

## **Chapter 1 Toxins - kill the primates, rule the world. Or: Don't turn your back on a fungus!**

Like most little girls being taken to their grand-parents for a Sunday treat, Innocenta probably bounced around in the back of the car like an over-excited rabbit. Just three years old, she was the only native-born American in the family. Her parents and grandparents had immigrated to southern Illinois several years before. Now, after a recent move to the Gulf coast of Mississippi, they were having a family get-together one Sunday in 1995.

Grandfather particularly liked their new home because the wooded hillsides inland from the coast reminded him of home. He had taken readily to roaming through the woods and had found many plants and mushrooms he recognized as being the same as those that he and his wife had collected back in the old country for more than thirty years. Amazingly, the locals didn't seem to be interested in this natural harvest and he had begun to collect again for his wife's cooking. For this special day he had found a good crop of succulent mushroom caps to collect with which his wife prepared breaded and deep fried wild mushrooms as a novelty dish for her family's Sunday brunch.

The day went well until about twelve hours after the meal when four of the five family members who had eaten the mushrooms were rushed to the local hospital's emergency department with nausea, watery diarrhoea, and abdominal pain. Grandfather did not have any symptoms and refused medical treatment.

The medical staff in the emergency department realized the possible significance of the meal of mushrooms and treated their patients so as to relieve their symptoms. Giving intravenous fluids and anti-emetics. Importantly, they also monitored liver function and when liver enzyme abnormalities were recognized the next day, all four patients were transferred to the medical centre at the University of South Alabama.

Meanwhile, an expert on mushrooms from the State of Mississippi Toxicology Laboratory had been called to examine mushroom specimens collected by Grandfather. He examined them in detail and identified them as the Fool's Mushroom, which is known scientifically by the name *Amanita verna*. The family members were all suffering from mushroom poisoning.

At the time they were transferred to the University medical centre, all four patients had normal vital signs and were free of symptoms. But the continued medical concern was over the possibility that toxins still coursing through their blood streams were doing irreparable damage to their livers. The function of this body organ is to remove toxins from the blood, but by removing and concentrating them it is itself in great danger of toxin damage. So liver function was closely monitored in all patients and abnormalities found which reached a peak about two to three days after that unfortunate meal. The patients were treated with intravenous fluids to keep their electrolytes in proper balance, with drugs to control bowel movements, and activated charcoal to absorb and remove the remaining toxins from their guts to give some help their livers with toxin removal. The three adult patients were at their worst about two days after the meal but began to recover during the next two days.

Innocenta was not so lucky. Although she was free of symptoms three days after the meal, the little girl's liver was in rapid decline and just one day later the child was transferred to a liver transplant unit where she was listed for an emergency transplant. On day five she required ventilator support, on day seven she was removed from the transplant list when she developed pneumonia. She died from sudden liver failure and general infection on day eleven.

This tragic story is likely to be repeated more frequently as time goes on. 'Natural' foods have

become much more fashionable and their increasing popularity has already lead to an increased incidence of mushroom poisoning among the general population. An incidence which will probably continue to rise. Ironically, though, mushroom poisoning often occurs in experienced collectors. On the European mainland amateur mushroom hunting is a popular pastime which has been practiced for centuries, yet this region has the highest reported incidence of mushroom poisoning in the world. In the United States, too, mushroom poisonings have been increasing since the 1970s in step with changing fashion. Not only are natural foods generally thought to be more wholesome, with wild mushrooms being especially promoted as 'gourmet delights' in magazines, cook books and restaurants, but the 'alternative' use of mushrooms - for their hallucinogenic effects - has also tempted more people into mushroom collecting.

Yet the problem is not limited to those new to mushroom hunting. A Chinese magazine reported large scale mushroom poisoning among villagers in Yunnan province in central China. Sixty-one people were engaged in making a plantation on a hillside on June 28 1997. During the day fifty of them collected mushrooms for a communal meal in the evening. These were people who have lived with and used mushrooms throughout their lives, yet the mistake was made. Poisonous mushrooms were included in the meal. The first illness occurred at 6 am the following day and the first death, of a pregnant woman, occurred on 30 June. By July 1, fifty-one of the villagers showed symptoms of food poisoning (ten people had been too busy to take part in the meal!), and a total of twenty-six of them died. Subsequently, *Amanita verna* and the Death Cap, *Amanita phalloides*, were identified as being in the collection used for the meal.

