the mental state of the French peasants, which contributed to the French Revolution in 1789, and in the accusations of witchcraft in Europe and the United States. The hallucinogenic lysergic acid diethylamide (LSD) is derived from an alkaloid of ergot. Accusations of witchcraft have been shown to correlate with historical weather data, including cold, wet weather in winter and spring. These accusations are concentrated in areas of France, Central Europe, and the Rhine Valley where rye was a staple cereal.

Midwives have long used ergot to induce abortion and as an aid in childbirth, which explains the ergot’s common name of “mothercorn.” The powerful alkaloids can be purified and, with the dose carefully measured, used in modern medicine to reduce postpartum bleeding. Other modern pharmaceutical uses of ergot derivatives include the migraine headache medication cafegrot and various compounds used for the treatment of mental conditions.
problems, blood pressure, and other ailments. Ergot infection of rye is deliberately induced in some areas for these uses.

G. L. Carefoot and E. R. Sprott, in *Famine on the Wind*, also describe the role of ergotism in history. For instance, Peter the Great was halted in his attempt to capture Constantinople (Istanbul) and gain access to warm-water ports when his troops and their horses were poisoned by ergoty grain at the mouth of the Volga River in 1722. Many serious outbreaks of ergotism have been recorded in Russia and probably would have continued if the New World potato had not replaced rye as the major food crop of the poor.

*C. purpurea* can infect other grain species in addition to rye, including the less susceptible wheat and many wild grasses. Careful harvesting and milling practices make ergot poisoning unlikely in modern times, although animal poisonings are occasionally reported because animal feed is not as carefully monitored. This is most common when animals, such as horses, feed on wild grasses in pastures following prolonged wet springs, when ergot infection is more severe. Since the 1970s, when male-sterile breeding lines of wheat and barley were developed, there has been concern about increased susceptibility of these lines to ergot infection because they have an extended flowering period, leaving the plants open to infection longer. Breeding lines are being carefully evaluated to avoid selection of new grain cultivars in which ergot might be an important problem. Other *Claviceps* species infect other plants, as well, causing problems in corn in Mexico and in sorghum and pearl millet, especially in tropical Africa.

Related fungi have been discovered in pasture grasses, where they have caused serious diseases of grazing animals. Investigations demonstrated that the hyphae of these fungi are found within the tissues of the infected plants. The fungi are *endophytes*; that is, they exist entirely within the host plant. The mycelium infects developing seeds, so new seeds contain the fungus in the next generation. Although this is a disadvantage in pasture grasses because of their toxic effects on animals, these same fungi have been found to deter insect feeding on turfgrasses (grasses used for lawns and golf courses). Endophyte-containing seed is now sold as a biological control against sod webworms and other turfgrass pests in perennial ryegrasses and fescues.

### Decay Fungi in Foods

We find rotting, moldy food objectionable because the microorganisms responsible for the rot produce distasteful or even toxic compounds. An example of such a compound is ethyl alcohol, a product of anaerobic fermentation produced by yeasts, which are Ascomycetes, and bacteria. Yet, most human cultures have developed a taste for alcoholic beverages despite their negative health effects, and people deliberately create conditions conducive to enhanced production of alcohol in various products such as corn, rice, rye, potatoes, and grapes. We are not the only species to find the alcoholic

![Fig. 9-4. Ergots in rye (left) and bromegrass (right).](image)

![Fig. 9-5. Leg lesions of a calf affected by ergotism. Note the sharp demarcation between living (top) and dead (bottom) tissue.](image)
content of foods attractive; some birds and small mammals ingest fermenting
berries and other fruits to the point of intoxication. Even the word
*intoxication* reflects the knowledge that the product is not healthful despite
its deliberate ingestion.

Many fungi invade moist food products and contribute to their decay.
Most do not produce toxic compounds. Even though the fungi make the
food distasteful, it may still remain edible. An unappealing but edible
example is a potato tuber rotted by *Phytophthora infestans*. Some of the
rot fungi, such as the common secondary invaders of the genus *Alternaria*,
do produce toxic compounds. The most important *mycotoxins*, or poisons
produced by fungi, are those that invade grains and seeds. Infections
generally occur while the food products are in the field before harvest.
Certain conditions increase the chance of invasion, including wounds in
the seed coat and high seed moisture levels that allow more extensive invasion
by the fungi. Seeds containing high amounts of oil are particularly vulnerable
to infection and mycotoxin contamination. Examples include sunflower
seed, peanuts, soybeans, cottonseed (which is commonly used as a cattle
feed), walnuts, pecans, and coconuts. Grains are also very susceptible to
mycotoxin contamination.