By all accounts, a very similar tragedy was narrowly averted in Italy in 1984 when a mycologist spotted the Death Cap (*Amanita phalloides*) on sale in Potenza market. He saved the town from catastrophe by making radio and TV broadcasts and touring the town by car, warning the locals of the danger by loud-speaker.

It is this close resemblance between poisonous and edible, even delicious, mushrooms that creates the problem. A little knowledge can be dangerous and even experts get it wrong. The 1991 edition of the authoritative Larousse Encyclopedie had to be recalled because the illustrations of deadly amanitas were labelled 'indifferent' rather than 'poisonous'. By the time the error was noticed, 180,000 volumes had been distributed and 250 people had to be hired to visit 6,000 book stores in France, Belgium, Switzerland and Canada to overprint the correct wording on the right page.

In fact, fairly few mushrooms are poisonous to humans - fewer than fifty of the more than two thousand species of mushrooms which are commonly listed as occurring in most countries can be considered poisonous, and only about six are deadly. In a sense this point is irrelevant because, unfortunately, most serious poisonings are due to the very species of *Amanita* which contain two of the most potent toxins known, cyclic peptides called amatoxin and phallotoxin. *Amanita phalloides*, is aptly named Death Cap because it accounts for more than ninety percent of fatalities in the United States and Western Europe, and the other two deadly species are *Amanita verna*, and *Amanita virosa* (the common name for which is the Destroying Angel).

Both of the toxins interfere with basic aspects of normal cell biology. Amatoxins bind to the RNA polymerase enzyme and interfere with production of the messenger-RNA molecules which take the genetic information from the genes themselves to the protein manufacturing machinery in the cell. With this process stopped by amatoxin, the cell is unable to produce vital working proteins. The cell cannot function properly and so it breaks down and eventually dies. Body organs which normally have high rates of protein synthesis are particularly sensitive to this toxin. Liver cells (called hepatocytes) are in this group and are most commonly involved in mushroom poisoning, but other target organs include kidney, pancreas, testes, and white blood cells.

Phallotoxin causes irreversible disruption of the cell membrane and cytoskeleton and cell death inevitably results. Phallotoxin is not absorbed from the human gut, so is not thought to be responsible for the symptoms associated with human poisonings. Amatoxins are readily absorbed from the gut and are then concentrated in the liver where they do their damage. A patient poisoned with *Amanita* goes through four characteristic phases. For the first six to twenty-four hours the patient is free of symptoms; then there's a twelve to twenty-four hour period of severe abdominal cramps, nausea, vomiting, and profuse watery diarrhoea. In this stage patients are often misdiagnosed as having gastric influenza, and as in the third phase the patient's gastrointestinal symptoms improve over a further twelve to twenty-four hour period this misdiagnosis may seem to be right and the patient appears to be on the way to recovery. But all through this time the toxin is damaging the liver; killing its cells and literally ripping it apart from within. Symptoms of liver damage only appear four to eight days after the toxin was consumed, but the liver damage can progress rapidly. Accelerating all the time. Death from catastrophic failure of the liver can occur anything from six to sixteen days after the fatal meal was consumed.

Providing misdiagnosis can be avoided; providing liver function is monitored, and providing proper supportive care is made available, the patient has a fair chance of surviving the poisoning. In the long run, though, this may require a liver transplant. Even with the best medical care, death still occurs in twenty to thirty percent of cases and there is a mortality rate of more than fifty percent in children less than ten years old.

People who are most at risk of being poisoned through eating wild mushrooms are toddlers, who are likely to eat anything on an experimental basis, recreational drug users, who think that any trip is worth a try, and immigrants who are in danger of collecting toxic mushrooms that resemble species that are safe to eat in their home regions.

Evidently, cure is difficult and far from certain, so the best strategy is avoidance! Unfortunately there are no reliable rules to help you tell a poisonous mushroom from an edible one. One rule that you should obey, though, is take no notice of the old traditional advice that poisonous mushrooms make silver spoons turn black or that a mushroom that can be peeled is alright to eat. The most deadly poisonous of all mushrooms fail both of these 'tests' completely! There are a host of other, equally nonsensical, traditional beliefs to do with colour and shape, smell and taste, milky exudates from the flesh of broken mushrooms or change in colour of the flesh, ability to coagulate milk or change the colour of onion or parsley during cooking; all are valueless in identifying poisonous mushrooms reliably.

The only certain test is eating, but don't put any greater reliance on those other bits of traditional 'wisdom' which suggest that mushrooms showing evidence of being nibbled by rabbits or squirrels or eaten by slugs are edible. Slugs seem to be particularly fond of *Amanita phalloides*, and the rabbit digestive system is able to detoxify these mushroom poisons. In any case, while you are admiring its tooth-marks around the margin of the dainty morsel you've just found, what makes you think the animal itself is not lying dead down a burrow somewhere?

I don't think we should put any faith in traditional wisdom relating to identification and traditional methods of neutralizing the poisons in fungi by special methods of cooking are equally unreliable. From the time of the ancient Greeks, people have advocated boiling in oil or with meat or with pear stalks. Vinegar is also supposed by many to neutralize the toxins. In the mid-nineteenth century a French specialist experimented with a method involving steeping toxic mushrooms in vinegar or salt water for two hours, followed by boiling for half an hour. Repeated tests at the time gave some credence to the method, but it is doubtful that the original experiments really used the most toxic fungi, so the method is just as worthless as the rest. I've got to ask whether a mushroom, toxic or

not, that's been pickled in salt water or vinegar and then boiled for half an hour is worth eating - and my answer to my own question is no!

Nor do I have much confidence in traditional advice about identification or that about detoxification and, frankly, traditional treatments turn my stomach! One which received much publicity at the time was based on the belief that a rabbit can eat the Death Cap without ill-effect. This leads to the conclusion that the rabbit's stomach must be able to neutralize the liver toxin and its brain must be able to neutralize the nerve toxin. From this logic came the treatment regime: take the stomachs of three rabbits and the brains of seven, chop them up finely and give them (raw!) to the patient mixed with sugar or jam. Bearing in mind that the patient will have been suffering from distressing vomiting and diarrhea, I suspect that a meal of raw rabbit stomach and brain would not have been a high point in the recovery process.

Mushroom poisoning is far from being a new problem. It is mentioned in ancient Greek and Roman writings. Pliny the Elder said that '... Although mushrooms taste wonderful, they have fallen in disrepute because of a shocking murder. They were the means by which the emperor Tiberius Claudius was poisoned by his wife Agrippina...' Toxic mushrooms are very convenient for poisoning and attempts to use them have not been restricted to ancient Romans, though there are only a few cases on record. The most remarkable is a French case of 1918 in which a murderer who impersonated his victims to buy life insurance used various biological agents, including disease bacteria like anthrax and typhoid and toxic mushrooms, to dispose of those victims. He was eventually caught because an insurance company doctor realized that the corpse bore no resemblance to the 'insured' he had examined some time before the lethal event!

Although dramatic and tragic, death from mushroom poisoning occurs in a minority of cases. Most people who eat a poisonous mushroom suffer an unpleasant but relatively mild and short-lived bout of 'food poisoning'. The quicker that symptoms arise, the less severe is the attack. The toxins of deadly mushrooms are slow-acting and do not cause symptoms for six to twelve hours or more after eating. Those causing milder poisonings make people sick in two hours or less. After a few hours discomfort - vomiting, nausea and cramps, accompanied by diarrhoea - the victim recovers completely.

The milder symptoms of mushroom poisoning are considered desirable by some people. For generations in several areas of the world intoxicating mushrooms have been eaten in connection with religious ceremonies. For example, some tribes on the Kamchatka peninsula in Siberia have eaten the Fly Agaric mushroom (*Amanita muscaria*) because the toxins it contains give rise to intoxication, hallucinations, and, allegedly, superhuman feats of strength. The Vikings are said to have used this mushrooms for the same purposes. But the most widely known mushroom-eating ceremonies are the magical sacred rites which originated with the Aztecs. The mushrooms used by the Mexican Indians are species of *Psilocybe*, *Conocybe*, and *Stropharia*. They contain psilocybins, chemicals that cause visual hallucinations, an ecstatic state, altered perception of time and space, and, in a good 'trip', a general feeling of exhilaration and well-being lasting several hours. These are hallucinogenic fungi and, not surprisingly, mushrooms able to produce intense excitement in the consumer are now deliberately used for that purpose. Such psychoactive fungi, or 'magic mushrooms', are sought eagerly by those yearning for the narcotic intoxication they offer. They are not uncommon fungi, being mostly specialized to the degradation of waste plant material. They prefer disturbed ground and so are often found around people - in parks, gardens, construction sites - as well as in forests and fields around the world. Fresh supplies can be found in most places, and can be bought in many. Indeed, walk around the centre of Amsterdam and you will find well-appointed shops selling all of these, and *Amanita muscaria* as well. But there is no guarantee of a great trip! The psychotropic effect is a toxic effect. Different from the *Amanita* toxicoses, yes, but