**Grain Mycotoxins**

Hogs, cows, and other animals often reject moldy grain and feed, which
suggests that instincts have developed from previous unfortunate
experiences. Fungi of the genera *Penicillium*, *Aspergillus*, and *Fusarium*
produce toxic and carcinogenic compounds that threaten the health of
animals and humans who ingest them in food. While animals commonly
eat unprocessed grain or feed and refuse badly contaminated products,
humans usually mill or otherwise process grain products until we can no
longer detect the harmful compounds in our food. We must rely on careful
inspection of grain and the rejection of contaminated grain by federal or
state inspectors and food processors.

*A. parasiticus* also produces aflatoxins. These toxins are tasteless, odorless, and colorless. Environmental conditions affect the amount of aflatoxins produced, but even grain that shows no obvious infection by the fungi in the field or in storage may be contaminated by the poisons. Aflatoxins are some of the most powerful toxins and carcinogens known. Rats fed a diet containing one part per billion (ppb) of aflatoxin B1 develop liver cancer. Aflatoxins are thought to be 100 times more likely to induce cancer than polychlorinated biphenyls (PCBs) are. They are also mutagenic, toxic to the liver, and immunosuppressive. Besides affecting the body's immune response to pathogens, aflatoxins in the diet also affect the success of vaccinations against other important pathogens.

The historian M. K. Matossian, who has studied the role of ergot and
mycotoxins in history, suggests that the tremendously high mortality due
to the Black Death, or bubonic plague, in 1348–1350 may have been due
to the immunosuppressant effects of mycotoxins in moldy grains. These
same mycotoxins may have killed rats, which fed on the grain as well.
This increased the number of fleas looking for alternate sources of a blood
meal and, in the process, transmitting the bubonic plague bacteria to humans
and to domestic animals, which also died in high numbers during that
period.

Aflatoxins proliferate in peanuts and corn, particularly when the seeds
are not well dried before storage, when water leaks into storage areas,
or when storage insects feed on the seeds, making wounds that allow easy

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Fig. 9-6. Endophyte mycelium in grass seed. The fungus is related to the ergot
fungi and causes toxic effects in animals that eat endophyte-infected plants.

Fig. 9-7. Endophyte-infected turfgrasses are used as a biological control against
feeding by certain turfgrass insect pests. The perennial ryegrass cultivar Pennant
(right) resists bill bug damage better than ryegrass cultivars without endophytes.
invasion by fungi. For instance, aflatoxin levels can increase from 200 to 2,000 pph in just three days in high-moisture corn. Aflatoxins may remain even after the processing and baking of contaminated products. When dairy cows eat contaminated grain, aflatoxins are metabolized into another highly carcinogenic compound that appears in the milk. Because cottonseed is a significant feed source for dairy cows, the occurrence of aflatoxins in cottonseed is of concern. Treatment of cottonseed with ammonia greatly reduces the level of aflatoxin so that it can be fed to animals. Aspergillus species can also cause serious lung infections, known as aspergilloses, with symptoms similar to those of tuberculosis. Peanut growers in the south-

Table 9-1. Major Mycotoxins and Toxin-Producing Fungi from Corn, Cereals, Soybeans, Peanuts, and Other Products, and Some of Their Effects on Animals