the semi-conscious mild delirium and hallucinations are reactions of the nervous system to the toxins the psilocybin-fungus contains. Whatever their protagonists may claim, there is no more enlightenment in fungal intoxication than there is in alcoholic intoxication – it's all a matter of the misfiring of a nervous system tortured by chemicals it was not designed to cope with.

If you go out collecting wild mushrooms, the key is to be sure about what you collect for eating, but remember that *Amanita* fruit bodies are commonly mistaken for edible species, and cooking does not destroy the toxins. *Amanita* is widely distributed because it is mycorrhizal - a fungus which lives in symbiosis with the roots of a host tree, mainly birch - so it will be present in many woodlands. And these are the very places where you are most likely to go to find edible mushrooms. *Amanita* does not have a characteristic smell or taste, and colours vary with weather and soil conditions as well as age of the mushroom. There are three features which do identify *Amanita*: the gills underneath the cap are white, there is a skirt-like ring, or annulus, around the top of the mushroom stem, and there will be an inverted skirt (a volva) arranged like a pouch around the swollen base of the mushroom. As it is much easier to detect the presence of the swollen base by digging the entire fruit body out of the ground, it is not good practice to simply collect mushroom caps or even cut off the stems at ground level.

Eating wild mushrooms can be deadly and experience shows that even competent collectors can make mistakes, but over-dramatic reaction is not necessary, nor even helpful. I have seen suggestions that because of the risk of accidental ingestion, '...the public should be advised to collect the fruiting bodies of mushrooms as they emerge from the ground and dispose of them...' Indeed, a newspaper report of September 1998 told of the hysterical reaction at an English primary school when a child picked up a mushroom on the school playing field. The child was rushed to hospital, the school's annual sports day was postponed and the field was sprayed with fungicide. At the end of the day the child left hospital fit and well and the local university identified the 'deadly mushroom' as a common edible one! If this is the hysterical aversion end of the spectrum of reactions to mushrooms, I think the other end of the spectrum is equally ridiculous. This is the end which is populated (possibly temporarily in view of their habits) by dedicated mushroom collectors who tempt fate by attempting to distinguish between the fatal death-cap mushroom, *Amanita phalloides*, and its edible look-alike *Amanita caesarea*. To me this is not sensible mushroom collecting; rather, it is an extreme sport like free-fall parachuting where a major part of the attraction of the exercise is the very fact that you risk your life by doing it. I do not have much sympathy with either hysterical aversion or deliberate danger-seeking. Eating one of the most toxic mushrooms can lead to death. Indeed, some people have suggested that eating just one cubic centimetre of one of these mushrooms is enough to kill the average person. Data on serious mushroom poisonings are not widely collected so the exact numbers who die each year from this cause is not known. It's been suggested that deaths due to mushroom poisoning in the United States are fewer than those caused by bee stings or lightning. Probably not much consolation for the victims but neither does it make a logical reason for wanton destruction of the mushrooms in nature. In Britain, about 20 civilians are killed in road traffic accidents involving police cars each year. Should we make the police walk? My rule of thumb for eating mushrooms is only eat the ones with dark-coloured (brown, purple, black) spores. You might miss a few delicacies that way, but you'll miss the toxic ones, too! Oh, and cross the road carefully when you hear police sirens.