<table>
<thead>
<tr>
<th>Toxin or Syndrome</th>
<th>Fungal Source</th>
<th>Feeds or Foods Affected</th>
<th>Possible Effects on Animals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspergillus toxins</td>
<td><em>Aspergillus flavus</em> and <em>A. parasiticus</em></td>
<td>Cereal grains, peanuts, and other foods</td>
<td>Liver injury; carcinogenic; reduced growth rate; hemorrhagic enteritis; suppression of natural immunity to infection; decreased production of meat, milk, and eggs</td>
</tr>
<tr>
<td>Aflatoxins (primarily B1, B2, G1, G2, M1, and M2 are metabolites; M1 and M2 are important regional contaminants in milk)</td>
<td><em>A. ochraceus</em> and <em>Penicillium viridicatum</em></td>
<td>Cereal grains</td>
<td>Toxic to kidneys and liver; abortion; poor feed conversion, reduced growth rate, general unthriftiness; reduced immunity to infection</td>
</tr>
<tr>
<td>Sterigmatocystin</td>
<td><em>A. nidulans</em> and <em>A. versicolor</em></td>
<td>Cereal grains</td>
<td>Toxemia; carcinogenic</td>
</tr>
<tr>
<td>Tremorgenic toxin</td>
<td><em>A. flavus, P. cyclopium, and P. pustulans</em></td>
<td>Cereal grains, soybeans, peanuts, and other foods</td>
<td>Tremors and convulsions</td>
</tr>
<tr>
<td>Fusarium toxins</td>
<td><em>Fusarium graminearum</em> (sexual state Gibberella zeae), <em>F. tricinctum</em>, and in a minor way, <em>F. moniliforme</em></td>
<td>Cereal grains, corn</td>
<td>Hyperergotism, infertility, stunting, and even death</td>
</tr>
<tr>
<td>Zearalenone (estrogenic syndrome)</td>
<td><em>F. graminearum</em> (sexual state G. zeae)</td>
<td>Cereal grains, corn</td>
<td>Food refusal by swine, cats, dogs; reduction in weight gain</td>
</tr>
<tr>
<td>Emetic or feed refusal factor (deoxynivalenol [DON] or vomitoxin)</td>
<td><em>F. graminearum</em> and <em>F. tricinctum, some strains of F. graminearum, F. equiseti, F. laevidiutum, F. incarnatum, F. nivale, and F. terreus</em></td>
<td>Cereal grains, corn</td>
<td>Severe inflammation of gastrointestinal tract and possible hemorrhage; edema; vomit-</td>
</tr>
</tbody>
</table>


![Fig. 9-8. *Aspergillus flavus* (×1,000).](image-url)
eastern United States have strict aflatoxin testing standards because of the continual risk of contamination of peanuts in the humid Southeast.

Knowledge of the health effects of aflatoxins and other grain mycotoxins is relatively recent. The Food and Drug Administration set the first “action level” (permissible level) for aflatoxins in food products in 1965. Federal regulations on interstate shipment of grain in foods such as cornmeal and peanut butter set the legal limit at 20 ppb. The same threshold is used for feed for dairy cows, but higher levels of aflatoxins may be present in feed for animals raised for meat. The recent development of immunoassays for rapid detection of aflatoxin in food products has improved our ability to screen food products for safe aflatoxin levels. However, there is some concern about the 20-ppb level. Some scientists believe that the limit should be more restrictive. The Soviet Union has a 5-ppb standard, and Japan is considering a 0-ppb limit. Many countries, including the Soviet Union and some countries in Europe, have rejected U.S. grain that does not meet their stricter aflatoxin standards. Controversy also exists about the statistical methods used for sampling large quantities of grain accurately, since pockets containing high levels of aflatoxins are common in large storages. The

Fig. 9-9. Sclerotia of *Aspergillus flavus*. a, Germinating in soil; b, producing conidia.

Fig. 9-10. Moldy corn. Top, in storage; bottom, in ears taken from a field where hail damage occurred.
government sometimes allows grain that is excessively high in aflatoxins to be diluted with other grain to reach the 20-ppb threshold.

The health effects of aflatoxins are well known. They were first dramatically demonstrated in Great Britain in 1960 when 100,000 turkeys and many other birds died of “Turkey X Disease” after eating a shipment of peanut meal from Brazil. The primary area attacked is the liver, and ingestion of high levels of aflatoxins has been demonstrated to cause liver cancer. Further evidence comes from epidemiological studies of high liver cancer rates in Africa, where large amounts of peanuts are eaten, and Asia, where large amounts of soybeans are eaten (although these same areas also have high rates of Hepatitis B virus infection, which is also correlated with the incidence of liver cancer). Aflatoxin contamination of soybeans is a major health concern in Taiwan. It is interesting that the Delaney Clause, which was added to the Food, Drug, and Cosmetic Act to protect food supplies from carcinogenic food additives, does not apply to aflatoxins, which are some of the most potent natural or synthetic carcinogens known.