Mushrooms are not the only fungi able to produce toxins which have plagued humanity over the years. Imagine a disease whose victims feel as if they are burning up, literally on fire, or as if ants, or even mice, are crawling about beneath their skin. Imagine that as the disease develops they may suffer terrible hallucinations- so bad as to sometimes drive them insane. If they avoid the hallucination and madness an even greater suffering awaits. After a few weeks limbs become swollen and inflamed, violent burning pains alternate with feelings of deathly cold and gradually

the affected parts become numbed, then simply fall off the patient's still living body. Fingers, toes, hands, feet, arms and legs, all may separate from the body and rot away.

The first record of this terrible disease dates from 857 AD; a chronicle from Kanten in the Lower Rhine region of Germany describes a '... great plague of swollen blisters that consumed the people with a loathsome rot, so that their limbs were loosened and fell off before death ...?'. At intervals throughout the following centuries there were epidemics in which an enormous number of victims died. One bad outbreak in France in 944 AD reputedly killed 40,000 people; the last epidemic of any considerable extent was in the 1880s. Records in the eleventh and twelfth centuries refer to this plague as holy fire, but it became associated with St. Anthony, as those suffering from the disease started to visit the saint's relics to seek relief and solace in faith. Most cases of what therefore became known as St. Anthony's fire occurred in France and its distribution reflected the cultivation of rye and use of the grain to make bread. The human disease is caused by eating bread prepared from rye which is itself suffering a fungal disease. The plant disease, called ergot, is caused by the fungus *Claviceps purpurea* which is parasitic on wheat, rye and other grasses. As it is most common on rye it is often called ergot of rye. Spores of the fungus are carried on the wind until they land and infect the flower of the grass. The spore germinates on the stigma of the flower, and then grows into the ovary. There it uses the food intended to nourish the seed to produce a curved mass of compacted cells, called a sclerotium, in place of the seed. When ripe, the sclerotium projects from the head of the rye as a hard purple to black, slightly curved thorn. Ergot is the French name for the spur on the foot of some birds which the sclerotia resemble, hence the common name of the disease. In wild plants, these sclerotia fall to the ground in autumn where they over winter and germinate in spring to infect the next generation of plants. But on farms the sclerotia are harvested and milled with the grain. The flour then becomes contaminated with the toxins the sclerotia contain.

Ergot yields three groups of compounds: (ergotamines, ergobasines and ergotoxines) which have now become medically important. The first two groups excite smooth muscles (which control the uterus, blood vessels, stomach and intestines) and are now used to promote contractions of the uterus in childbirth, to stem haemorrhaging and to control migraines. The third group, ergotoxines, have an inhibitory effect on those parts of the nervous system that affect mood and emotional state, and are used to treat psychological disorders such as delirium tremens and hysteria. The use of ergot as a clinical drug has been recorded from the sixteenth century, when the ergots themselves were used by European midwives to induce labour and to terminate unwanted pregnancies.

Ergot poisoning, now known as ergotism, causes two groups of symptoms which are manifestations of the same type of poisoning, gangrenous and convulsive ergotism. The former was frequent in certain parts of France, the latter occurred mainly in Central Europe. The convulsive form results from ergot poisoning combined with a deficiency of vitamin A, probably itself caused by a lack of dairy produce in the diet.

In gangrenous ergotism it is constriction of the blood vessels, especially to the extremities, which eventually causes gangrene and death. In convulsive ergotism the effects of the ergot alkaloids on the nervous system are most pronounced. Twitching, spasms of the limbs, and strong permanent contractions, particularly of hands and feet. In severe cases the whole body is subject to sudden, violent, general convulsions, all coupled with hallucinations and visions. Ergotism has been suggested by some to explain the convulsive fits which took hold of eight young girls in Salem, and led to the witch trials of 1692, during which nineteen people, mostly women, were pronounced guilty and hanged.

Britain has been singularly free from ergotism. There is only one record of typical gangrenous type,

in the family of an agricultural labourer from near Bury St. Edmunds, in 1762. Mother and five children all lost one or both feet or legs: the father suffered from numbness of the hands and the loss of finger nails. Recognition of the disease, improvements in grain preparation and, especially, the introduction of antibiotics in the latter half of this century to control the bacterial infections have all helped to remove human ergotism from our catalogue of miseries. The only UK epidemic occurred in Manchester in 1927 among Jewish immigrants from Central Europe, who lived on bread made from a mixture of wheat and rye. All two hundred suffered symptoms of mild convulsive ergotism. At the same time, September 1926 to August 1927, and perhaps because the grain came from the same source, there were twelve thousand cases of convulsive ergotism in Russia. The last recorded outbreak, in France in 1951, caused more than two hundred people to suffer from hallucinations, and five died. One young victim believed she saw geraniums growing out of her arms. But ergotism in livestock remains a common problem arising from the ergot infection of fodder grasses. Sheep suffer from inflammation and ulceration of the tongue. In cattle, the symptoms are lameness and dry gangrene in the feet, and at times the whole foot can be lost.