High aflatoxin levels in crops occur periodically. In 1977, aflatoxin contamination of more than 60% of the corn grown in the southeastern United States resulted in losses of $200 million. The severe drought in the midwestern United States in 1988 resulted in aflatoxin contamination of 5-25% of the corn crop. One third of the 1988 corn crop tested in Iowa and Illinois contained dangerous levels of aflatoxins. Since aflatoxin is not commonly a problem in midwestern corn, milk had to be dumped in more than five states when the contaminated grain was discovered. Since the average American consumes about 160 pounds of corn and corn products each year, this represents a significant public health concern. Increased rates of abortions in hogs in 1989 have also been linked to aflatoxins in feed.

The midwestern corn crop became highly contaminated during the recent drought because of poor husk coverage of the tips of the ears, ear feeding by insect pests that introduced the *Aspergillus* conidia, and cracks in the corn kernels that increased infection by fungi. Speculators bought aflatoxin-contaminated corn and held it until later in the year when desperate cattle raisers would pay higher prices for the contaminated corn because good feed was no longer available.

*Aspergillus* species are not the only fungi that produce mycotoxins in grains and other foods. *Penicillium* species are familiar causes of mold and decay of leather, books, and other materials stored in basements and damp closets. *P. italicum* and *P. digitatum* cause the common blue and green molds on citrus fruits. A number of *Penicillium* species produce mycotoxins that damage the liver, lungs, brain, and kidneys; some are also carcinogens. Several species produce the mycotoxin patulin, which is often found in cider squeezed from damaged apples already invaded by these fungi. Some scientists have speculated that the level of patulin in apples might increase as growers, bowing to public pressure, stop using Alar, a

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**Fig. 9-11.** Green mold on sweet oranges caused by *Penicillium* species.

**Fig. 9-12.** Fungi important in postharvest losses: *A*, *Penicillium*; *B*, *Fusarium*; *C*, *Aspergillus*; *D*, *Botrytis*. 
growth regulator that helps apples resist decay by increasing the natural hemicellulose levels in the fruit. *Penicillium* species also invade grains and contaminate them with mycotoxins.

A third important fungal genus, *Fusarium*, also commonly invades grains and may spread under conditions of high moisture in storage. A rule of thumb has generally been that aflatoxin is a southern problem and *Fusarium* toxins are a northern problem. *Fusarium* toxins, mostly *trichothecenes*, are common in corn and other grains in late harvests after wet summers. The mycotoxins produced by these pink to yellow fungi include “vomitoxin” and “refusal factor,” both of which cause animals to refuse or avoid contaminated grain. *Zearalenone*, another *Fusarium* toxin, has estrogenic effects in animals that reduce fertility. Fumonisins in corn cause a disease of horses called “blind staggers.” T-2 toxin was first identified as the cause of alimentary toxic aleukia in Russia in the early 1900s, but it and other fusarial toxins have probably been a health threat in moldy, poorly stored grains for centuries.

By the time we purchase processed grain products that may be contaminated by these mycotoxins, we can no longer detect their presence by the observation of mold, off-taste, or odor changes. We must trust farmers, food manufacturers, processors, and government inspectors to appropriately harvest, store, and test the foods before they are processed. Since many of the infections take place in the field before harvest, management strategies revolve around protection of the ripening grains and seeds. These include good growing conditions, sufficient field drying, and harvesting techniques that reduce injuries to the seed coat. Once harvested, grain must be kept cool, dry, and free of rodents and insects that cause wounds. Organic acids, such as propionic acid, reduce the growth of fungi that produce mycotoxins, although the treated grain can be used only as animal feed. Most scientists agree that improved accuracy and speed in mycotoxin detection will result in improved healthfulness of the food we eat.

**Edible Fungi**

Humans eat fungi throughout the world. It is well known that some fungi, such as the “death cap” mushroom (*Amanita phalloides*), are deadly, whereas others, such as the common meadow mushroom (*Agaricus bisporus*), are cultivated for supermarkets by the ton. A number of fungi are deliberately added to food and food products to enhance their flavor with no apparently toxic effects.