There's one last fungal toxin that's worth a mention - and certainly worth avoiding! It's called aflatoxin. As you might expect, there are several related aflatoxins, which are produced primarily by the moulds *Aspergillus flavus* and *Aspergillus parasiticus*. They are considered to be the most active cancer-causing (carcinogenic) natural substances known. These toxins arise in crops like corn, peanuts, and to a lesser extent rice and soybeans, even before harvest, but particularly when stored under the warm and moist conditions which permit growth of the infesting fungi. This is a problem in developing countries, especially throughout tropical Africa, and the South East of the United States.

When the tainted crop is eaten, the aflatoxin is absorbed and metabolically activated by liver enzymes. Activated aflatoxins interact with the workings of liver cells leading to their death, which can cause hepatitis, or transformation into cancerous growths. The human liver has a fairly slow metabolism, so susceptibility to acute poisoning is relatively low. Animals are more in danger, particularly poultry. In the 1960s one hundred thousand turkeys died on poultry farms in England. The disease was unknown at the time and first became known as turkey X-disease. It was eventually found to be due to contaminated poultry feed and this costly event initiated aflatoxin research. More serious to humans is that prolonged exposure causes primary liver cancer as well as other cancers.

Aflatoxin health risks differ considerably around the world. Regions with a warm, humid climate generally favour able to mould growth and with poor regulatory and control systems, countries like Kenya, Swaziland, Uganda, China and Thailand may have as much as five hundred times higher incidence of liver cancer than is common in developed countries. The European risk comes mainly from importing of contaminated foods (mainly nuts) and animal feeds. Aflatoxin can enter the human food chain from such contaminated feeds. Grains, peanuts, other nuts and cottonseed meal are among the foods on which aflatoxin-producing fungi usually grow. Meat, eggs, milk and other edible products from animals that consume aflatoxin-contaminated feed are also sources of potential exposure to humans. To protect against too much exposure, most developed countries measure aflatoxin levels and acceptable levels in human food and animal feeds are controlled by legislative authorities. Still, Americans may consume up to half a microgram of aflatoxins every day, and aflatoxins are among the more than two hundred environmental chemicals that have been found in samples of human breast milk. The levels of hazardous chemicals like this in human breast milk are a measure of our progress (or lack of it) in cleaning up our environment. One expert says that 'there is no uncontaminated mothers' milk anywhere in the world... all mothers carry environmentally-derived chemicals in their bodies.' Nevertheless, the benefits of mothers' milk to a baby are incalculable because the baby is provided with antibodies against infection, protection against

allergies and particular nutrients vital to intellectual development. So although the levels of toxins found in breast milk are a cause for concern there isn't any hard evidence yet that they can harm baby and the advice is still that most women should breast-feed. The emphasis is shifting towards avoidance of the toxic chemicals. On the one hand, mothers-to-be could avoid meats and freshwater fish that could be contaminated - that usually means avoiding the cheaper products because corner could have been cut to make them cheap. Reduced consumption of dietary fat is a good idea too, because many toxins are accumulated in an animal's fat.

On the other hand the industrial watchdogs and regulators can control the basic crops that are at risk to minimize contamination and prevent entry of contaminated materials into the food chain. Crop rejection and losses associated with laws regulating the aflatoxin contents have significant economic impact, primarily, but not only, on developing countries. Costs caused by aflatoxin contamination of corn, for example, were estimated to be about two hundred and forty million US-dollars annually throughout the 1980s. The United States poultry industry losses from aflatoxin poisoning greatly exceed one hundred million US-dollars per year. Most of the loss resulting from slow weight gain and reduced feed efficiency rather than from mortality.

Control of aflatoxin is restricted to physical separation of contaminated lots, partial decontamination, and prevention of further contamination by provision of good storage conditions. Procedures that try to remove aflatoxins are generally inadequate so the best chance of making sure that foods and feeds are free of aflatoxins is by preventing mould infestation and toxin production in the first place. Condensation of moisture on roofs and walls, leaking roofs, seepage of water into warehouses and the like are some of the causes of mould growth during storage. Every place where moisture can get to the product and cause mould growth must be eliminated.

Refrigeration and climate control is useful. Reducing the oxygen concentration (to less than one percent) and increasing the carbon dioxide (to above twenty percent) can also inhibit mould growth. Sadly, all of these measures are costly and since huge tonnages of aflatoxin contaminated produce come from developing countries in the tropics, most of these desirable storage measures are completely impracticable. We have to fall back on continued vigilance. Rely on this: the mould will be there. Don't drop your guard!

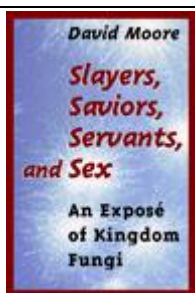
These fungal toxins are so effective at killing, maiming and disorienting humans that it is worth asking why the fungi produce such deadly toxins. Amongst the mushrooms and toadstools they are fairly obviously protective agents, defending the mushrooms against destruction by grazing. The mushrooms are structures intended for spore distribution. They are the shape that they are to protect the spore distribution mechanism from the rain. The ones that contain toxins contain such chemicals to protect the mushroom against animals that might otherwise eat it. An eaten mushroom is not a successful mushroom! My argument about the purpose of toxins is an evolutionary one. I interpret toxins as products which are intended to improve the evolutionary fitness of the mushroom. Survival of the fittest is the basic Darwinian statement. What it means is that the individual that is best suited will survive to produce progeny. The crucial point is that the Darwinian selection process must benefit the individual. The benefit to the mushroom is that it should not be eaten, but survive to distribute its spores. Yet the *Amanitas* are fairly anonymous species - people die because they mistake the *Amanita* for some innocuous mushroom. The toxin is not a warning because there is no accompanying warning coloration which the eater could learn to avoid on some future occasion. So I believe the toxin is an immediate deterrent. Something which will cause immediate distaste, slight injury, or a jolt to the nervous system which will make the eater stop eating and go elsewhere. Despite the lurid title of this chapter, I don't think the toxins are aimed at large mammals like humans. A large animal will eat the whole mushroom; then both will end up dead - the animal killed by the toxins and the mushroom, with its load of progeny spores, being digested in the



stomach of what will by now be a rapidly rotting corpse. No, I think the toxins are aimed at small animals. They are chemicals that make the fungus tissue immediately distasteful to insects, slugs, small mammals which might chew away small chunks of the mushroom at a time. Animals of this sort are not intellectual heavy-weights. There is no place for subtlety here, so a very potent toxin (and the higher the potency the less the mushroom will need to produce) which effectively kicks the grazer in the mouth will do the job very nicely. Poisonous fungi have enzymes similar to those the toxins attack but the fungal enzymes are more resistant to the toxins than the same enzymes of animals.

This could certainly also apply to ergot. Remember that the ergot sclerotium must last out the winter to perform its biological function of reinfecting plants in the spring. The toxins it contains may well deter soil animals from eating too much of the ergot during its winter of waiting. The same argument is applicable to aflatoxins because the moulds which make them are in competition with mites, weevils and insects for the food value which the stored crop represents. Producing a toxin powerful enough to drive away these competitors could be an important way of tipping the balance in favour of the fungus. Unfortunately for us, such potent toxins also kill the primates.

I've seen it suggested that the simple answer that toxins protect against being eaten is not very satisfying. The argument runs that if this feature were so important poisonous mushrooms would be expected to be more abundant than the innocuous ones and eventually would be expected to become the dominant type (which has clearly not happened). This line of argument is too silly to take seriously. The point is that no-one is suggesting that producing the toxins is the only, still less the best, survival strategy. It is one of a number of potential strategies. Some organisms may take advantage of the possibility that being eaten helps disperse the spores (spores are not easily digested and may pass through the intestines). Others may strengthen their cell walls to become too tough to eat, or adapt to become inconspicuous. They are all valid evolutionary strategies. All contributing to life's rich pageantry.



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