The knowledge of the edibility, toxicity, and medicinal value of plants and other foods has slowly accumulated over centuries of trial and error by people throughout the world. Mistakes can be deadly, as when a group of newly arrived Asians died a painful death in California after eating mushrooms that appeared to be the same as some edible types from their homeland. Other fungi are toxic to some individuals only. Mushroom hunters who decide to try a new species, after careful identification by an expert, are cautioned to eat only a small amount at first. Some mushrooms, like the inky caps of the genus *Coprinus*, can cause gastric upsets when ingested with alcohol, such as wine, at a meal.

One very familiar fungus, *Penicillium roquefortii*, produces the blue-green color and distinctive flavor of blue cheese. It apparently originated as an accidental contaminant of cheese ripening in caves in France. The name *Roquefort cheese* is reserved for cheeses from that area of France, just as the name *champagne* can only be used in France for sparkling wines from the province of Champagne. The mycelium and conidia of the fungus colonize the cheese until the blue-green veins are visible throughout. Another species, *P. camemberti*, is used for the famous surface-ripened cheese, camembert. The genus *Penicillium* also contains a number of species that produce powerful antibiotics known as penicillin, useful for medicinal purposes. *P. notatum* and *P. chrysogenum* are the most prominent species in commercial antibiotic production.

*Aspergillus* is another common fungal genus that is responsible for mold and decay of food and stored materials but that also includes species used in food production. One species of the genus, *A. oryzae*, is added to rice and soybeans and allowed to ferment for the production of sake and soy sauce, respectively.

Gray mold is a common disease of small fruits, flowers, and the older leaves on many kinds of plants. The distinctive brownish gray mold is visible on infected plant materials in humid weather. The fungus is *Botrytis cinerea*, which produces prolific conidia for dissemination and sclerotia for survival in plant debris. This mold is commonly observed on strawberries, raspberries, and aging leaves and flowers of petunias, tulips, marigolds,

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**Fig. 9-13. Tulip “fire,” or Botrytis blight.**
and geraniums. It is also a common parasite of grapes. Under ideal conditions of cool night air, humid soils, and sunny, warm afternoons, infected grapes become sweeter as the natural sugars and tannins concentrate and the water content of the grapes decreases. Once infected by this “noble rot,” such grapes are used to make special types of wines, particularly dessert wines (golden Sauternes) and other wines, including Riesling, Semillon, and Sauvignon Blanc.

The correct environmental conditions that produce infected grapes without the development of the destructive gray mold disease are found only in certain areas. The fungus is endemic in the vineyards and, therefore, develops naturally in areas conducive to its growth and spread. The infected grapes must be harvested slowly throughout the season as different grapes reach the proper stage for wine making. Botrytis gives a distinctive taste to the wines that bear the name of the fungus or the word botryzed on their labels.

Smuts

Just as it is not possible to judge the danger of a compound based solely on its origin in nature or the laboratory, one cannot necessarily determine the toxicity of fungi and fungal by-products based on their appearance. Another group of fungi has the unappealing common name of “smut.” The name originates with the black masses of dusty spores produced by mature smut fungi. These black spores are formed from the darkening and thickening of the individual cells of the mycelium of these Basidiomycetes. The smut fungi vary in their life cycles, but the black smut spores germinate at some point to produce spores that function in the production of a new dikaryotic mycelium capable of invading a host plant. Smuts are parasites of many kinds of plants, but some of the most serious smut diseases affect the cereals.

One type, loose smut, comprises several species that infect various host plants such as wheat, barley, oats, and rye. Harvested grains from infected plants appear healthy but are already invaded by the mycelium of the smut fungus and will result in a new grain head that bursts with black smut spores instead of healthy flowers, contaminating and infecting the flowers of neighboring healthy plants. Systemic fungicides capable of killing the fungus before the grain is planted have greatly reduced the incidence of loose smut in cereals.

Another important type of smut has several common names including bunt, covered smut, and stinking smut. These smuts are in the genus, Tilletia, named for M. Tillet, a French biologist who in 1755 demonstrated the contagious nature of the smut disease. He dusted the black spores onto healthy grain because he believed that a poisonous substance was associated with the dust. His work was a key step in the early study of fungi as plant parasites. Stinking smut has a different life cycle from the loose smuts in that the smut spores are released at harvest so that any newly harvested grain is contaminated with the spores. Stinking smut spores also survive in the soil and infect germinating grain seeds. In years of heavy stinking smut infection, dusty clouds of smut spores are released when infected grains are broken open during harvesting. The air can be so filled with
spores that farmers have been killed when sparks from harvesting machinery or at grain elevators touched off explosions.

*T. caries* and *T. foetida* do not produce mycotoxins, but smutty grain has an unappealing "rotting fish" taste and smell. Flour made from smutty grain is also brownish due to the presence of the smut spores. It is said that gingerbread was invented to disguise flour made from smutty grain by covering the bad taste with spicy ginger flavoring and adding molasses to mask the brownish color. *T. controversa* causes dwarf bunt of wheat, which is most common in the Pacific Northwest. Because this disease is not yet present in the People's Republic of China, fear of dwarf bunt contamination has interfered with U.S. grain exports to that country.

A common smut familiar to home gardeners is corn smut. This fungus is more common in sweet corn than in field corn. It is recognized by the silvery white, translucent galls found nearly anywhere on the corn plant, including the tassels, ears, and leaf whorls. It usually damages the corn yield and quality only when it invades the ear. The galls produce huge quantities of black smut spores that overwinter in debris and infect corn produced the next year. Despite its unappealing look, this fungus is edible and highly prized in its earlier stages when the developing mycelium has colonized the gall tissue produced by the corn plant. It has been cultivated in Mexico for many centuries and has recently come to the attention of U.S. gourmets.

The fungus is called by the Latin binomial, *Ustilago zeae* (and commonly also *U. maydis*) and by the common Spanish names *huitalacoche* or *cuitlacoche*, which do not have appealing English translations. U.S. markets are using the common name, *smoky maize mushroom*. Farmers in Pennsylvania and some midwestern states are now cultivating corn smut for gourmet food markets, where the fungus sells for a retail price of about $50 per pound on the corn ear, which includes about 3.5 pounds of fungus. It is used like truffles, another type of edible fungus fancied by gourmets. Truffles are fruiting bodies of parasitic Ascomycetes produced on the roots of host trees. They are found by animals that detect their aroma and dig them up, thus dispersing the spores when the truffle is broken open. Humans who desire these fungi train dogs and pigs to find and dig up the highly prized truffles.

Food safety regulation by the federal government is popular with most citizens. Compounds in food that occur naturally in the original plant, animal, fish, or fungus or potential contaminants such as mycotoxins and parasites are regulated, as well as deliberate food additives such as preservatives, colors, flavors, and pesticide residues. The regulation of these potential health hazards is based on their biological activity as toxins, carcinogens, mutagens, and teratogens. The activities are the result of their

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**Fig. 9-16.** Release of "stinking smut" spores (*Tilletia caries* and *T. foetida*) at harvesttime. The spores contaminate harvested grain and soil where they land.

**Fig. 9-17.** Corn smut.
molecular structure, not their origin. Some “natural” compounds are distinctly more important health risks than many “synthetic” compounds. Risk assessment must include dose, frequency, and biological activity of the various compounds. This is why many scientists believe that much more study and screening should be done of grains and seeds potentially infected by mycotoxin-producing fungi than of the extremely low levels of pesticide residues found in most foods.

There is also concern that reduced use of some pesticides may actually increase health risks. Food not protected by fungicides is more likely to possess spots and blemishes that are vulnerable to infection by mycotoxin-producing storage fungi. Thus, “organic” produce may not necessarily be more healthy than commercially grown produce. Optimal storage conditions and protection against infection by storage fungi are requisites of a healthy food supply. In temperate climates, cool storage of food supplies is possible during the winter for free. Tropical countries, which have a year-round warm climate, face much higher losses of food to storage fungi and more danger from mycotoxin production. Dry and well-ventilated food storage structures are a high priority in the development of a healthy reserve food

Fig. 9-18. Disease cycle of corn smut, *Ustilago zeae*.

![Disease cycle of corn smut](image)

Fig. 9-19. Portion of a mural painted by Guillermo Lourdes in the city of Durango in Mexico, showing the importance of corn in agriculture and in Mesoamerican culture. Young smut galls, which are used as food in parts of Mexico, can be seen in the background. From a painted copy by Rudy Cruz.

![Mural showing corn smut](image)

Fig. 9-20. Woman hunting truffles with a pig trained to sniff out and dig up the underground fungi.
supply in some of the most populated parts of the world in Asia, Africa, and Central and South America.

Selected Readings


